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Ambient neighbourhood noise and children's mental health

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Objectives: To investigate the relation between typical ambient noise levels (highway, rail, road) and multiple mental health indices of school children considering psychosocial and biological risk factors as potential moderators.

Methods: With a two stage design strategy (representative sample and extreme sample) two cross sectional samples (n=1280; n=123) of primary school children (age 8–11) were studied. Individual exposure to noise at home was linked with two indices of mental health (self reporting by the child on a standard scale and rating by the teacher of classroom adjustment on a standard scale). Noise exposure was modelled firstly according to Austrian guidelines with the aid of a geographical information system and then calibrated and corrected against measurements from 31 locations. Information on potential confounders and risk factors was collected by mothers and controlled in regression modelling through a hierarchical forward stepping procedure. Interaction terms were also analysed to examine subgroups of children at risk—for example, low birth weight and preterm birth.

Results: Noise exposure was significantly associated in both samples with classroom adjustment ratings. Child self reported mental health was significantly linked to ambient noise only in children with a history of early biological risk (low birth weight and preterm birth).

Conclusions: Exposure to ambient noise was associated with small decrements in children's mental health and poorer classroom behaviour. The correlation between mental health and ambient noise is larger in children with early biological risk.

The present study focuses on the relation between typical, everyday neighbourhood noise levels and children's mental health. Mental health in children is usually measured in one of three ways: psychiatric evaluation of anxiety, depression, conduct disorder, or psychoses; self reported questionnaire measures of clinical symptoms of anxiety, depression, and conduct disorders; or by either teacher or parent ratings of behavioural adjustment. Numerous studies in the child psychiatric and clinical literature indicate the reliability and validity of all three types of measurement for non-clinical populations, with psychiatric evaluation reserved for more serious, clinically relevant symptoms of serious disorders such as disassociation.[1–4] In the present study we incorporated child self reported and teacher ratings of mental health. We judged that psychiatric evaluations were overly intrusive and unnecessary for the modest increases in the non-clinical range of mental health symptoms we expected to see in a sample of the general population.

There are several important gaps in the noise and mental health literature considered by the present study. Firstly, previous studies of noise and mental health all focused on major noise sources—such as airports or major highway traffic—and neglect typical neighbourhood noise as experienced by most of the population.[5–8] Although it is obviously important to focus noise protection efforts on those most heavily impacted, we should not overlook the fact that nearly half of the World Health Organisation European region is exposed to daily community noise levels of between 55 and 65 adjusted noise levels (dB,A).[9] This ambient noise level exceeds recommended criteria for residential areas, schools, and hospitals.[10 11]

A second gap we considered is that nearly all studies of noise and mental health have focused exclusively on adults. Only one study has examined noise and mental health in children. Bullinger *et al* studied 326 8–11 year olds attending primary schools in high noise impact zones and comparison groups around the Munich-Riem airport.[12] A third shortcoming in the noise and mental health literature that we considered is the problem of the small sample sizes. This has precluded the incorporation of sufficient multivariate controls for other relevant mental health factors (biological, psychosocial, and environmental risks) and therefore not provided researchers with the opportunity of examining mental health within a multivariate risk model.

A common analytical strategy in environmental epidemiology is to examine the relation between an environmental agent and health, while statistically controlling for other variables. The application of general linear models depends upon the assumption that the slopes of the regression plots for the predictor are parallel across each statistical control (the regression plots all have the same slope).[13–15] When this assumption is violated, there is an interaction between the predictor variable (noise) and the control variable as they affect the outcome of interest (mental health). Therefore, it is important to first test whether any control variables interact with the environmental risk factor of interest. The presence of a statistical interaction is also important for theoretical and practical reasons.[16 17] Statistical interactions can help us better understand the nature of environment and health relations, pointing towards potential explanatory mechanisms and processes. From a policy perspective, interactions may uncover vulnerable population subgroups, hidden by weak or non-significant overall effects.

Thus the present study was designed to investigate whether a large representative sample of children living under typical neighbourhood noise levels in small, alpine towns and villages in Central Europe (Austria) would show

Abbreviations: Leq, noise exposure; dB,A adjusted noise levels; dB,A,Leq, equivalent sound pressure level; dB,A,Ldn, equivalent sound pressure day-night level

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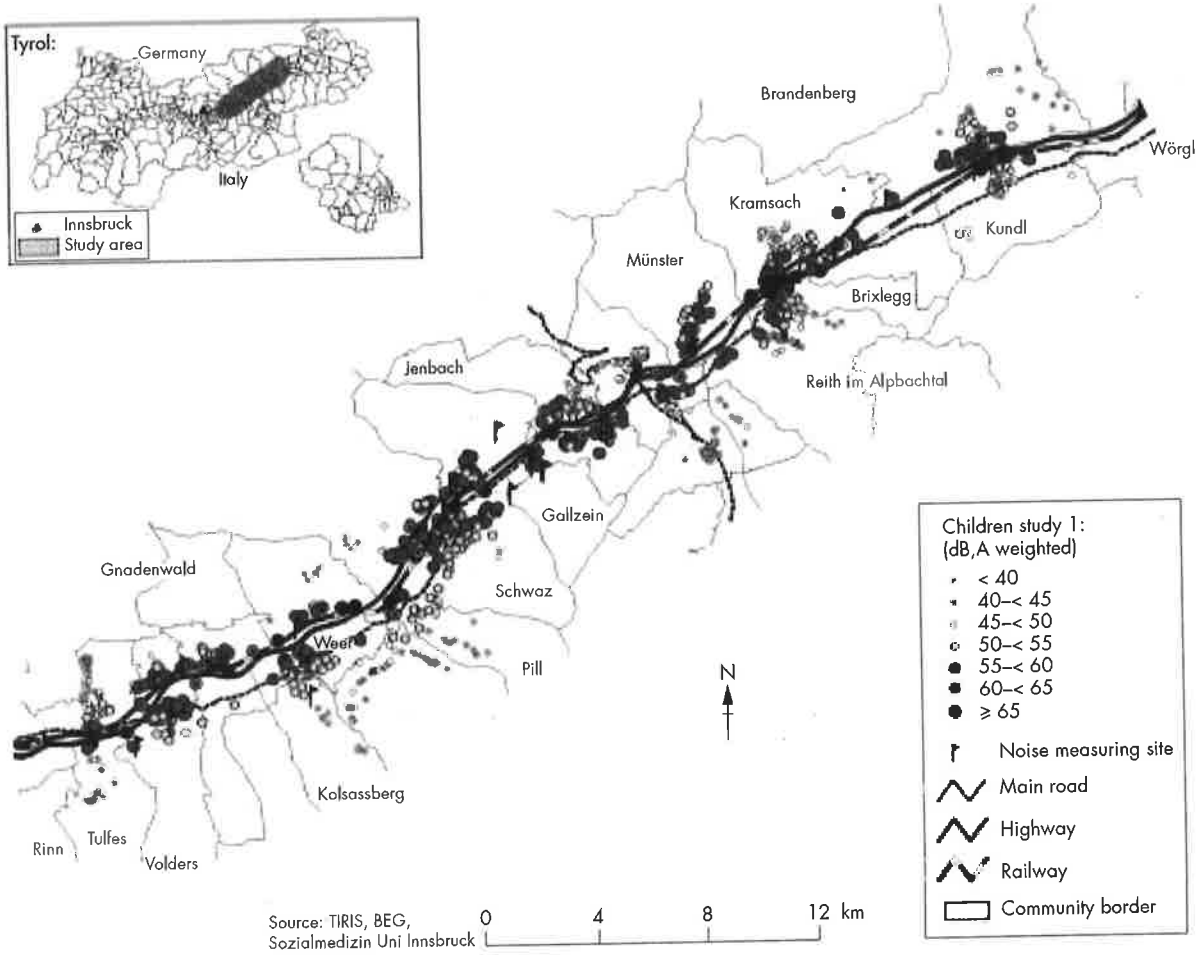

Figure 1 Children study 1. Sample distribution across study area by noise exposure levels: from a representative sample of a population study.

any relation between a broad range of exposures to noise and two indices of mental health. The sample was sufficiently large so that we could examine multiple biological and social risk factors and as already explained, investigate statistical interactions. We also employed an innovative variant of a classic two stage study design.[18] As well as obtaining a broad, representative sample of the population, we also oversampled a subset of children at the high and low ends of the range of exposure to noise in the community. We did this because extreme group analysis, whereas not ecologically representative, has a distinct advantage—they provide substantially greater statistical power.[19 20] This enables investigators in a new area to identify suspected environmental risk factors—such as noise for children—where there is a paucity of knowledge. Such research designs on extreme exposure can uncover early warning signs of a potential public health problem before they reach levels sufficient to be seen in the overall population. This research design also provides possibile internal replication. Do the conclusions from the highly exposed population generalise to the dose-response analysis in the general population?

Methods

Subjects and procedures

In June 1998 all school children in grades 3–4 (mean 9.44 years) were approached in a demarked area defined by the Austrian Government as an environmental health impact assessment. A total of 1280 children from 26 local schools participated (response 79.5%) after a letter was sent to their parents (population study). Children, their mothers, and their teachers were informed that this was a study of traffic, environment, and health required by law to supplement the environmental health impact assessment. The survey area (about 45 km long) is in the lower Inn Valley of Tyrol in Austria (fig 1. This mainly rural, alpine area consists of small towns and villages with a mix of industry, small business, and agricultural activities outside Innsbruck. A geographical information system was set up to enable multiple data linkages and to ease additional sampling.

After the geographical information system link of calculated noise exposure (equivalent sound pressure level, dB,A,Leq) with the child's home address two new samples of children in grade 4 were drawn in September 1998 from the extremes of the available exposure information (< 50 dB,A,Ldn $\nu > 60$ dB,A,Ldn (day-night levels)). Sixty three children inhabited low and 60 resided in moderate and higher noise exposure locations (fig 2). Participation rates in the extreme analysis was lower (64%) because the data were not collected at school and required more time. However, as table 1 shows, the two samples did not differ significantly on various social, lifestyle, and biological factors.

Background information

Sociodemographic data and biological risk information were collected from each child's mother to assess standard risk factors and to check for possible statistical interactions. Prenatal and perinatal data were assessed from doctor's entries in the "mother-child-passports"—every pregnant mother in

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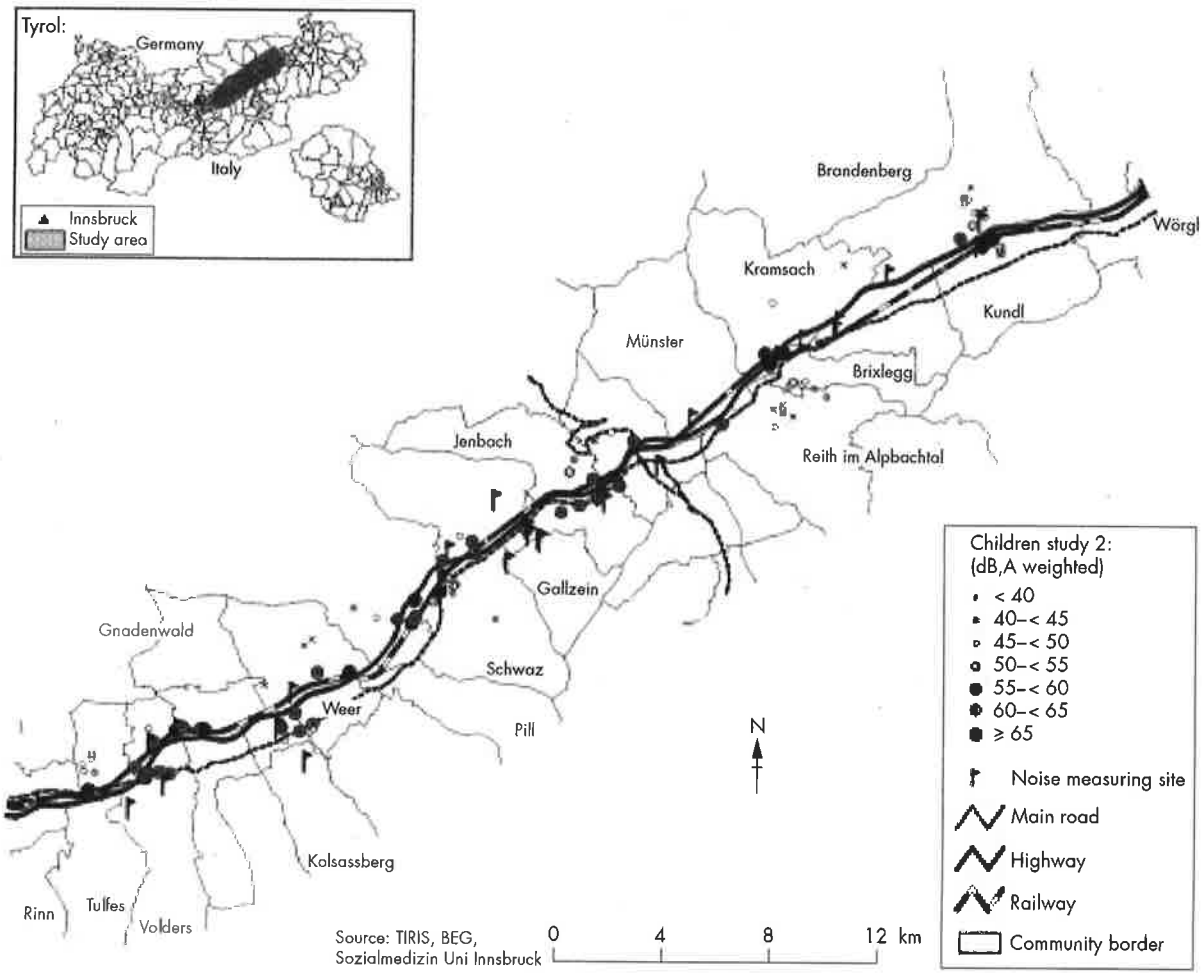

Figure 2 Children study 2. Sample distribution across study area by noise exposure levels: study sampling on extreme exposure (<50 bD,A,Ldn; >60 bD,A,Ldn).

Austria receives one of these. Biological risk (0 or 1) was defined as low birth weight less than 2500 g or preterm birth less than 37 weeks gestation. Other biological variables recorded were maternal age, parity, and birth order. Further biological, social, and environmental data were collected with a self administered, standardised questionnaire from the mother. Mother's education was scaled along a five point continuum from 9 years of school to graduate work. Family size, single parenthood, house type (1=single family detached, 2=terraced house, 3=multiple dwelling units), duration of residence (years), and months of breast feeding, were recorded.

Exposure assessment

Residential noise exposure is usually assessed by an index that describes the average noise exposure (dB,A) over a specified time period (Leq, day or night or 24 hours). In this study noise exposure (Leq) was assessed first by modelling (Soundplan) the three major sources (highway, rail, local main road) according to Austrian guidelines (ÖAL Nr 28+30, ÖNORM S 5011). The dichotomous sampling for the pilot study was based on this information. Afterwards a calibration study (31 measuring points) was conducted (day and night measurements) and linear corrections were applied to the modelled data when the difference to the measured data exceeded 2 dB. Based on both data sources approximate day-night levels (dB,A,Ldn) were calculated for each child's home to enable comparison with available dose-response data. This calibrated noise exposure information (combined levels from all sources) was used in all the dose-response analyses. The noise range in the field study was between 31 and 81 dB,A,Ldn (95% within 40–65 dB,A,Ldn). The range in the pilot study was 31 to 72 dB,A,Ldn (95% within: 34–50 dB,A,Ldn) in the low exposure group, 52–71 dB,A,Ldn in the high exposure group.

Psychological health

A 22 item scale was formed from two subscales of the KINDL,[21] a valid and reliable index of children's quality of life and four items on a sleep disturbance scale. Analyses of the intercorrelations of the three scales showed the potential to combine the scales, which yielded one internally consistent index of psychological health (Cronbach's α=0.87). Children reported from 0=never to 4=very often, how often they experienced various symptoms indicative of anxiety and depression appropriate for a non-clinical population (I feel lonely; I have trouble falling asleep at night; everything I start turns out right). The psychological health scale was administered to children in their classroom by two graduate students who were blind to the child's exposure to ambient noise.

Classroom adjustment

Each child's teacher was asked to rate the child on a standard index of behaviour.[22] This 11 item, dichotomous (yes or no)

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Table 1 Main characteristics of both study samples

Characteristic	Population study*	Extreme exposure study†
Sample size (n)	1280	123
Age child (y, mean (SD))	9.44 (0.70)	8.96 (0.45)
Age mother(y, mean (SD))	36.33 (4.90)	36.48 (4.68)
Sex (% male)	51	55
Body mass index (kg/m^2, mean (SD))	17.28 (2.61)	17.49 (2.79)
Birth order (%)		
First born	47	44
Second born	33	37
Maternal education (%)		
Basic	27	23
Vocational	33	34
Technical/trade	24	25
High school	8	8
Graduate school	8	14
Single parent (%)	6	7
Housing type (%)		
Multiple dwelling	22	26
Row house	18	21
Single family detached	60	53
Mother current smoking (%)	29	28
Density (people/room, mean (SD))	0.85 (0.28)	1.04 (0.34)
Duration of residence (y, mean (SD))	10.23 (7.63)	9.96 (6.33)
Breastfeeding (months, mean (SD))	3.66 (4.74)	3.72 (5.35)
Noise exposure (dB,A,Ldn, mean (SD))	51.67 (7.69)	53.04 (10.47)
Mental health (self report, mean (SD))	93.06 (13.65)	92.84 (12.97)
School behaviour (teacher, mean (SD))	19.60 (2.54)	20.04 (2.26)

*Representative sample; †sampling on exposure extremes (<50 dB,A,Ldn; >60 dB,A,Ldn).

scale was reliable (Cronbach's α=0.80). Sample items included. In general is this child functioning as well as other children his or her own age? Is this child easily distracted during his or her work? The teacher was blind to each child's level of ambient noise exposure.

Statistical procedures

Exposure and survey data were linked through the geographical information system, and statistical analysis was conducted with SPSS 8.0 and S+4.5 including F Harrells' HMISC and DESIGN libraries. Multiple linear regression techniques were used and 95% confidence intervals (95% CIs) were calculated based on normal approximation. Based on existing knowledge, a hierarchical forward stepping procedure was applied.[23] We entered first a minimum set of standard risk factors (sex, maternal education, density, house type, biological risk) and noise exposure. Then further testing for the effects of other variables followed (breast feeding, lone parent, birth order, duration of residence). The most consistent and parsimonious set of variables across the four analyses was chosen to test for three prespecified interactions with noise exposure (biological risk, sex, education). No higher order interactions were uncovered. Finally, sensitivity analyses were conducted to test the stability of the estimates.[24]

Results

Psychological health

Table 2 describes the results for child self reported psychological health. In the population study there are significant main effects due to education, house type, household density, and a borderline effect for sex. While higher education is associated with better psychological health, all the other variables (household crowding, apartment block housing versus single detached housing, male sex) show a negative impact. The main finding of interest, however, is the significant interaction of early biological risk and ambient noise exposure on psychological health. Figure 3 shows the effect of noise exposure (adjusted for maternal education, sex, number of persons in household, and house type) on those with early biological risk whereas children without this risk seem unaffected by noise exposure.

Although the main effects are less consistent in the extreme noise exposure sample, the interaction is replicated and shows

Table 2 Multiple regression model of children's psychological health (self reporting scale) (comparison of the effects (mean differences (95 % confidence intervals)) in the two samples

	Population study*		Extreme exposure study†	
Coefficient	Mean difference‡ (95% CI)	p Value	Mean difference‡ (95% CI)	p Value
Maternal education (1–5)	3.96 (1.88 to 6.04)	0.0002	2.12 (–3.65 to 7.88)	0.4733
Sex (male-female)	–1.47 (–3.09 to 0.15)	0.0762	2.03 (–2.53 to 6.60)	0.3853
People in household (1–9)	–2.47 (–4.59 to –0.35)	0.0227	–0.55 (–5.65 to 4.55)	0.8330
House type (1–3)	–3.32 (–5.32 to –1.32)	0.0012	4.47 (–1.08 to 10.03)	0.1180
Biological risk (0–1)	–5.85 (–10.07 to –1.64)		–15.01 (–22.83 to –7.19)	
Noise exposure (30–75)	–2.77 (–5.46 to –0.08)		–16.90 (–28.37 to –5.42)	
Risk×noise		0.0344		0.0470
	R^2=0.04		R^2=0.23	

*Representative sample; †sampling on exposure extremes (<50 dB,A,Ldn; >60 dB,A,Ldn); ‡differences based on the following contrasts: maternal education (high school *v* basic), male *v* female, persons in household (6 *v* 3), house type (multiple dwelling *v* single family), interaction term: noise exposure=1 (65 dB,A,Ldn ; >60 dB,A,Ldn) and biological risk=1 (birth weight <2500 g or <37 weeks of gestation).

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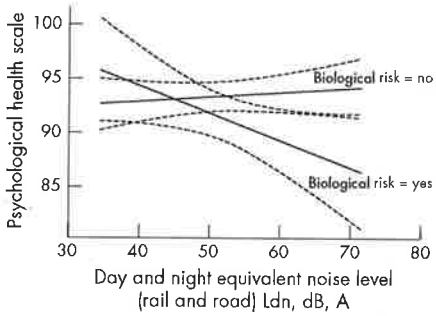

Figure 3 Dose-response (adjusted for maternal education, sex, number of people in household, and house type) for noise exposure and children's psychological health (self reported, based on the KINDL[21] and a sleeping scale) by early biological risk (birth weight <2500 g or <37 weeks of gestation) from a representative sample of a population study.

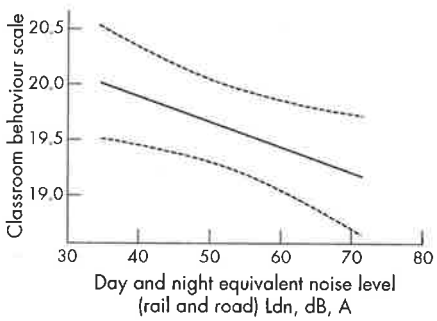

Figure 4 Dose-response (adjusted for maternal education, sex, number of people in household, and house type) for noise exposure and children's classroom adjustment (teacher rating scale, index of behavioural conduct[22]) from a representative sample of a population study.

large effects (table 3). Note, that the explained variance of the model increases to 23% in the extreme exposure group design.

Classroom adjustment

Table 4 presents the regression results for the second outcome measure—the teacher's rating of class room adjustment. In the population study no interaction was detected. However, significant main effects were evident for education, sex, house type, and exposure to noise, and biological risk was marginally significant. The direction of the effect was replicated for all factors, although household density did not reach significance. The total explained variance (R^2=0.09) was slightly higher than for psychological health as rated by the children (R^2=0.04). Figure 4 presents the adjusted dose-response pattern for the relation between noise and behaviour. A decrement in teacher ratings with increasing ambient noise was evident.

The model results for the extreme exposure analyses resemble the field findings, except that house type and household density reversed their significance. The model total R^2 increased to 0.21 from 0.09, again showing enhanced statistical power with the extreme exposure group design.

Discussion

Ambient levels of noise in the community are associated with decreased mental health in elementary school children. This association, a linear dose-response function in a large population study, holds with multiple statistical controls, replicates in two samples, and was robust across reliable and valid self reported measures and teacher ratings. Furthermore, children with low birth weight and preterm delivery may be at greater risk of noise related mental health outcomes.

Data from the population survey indicate that variations in typical community noise levels were associated with psychological symptoms and quality of life reported by the child, but only in those with a pre-existing biological risk (low birth weight, fig 3). This significant interaction was replicated in the extreme exposure study as shown in table 3. It is also interesting to note the evidence of a dose-response function for the high risk subsample in the general population study. For

Table 3 Children's psychological health*

	Biological risk§	
	No	Yes
Noise exposure <50 dB,A,Ldn‡	n=50	n=7
Mean (95% CI)†	96.2 (93.0 to 99.4)	94.6 (87.4 to 101.8)
Noise exposure >60 dB,A,Ldn‡	n=43	n=15
Mean (95% CI)†	92.8 (89.4 to 96.1)	80.9 (70.3 to 91.5)

*Self reporting scale; †adjusted for maternal education, sex, number of people in household, and house type; ‡sampling on exposure extremes (<50 dB,A,Ldn; >60 dB,A,Ldn); §birth weight <2500 g or <37 weeks of gestation.

Table 4 Multiple regression model of children's classroom adjustment (teacher-rating scale) (comparison of the effects of the differences (mean (95 % CIs)) between the two samples)

	Population study*		Extreme exposure study†	
Coefficient	Mean difference‡ (95% CI)	p Value	Mean difference‡ (95% CI)	p Value
Maternal education (1–5)	1.20 (0.82 to 1.58)	<0.0001	0.88 (–0.16 to 1.92)	0.1013
Sex (male/female)	–1.04 (–1.33 to –0.74)	<0.0001	–1.39 (–2.23 to –0.55)	0.0017
People in household (1–9)	–0.14 (–0.52 to 0.25)	0.4878	0.94 (0.02 to 1.87)	0.0489
House type (1–3)	–0.48 (–0.85 to –0.11)	0.0112	–0.04 (–1.07 to 1.00)	0.9472
Biological risk (0–1)	–0.33 (–0.71 to 0.05)	0.0855	–0.53 (–1.68 to 0.63)	0.3720
Noise exposure (30–75)	–0.58 (–1.06 to –0.09)	0.0196	1.11 (0.28 to 1.95)	0.0105
	R^2=0.09		R^2=0.21	

*Representative sample; †sampling on exposure extremes (<50 dB,A,Ldn; >60 dB,A,Ldn); ‡differences based on the following contrasts: maternal education (high school *v* basic), male *v* female, people in household (6 *v* 3), house type (multiple dwelling *v* single family), biological risk=1 (birth weight <2500 g or <37 weeks of gestation) *v* 0 (normal birth weight/length of gestation), noise exposure (65 *v* 40 dB,A,Ldn ; >60 *v* <50 dB,A,Ldn).

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teacher ratings of children's behavioural adjustment, both in the general population and the extreme community noise exposure comparisons (table 4), there were significant main effects of noise on behaviour. All these significant results incorporate multiple adjustments for individual and social factors.

Examining each of our objectives, we showed that typical fluctuations in ambient community noise are associated with mental health among children. This finding replicates the only previous study of noise and mental health among children which showed an association between exposure to airport noise and psychological distress in 8–11 year old children.[12] We extend this study by showing similar effects at lower, more typical levels of ambient noise, across two different measures of mental health in children, with a substantially larger array of statistical controls. We also provided the first evidence of a dose-response function between exposure to noise and mental health in children. Only two studies with adults have uncovered dose-response functions between those with high levels of transportation noise and mental health.[25 26] Thus the present study adds to the small amount of literature on noise and mental health in children and provides further evidence of a relation between noise and mental health.

As well as showing that everyday noise may have mental health consequences among children, we examined the potential moderating role of several biological and social risk factors. The data indicate that children who had a low birth weight or were born preterm may be more vulnerable to the adverse mental health consequences of exposure to ambient noise. These children could be more reactive to the harmful consequences of noise because of their known greater susceptibility to behavioural problems in early childhood[27–30] or because of physiological effects of "early programming".[31–34] The evidence for greater vulnerability to noise among children with low birth weight is mixed, however. This interaction was restricted to the standardised self reported measure of mental health symptoms. We did not find a similar interaction between noise and biological risk for teacher ratings of behavioural adjustment in the classroom. One possible explanation for this are differences in the symptoms assessed by the two indexes. The self reported symptom checklist focuses primarily on anxiety and depression, whereas the teacher ratings emphasise interpersonal social skills and emotional and attentional regulation in the classroom.

The data on psychological distress reported by the children also illustrate the potential value to public policy of examining moderator functions. In the overall population, there is little or no evidence of mental health sequelae of exposure to noise in the community. This could have led to the incorrect conclusion that therefore ambient levels of noise are irrelevant for mental health among children. As figure 3 and table 3 clearly show, such a conclusion would be incorrect. There is a subsample of children who are reliably at risk of poorer mental health relative to even the lower levels of ambient noise found in rural, central European communities. Furthermore as explained earlier, the uncovering of such an interaction prohibits use of the same variable (low birth weight) as a statistical control when examining the main effects of noise on mental health.

Another objective of our study was to show the potential use of a mixed research design, incorporating both a general population study and a smaller, more focused analysis of people exposed to extreme levels of the environmental risk factor. As a comparison of the results from the two samples shows, the evidence for adverse effects of noise is much stronger in the extreme level comparisons (compare the total R^2 in tables 2 and 4 for the two designs). Especially in the early stages of environmental risk investigations there is need for cost efficient designs[18 35] with high sensitivity to detect potential adverse environmental risk factors. The extreme exposure research design does have some limitations: it is not representative of the general population, and, relative to dose response evidence, is more subject to plausible rival hypotheses. None the less enhanced sensitivity to detect potential health effects, coupled with reduced costs and effort, make this type of design worthy of greater consideration in the field of environmental health. Ideally, investigators could combine both research designs in the same study.

It is important to reiterate that the cross sectional design of this study precludes drawing causal inferences. However, our data are stronger than a simple correlational study because of several features: (*a*) dose response function; (*b*) the replication and predicted stronger associations in the extreme exposure groups; (*c*) the use of multiple individual and social controls; (*d*) the predicted interaction between biological risk and noise. Nevertheless, unmeasured confounders and measurement error could bias the results.[36] Short of random assignment of children to different noise exposures, probably the best approach to strengthening the evidence for the casual effects of noise on children's mental health would be to conduct a prospective longitudinal study comparing the same child across different noise conditions. It is worth mentioning that the study by Bullinger *et al* of aircraft noise and children's mental health was prospective.[12]

This *Journal* and other sources document adverse physical health consequences of suboptimal environmental conditions, both in the workplace and at home.[37–41] Similar exploration of the mental health consequences of unhealthy social and environmental conditions is just beginning.[42 43] Population studies generating dose-response functions along with comparisons of groups with extreme environmental risk can assist us in this new area of environmental medicine and health. Due consideration of potential moderators is warranted for conceptual, statistical, and policy reasons, in physical and psychological morbidity studies of occupational and environmental health.[44–46]

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CALIFORNIA CRUDE OIL PRODUCTION AND IMPORTS

STAFF PAPER

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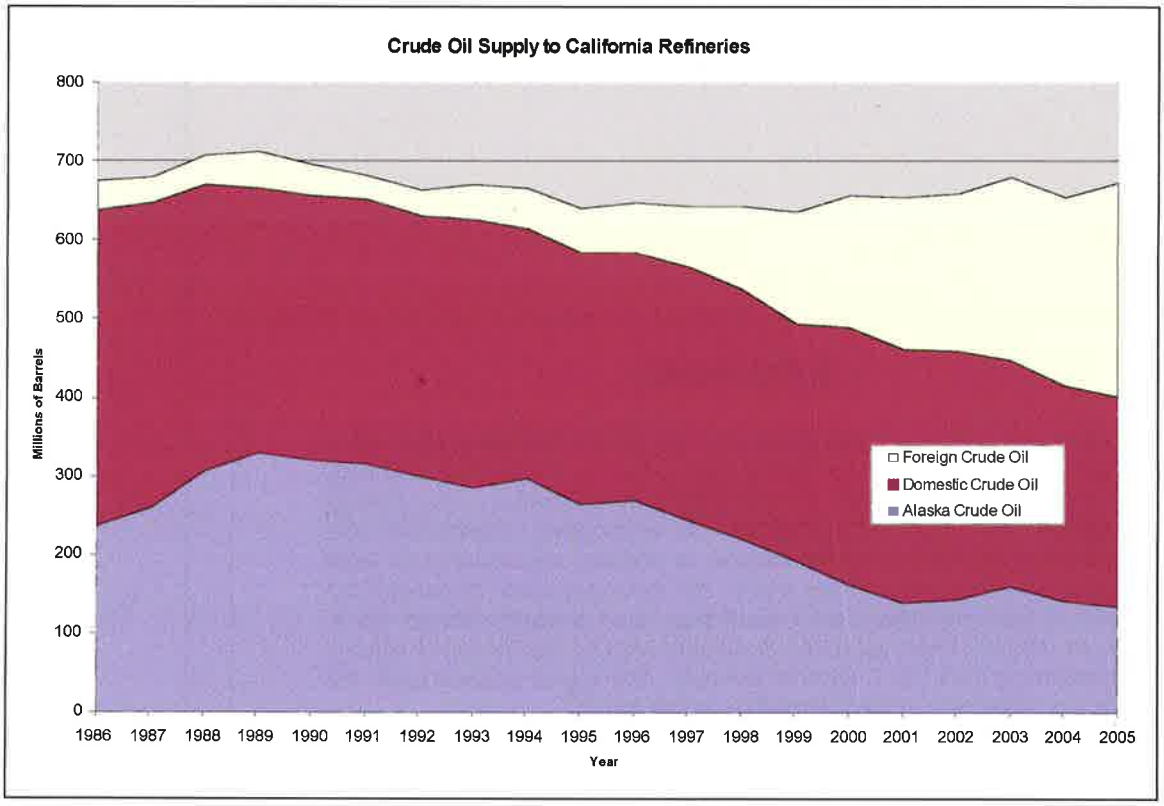
CALIFORNIA CRUDE OIL PRODUCTION AND IMPORTS

Introduction

Californians consume nearly 44 million gallons of gasoline and 10 million gallons of diesel every day.[1] California refineries produce these fuels and other products from crude oil and blending components. Transportation fuel production in California depends on the availability and quality of the crude oils used by refineries in the state. Figure 1 shows the average annual refinery receipts of crude oil from 1986 to 2005. The supply of crude oil to California refineries has changed substantially in the last 10 years. Most notably, receipts of foreign crude oil have increased as production sources from California and Alaska have continued to decline.

Figure 1



Source: Petroleum Industry Information Reporting Act

Historically, California has been relatively self-reliant in petroleum supplies. However, crude oil production in California has decreased by 23 percent since 1996.[2] This decline of supply in the state has increased reliance on foreign and domestic imports. Starting in 1994, California refineries received more imported

crude oil than in-state sources. In 2005, California crude oil accounted for approximately 37 percent of the total receipts.

The quality of the crude oil used by the refinery in conjunction with the complexity of processing units dictates the percentages of products produced. For example, lower quality crude oil is more difficult to refine into lighter products, such as motor and aviation gasoline. Refineries have minimum crude oil quality requirements that are determined by the processing units in the plant.

This paper presents information on crude oil characteristics, California crude oil production trends, and their possible impact on future transportation fuel production.

Crude Oil Characteristics

The quality of crude oil is determined by a number of characteristics that affect the proportions of transportation fuels and petroleum products produced when the oil is refined. The two most common measurements of crude oil quality are the specific gravity (which is measured in degrees) and the sulfur content of the oil. Acid content is also a factor in determining the corrosive properties of the crude oil entering the refinery.

Specific Gravity

The specific gravity is typically measured using the American Petroleum Institute (API) standard or the API gravity of the crude oil. The API gravity is the measure of the weight of crude oil in relation to the weight of water (water has an API gravity of 10 degrees). Crude oil is characterized as heavy, intermediate, or light with respect to its API gravity.

- Heavy Crude: Crude oils with API gravity of 18 degrees or less is characterized as heavy. The oil is viscous and resistant to flow, and tends to have a lower proportion of volatile components. Fifty one percent of California crude oil has an average API of 18 degrees or less.

- Intermediate Crude: Crude oils with an API greater than 18 and less than 36 degrees are referred to as intermediate. Forty eight percent of California crude oil has an average API between 18 and 36 degrees.

- Light Crude: Crude oils with an API gravity of 36 degrees or greater. Light crude oil produces a higher percentage of lighter, higher priced premium products.

Sulfur Content

Crude oil is defined as "sweet" if the sulfur content is 0.5 percent or less by weight and "sour" if the sulfur content is greater than 1.0 percent. Sulfur compounds in crude oil are chemically bonded to hydrocarbon molecules in the oil. Additional equipment in the refinery is required to remove the sulfur from crude oil, intermediate hydrocarbon feedstocks, and finished products. Transportation fuel specifications require extremely low sulfur contents, usually less than 80 parts per million (ppm).

Acid Content

Another characteristic of crude oil is the total acid number (TAN). The TAN represents a composite of acids present in the oil and is measured in milligrams (mg). A TAN number greater than 0.5 mg is considered high.[3] As an example, Wilmington and Kern crude oil have a TAN ranging from 2.2 to 3.2 mg, respectively.[4] However, some acids are relatively inert. Thus, the TAN number does not always represent the corrosive properties of the crude oil. Further, different acids will react at different temperatures – making it difficult to pinpoint the processing units within the refinery that will be affected by a particular high TAN crude oil. Nonetheless, high TAN crude oils contain naphthenic acids, a broad group of organic acids that are usually composed of carboxylic acid compounds. These acids corrode the distillation unit in the refinery and form sludge and gum which can block pipelines and pumps entering the refinery.[5]

The impact of corrosive, high TAN, crude oils can be overcome by blending higher and lower TAN oils, installing or retrofitting equipment with anticorrosive materials, or by developing low temperature catalytic decarboxylation processes using metal catalysts such as copper. Many California refineries already process high TAN crude. High TAN oils are sold on the market at a discount compared to higher quality crude oils.

High TAN oils account for an increasing percentage of the global crude oil market. Crude oil with a TAN greater than 1.0 mg increased in the world market from 7.5 percent in 1998 to 9.5 percent in 2003.[6]

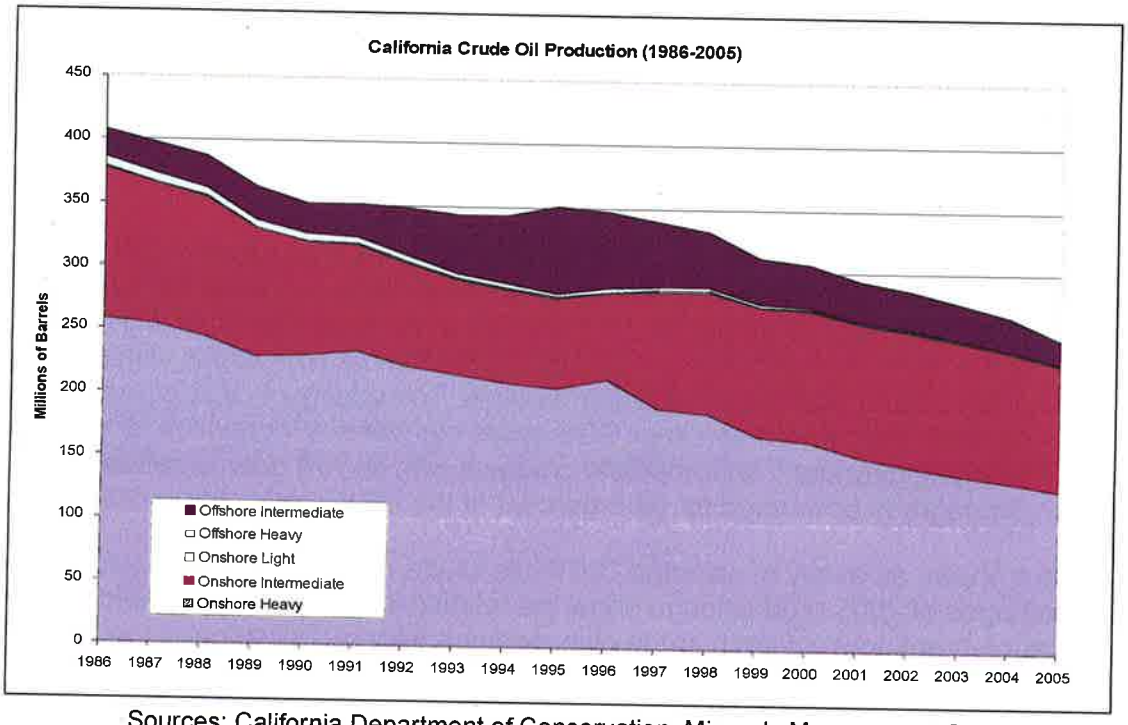
California Crude Oil Production

The discovery of oil in Kern County in the late 19th century heralded a long history of oil production in California. At the turn of the 20th century, crude oil was valued primarily for the heavier products and refining was oriented towards the production of heating oil and lubricants. In the early 1900s, with growing automobile use, gasoline became a more important commodity.

California is currently ranked fourth in the nation among oil producing states, behind Louisiana, Texas, and Alaska, respectively. Crude oil production in California averaged 731,150 barrels per day in 2004, a decline of 4.7 percent from 2003. Statewide oil production has declined to levels not seen since 1943.[7]

Figure 2 shows California onshore and offshore crude oil production over the last 20 years. The production of heavy, intermediate, and light crude oil production are broken out for onshore and offshore (or Outer Continental Shelf [OCS]) areas.

Figure 2



Sources: California Department of Conservation, Minerals Management Service

Production peaked in California in 1983. Production has declined at an average rate of 2.4 percent per year in the last 10 years.

Figure 2 shows a constant decline in onshore heavy crude oil production from 1986 through 2005 of 6.8 million barrels per year, or approximately 3.5 percent per year. Intermediate onshore oil production remained relatively flat. Offshore crude oil production peaked at 72 million barrels in 1995 and has declined by around 4.3 million barrels per year - or 10.2 percent per year - from 1995 through 2004.

The three major regions of California crude oil production are Kern County, the Los Angeles Basin, and the Outer Continental Shelf (OCS).

- **Kern County:** In 2004, oil from Kern County accounted for 77 percent of California's total onshore production and over 69 percent of the state's total oil production.[8] Approximately 58 percent of the crude oil has an API of 18 degrees or less. The Kern River oil field, located in the eastern San Joaquin Valley, accounts for approximately 24 percent of Kern County oil. Kern River oil is characteristically heavy and sour with an API of 13.4 degrees and a sulfur content of 1.2 percent.[9]

- **Los Angeles Basin:** The Los Angeles Basin is a sedimentary plain extending from central Los Angeles south through the Long Beach area. The two largest fields by area in this region are the Wilmington and the Huntington Beach fields with average APIs of 17.1 and 19.4 degrees, and average sulfur contents of 1.7 and 2.0 percent, respectively.

- **Outer Continental Shelf:**[10] The Federal Minerals Management Service oversees crude oil rigs located three nautical miles or greater from the coast. The OCS rigs accounted for 10.2 percent of the total California production in 2004. Many of these rigs are leased to commercial companies with pipelines extending to onshore processing facilities. The quality of OCS crude oil varies by field. Both sweet and sour OCS crude oils have API gravities ranging from 14 to 38 degrees.[11] Intermediate crude oil with an API gravity between 18 and 36 degrees accounted for 96.6 percent of the OCS production in 2004.

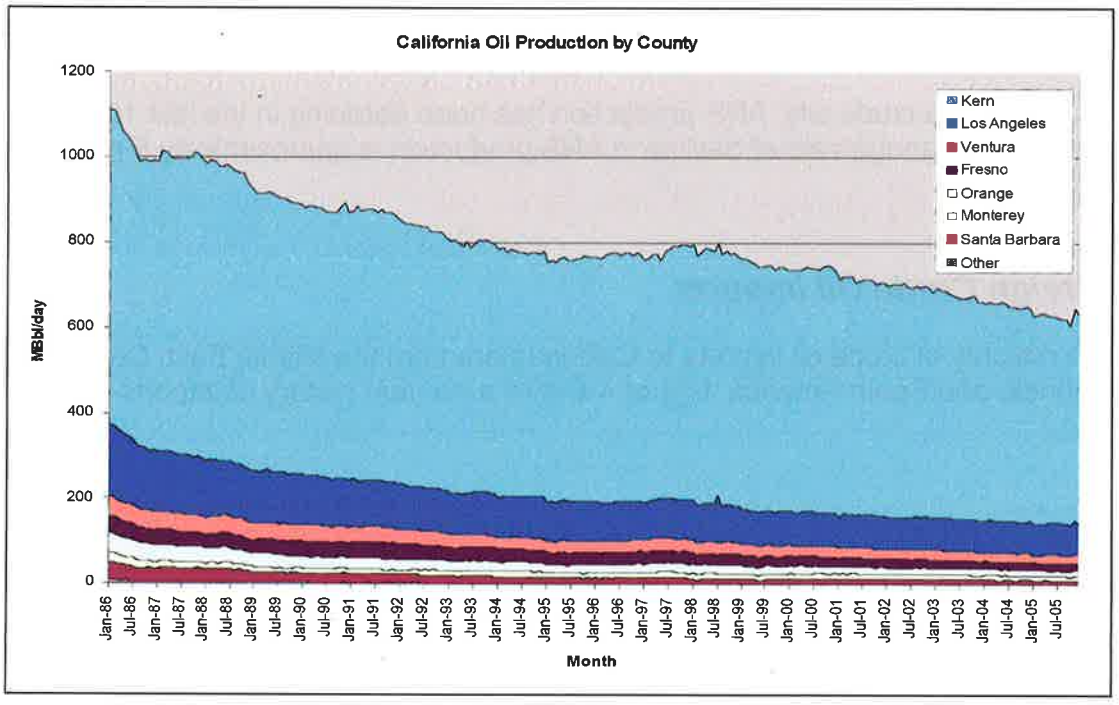
Table 1 shows an assay of selected California crude oils.[12] The table provides the percentages of 2005 production to show the relative importance of the field. The distillation breakdown of each crude oil provides a general guideline of the refining product suite that would result after the initial crude distillation has been completed. The actual ratio of finished refined products will vary depending on the complexity of the refinery. Note that unrecoverable gas losses occur in the assay, resulting in distillation product summations of less than 100 percent.

Table 1

County	Field	Percent of 2005 Production	API Gravity & Sulfur	Distillation breakdown (percent per volume) Total Gasoline & Naptha	Middle Distillates	Residuum	Lubes
Kern & San Luis Obispo	Midway Sunset	18.47%	12.6, 1.6%	0.00%	12.00%	50.30%	34.80%
Kern	Kern River	14.36%	13.3, 1.1%	0.00%	15.80%	56.10%	28.10%
Kern	Elk Hills	7.91%	34.6, 0.8%	34.30%	23.30%	25.00%	15.90%
Los Angeles	Wilmington	6.49%	17.1, 1.7%	9.50%	18.20%	52.80%	19.40%
Kern	Lost Hills	4.96%	18.4, 1.0%	7.60%	23.50%	42.70%	23.20%
Ventura	Ventura	1.75%	30.2, 1.0%	30.20%	20.80%	31.30%	16.30%
Kern	Belridge N. Lt.	1.63%	31.3, 0.3%	25.70%	25.70%	26.30%	20.90%
Monterey	San Ardo	1.52%	12.2, 2.3%	2.10%	14.50%	62.50%	20.50%
Los Angeles	Inglewood	1.24%	21.0, 1.8%	12.90%	27.60%	39.10%	19.40%
Orange	Huntington Beach	1.07%	19.4, 2.0%	12.00%	19.70%	48.90%	19.40%
Los Angeles	Long Beach	0.65%	25.0, 1.3%	18.90%	23.10%	40.60%	17.40%
Kern	Mount Poso	0.26%	16.0, 0.7%	0.00%	13.40%	52.00%	34.00%

Figure 3 shows the onshore production by county.

Figure 3



Source: Dept. of Conservation

California commonly uses Thermally Enhanced Oil Recovery (TEOR) techniques to help maintain crude oil production, because heavy, viscous crude oil requires heating to move the oil to the pump. Direct injection steaming and intermittent steaming are two types of TEOR. California crude oil production is also enhanced by injection of water (water flooding) and even carbon dioxide (CO_2) to help maintain sufficient pressure in the crude oil field. In the absence of more aggressive use of TEOR, California's crude oil production is expected to continue to decline at a rate of 3.5 percent per year through 2019.[13]

Well activity provides an indication of potential production in the state. In 2004, drilling increased to 2,451 wells, a 6.7 percent increase from 2003. The number of plugged wells decreased to 2,039 from 2,501 in 2003. Drilling and plugging activities in the state have fluctuated by more than 900 wells from year to year; however, the general trend is relatively flat.

Alaska North Slope Crude Oil

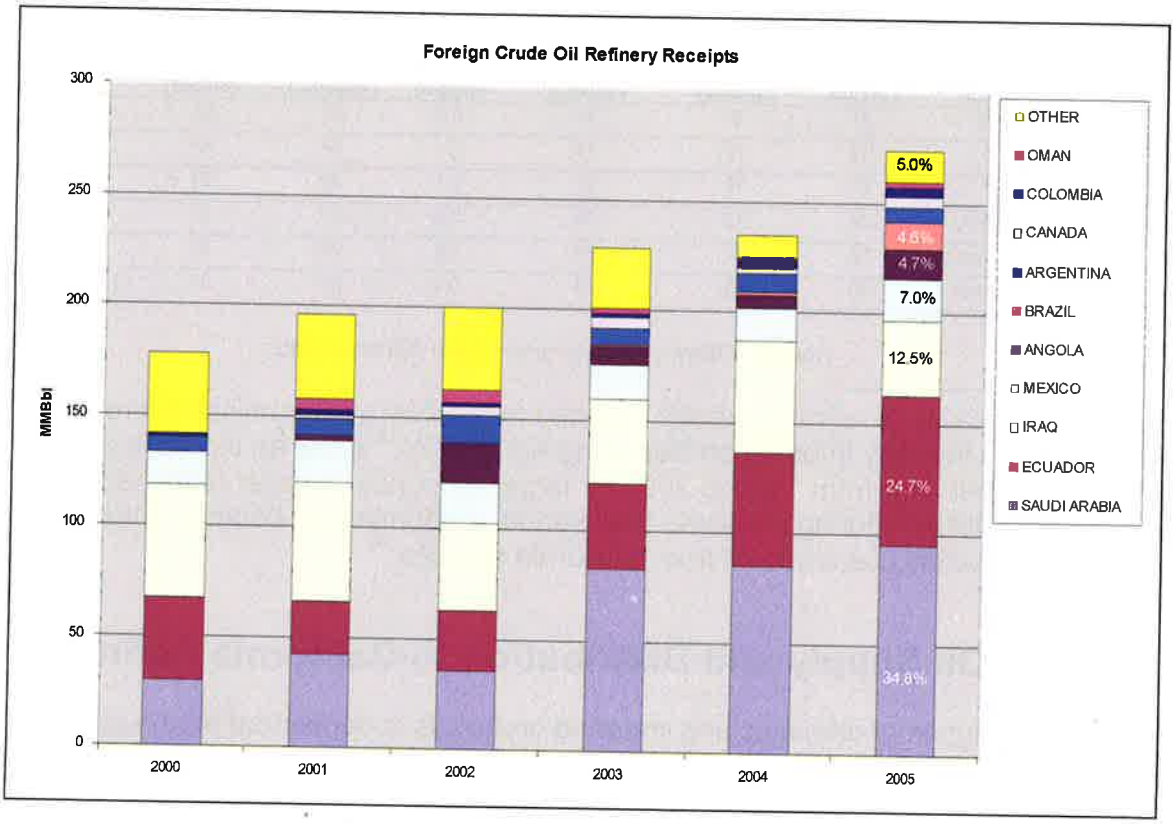
In 2005, California imported 21 percent of its total crude oil supply from Alaska. Oil fields in Alaska's North Slope produce a wide range of crude oils. API gravities from different fields range from 22 to 40 degrees. Alaskan refineries located along the Trans Alaska Pipeline System (TAPS) "top" the crude oil to produce light petroleum products and return residual products to the line. The resulting blended crude oil stream is referred to as Alaska North Slope oil (ANS). The ANS is an intermediate sour crude with an average API gravity of 29-29.5 degrees and sulfur content of 1.1 percent.

Like California crude oils, ANS production has been declining in the last 10 years. The average annual rate of decline in ANS production is approximately 5 percent per year.

Foreign Crude Oil Imports

The majority of crude oil imports to California are from the Middle East, Central America, and South America. Figure 4 shows a six year history of imports by region.

Figure 4



Source: Energy Information Administration

Crude oil imported from countries with volatile political and social structures leaves California vulnerable to changing world events. For example, attacks on Nigerian oil industry personnel led to the recent shutdown of nearly 9 percent of Nigeria's total oil production, which could impact global oil availability and increase feedstock costs for California refineries. Also, the growing political tension between the U.S. and Iranian governments over Iran's nuclear program could impact California's crude oil supply if the U.S. decides to impose sanctions on Iran.

Table 2 shows approximate crude oil characteristics for several imported crude oils.[14]

Table 2

Crude source	Paraffins Percent Volume)	Aromatics (Percent Volume)	Naphthenes (Percent Volume)	Sulfur (Percent Weight)	API gravity (Approx.)	Napht. yield (Percent Volume)	Octane No. (Typical)
Nigerian - Light	37	9	54	0.2	36	28	60
Saudi - Light	63	19	18	2	34	22	40
Saudi - Heavy	60	15	25	2.1	28	23	35
Venezuela - Light	35	12	53	2.3	30	2	60
Venezuela - Heavy	52	14	34	1.5	24	18	50
North Sea - Brent	50	16	34	0.4	37	31	50

Source: Office of Safety and Health Administration

The API gravity of refinery imports reported to the Energy Commission through the Petroleum Industry Information Reporting Act (PIIRA)[15] show an increase of 0.27 API per year from 1996 to 2005 for larger refineries. Smaller refineries show a relatively flat API during the same time period, predominantly because these smaller refineries solely use crude oil from California sources.[16]

Crude Oil Supply and Distribution to California Refineries

The distribution of domestic and imported crude oils is dependent on the port, pipeline, truck, and rail transport infrastructure within the state. All ANS and imported crude oils enter the state through ports in Los Angeles, Long Beach, and the Bay Area.

Water depth limits access to Bay Area ports. The water depth of these ports is typically between 32 to 45 feet, which is too shallow for large crude oil carriers. As an example, a carrier with a capacity of 1.3 million barrels will require a minimum water depth of at least 66 feet. For shallower ports, large vessels will anchor in a designated zone outside of the ports and smaller barges will transfer oil to the ports, a practice referred to as "lightering." This practice adds to the delivery cost of crude oil to the refinery and increases the risk of accidental release of crude oil into the environment.

Another complication for the Bay Area ports is silting in the bays. Dredging of the bays is controversial in that habitat is disturbed and dredged material must be disposed of in an environmentally sound manner. For example, approximately 4 million cubic yards of sediment are dredged from the Central and South Bay per year.[17]

Pipeline networks tie the San Joaquin Valley crude oil production with refineries in both the Los Angeles and the Bay Area. Figure 5 shows the major crude oil pipelines in California.

Figure 5

Source: California Energy Commission

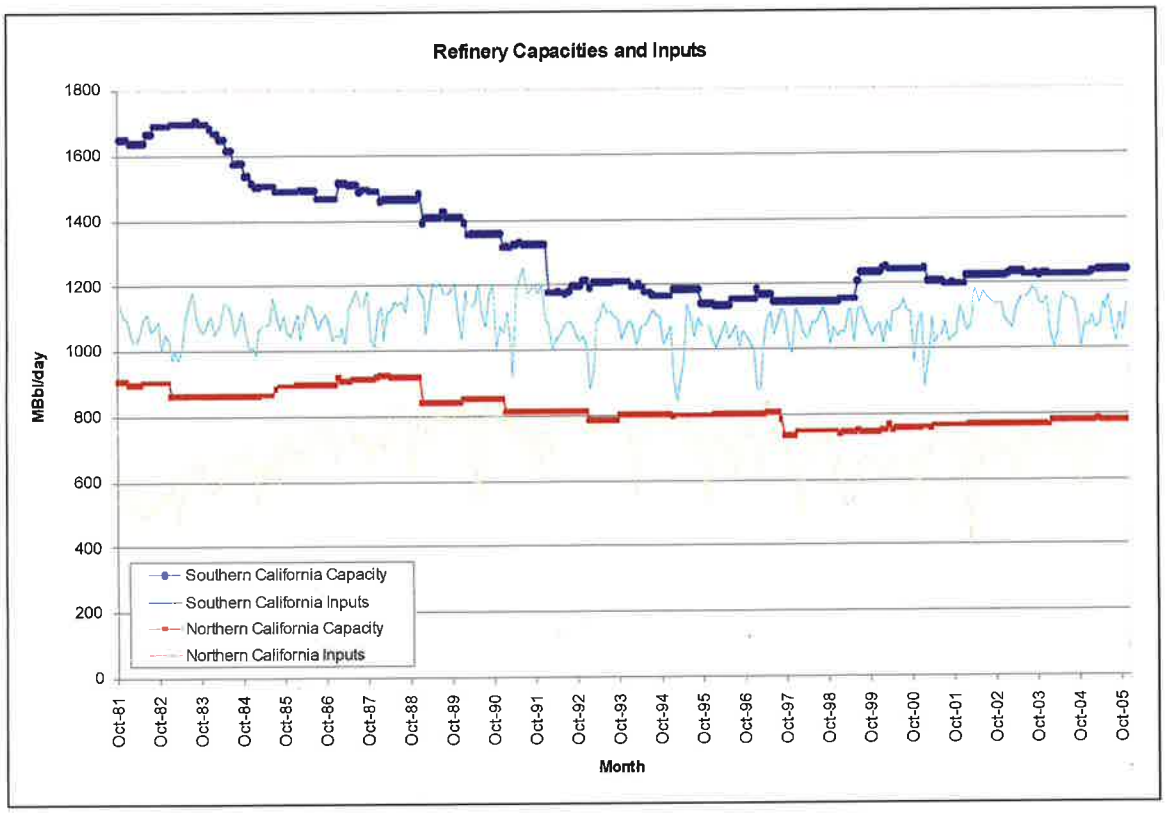
In California, 51 percent of the crude oil produced in the state is heavy crude. The transport of heavy crude through pipelines is complicated by the viscosity and inertia of the oil. Thus, some of the crude oil pipeline systems throughout the state require external heating. Booster stations are placed at intervals on the line where heating and/or pumping units facilitate the flow of the crude through the line. The proximity of booster stations is determined by the viscosity of the crude and by the average heat loss from the pipes from ambient weather conditions. Heavier crude oils are also blended with lighter crude oils to reduce viscosity, allowing transportation through pipelines without any heating.

Inland California crude oils are typically first piped to local refineries (Bakersfield and Santa Maria) because they are nearby and do not have port access. The balance of inland crude oils are piped to Northern and Southern California refineries.

Refinery Operations

In the last two decades, California refineries have been running increasingly closer to capacity levels. Figure 6 shows the total crude oil throughput refining capacity and the throughput oil inputs to the refinery by area.

Figure 6



Source: Petroleum Industry Information Reporting Act

The steady decline in refinery capacity during the 1980s and early 1990s is followed by a noticeable creep upward in the late 1990s and early 2000s. With refinery creep and greater import capabilities in the Los Angeles area, southern refineries are less constrained than their Bay Area and Central California counterparts. Southern California refineries also show an increasing level of crude oil imports.

Refinery operations must also consider recent diesel regulations by the U.S. Environmental Protection Agency (EPA) and the California Air Resources

Board (ARB). The EPA regulation lowers the allowable amount of sulfur in on-road diesel fuel from less than 500 parts per million (ppm) to less than 15 ppm. This requirement will take effect on June 1, 2006. The sulfur content and API gravity of crude oil input to the refinery in conjunction with the complexity of process units will affect the quantity of ultra-low sulfur diesel produced by the facility.

The hydrocracking and hydrotreater units remove sulfur within the refinery. Hydrocracking units break hydrocarbon molecules into lighter compounds in the presence of hydrogen. Hydrotreatment involves the chemical reaction of hydrocarbon compounds with hydrogen in the presence of a catalyst such as cobalt or alumina.[18]

Refineries throughout the U.S. are currently upgrading their desulfurization processes in order to meet the new diesel sulfur standards. This upgrade typically involves techniques such as changing the catalyst in the hydrotreater or installing booster pumps to force more feedstock through the unit. Both hydrocrackers and hydrotreaters also remove heavy metals and aromatics from the feedstock. This is particularly important in California where lower aromatic standards will be required along with the new ultra low sulfur diesel standards.

Findings

- The declining crude oil production in South-Central California has resulted in higher crude oil costs because of reliance on higher priced imported crude oils.

- Pipeline utilization rates are decreasing and the procurement of crude oil to inland refineries is becoming increasingly difficult as local supplies decline.

Current and Future Work

Additional reporting requirements in the Energy Commission's new petroleum industry data collection regulations will greatly enhance the agency's understanding of crude oil and finished product movements within the state. The addition of port, terminal, and pipeline information will provide the details needed to track infrastructure use within the state. This additional information will be essential in: assessing near-term petroleum infrastructure demand shifts, reviewing project expansion plans, and completing contingency studies.

Research and analysis should focus, in particular, on the following areas:

- Crude oil quality: The growing dependence of California refineries on imported crude oils requires a more detailed look at the characteristics of overseas crude oils entering ports in the state. The general trend of

international crude oil production reflects an increase in low API, high sulfur content crude oil. However, overall API gravity in California refineries has increased primarily from the decline in heavy California crude oil production. The examination of supply information from secondary sources and from PIIRA reporting data will help to identify areas of constraint in the state.

- Total Acid Number (TAN): The increase in world production of heavy, sour, and high TAN crude oils will impact California refineries. An assessment of the crude oil processing capabilities of California refineries is needed to understand the potential implications of future changes in the global crude oil market.

- Crude oil pipelines: The decrease in crude oil production in the state has led to changes in the utilization rates of some crude oil pipelines. Modifying current pipeline systems and/or making new investments in distribution infrastructure may be necessary to provide more stable sources of crude oil for refineries without port access.

Endnotes

[1] California State Board of Equalization data for 2004. Taxable gasoline figures amounted to an average of 43.5 million gallons per day, while taxable diesel fuel sales figures have been adjusted upward to reflect an estimated 22 percent distribution of exempt and refund diesel sales that are excluded from their taxable gallons.

[2] Based on data compiled from the California Department of Conservation database production files, http://www.conservation.ca.gov/DOG/prod_injection_db/index.htm and MMS Offshore data, http://www.gomr.mms.gov/homepg/pubinfo/pacificfreeasci/product/pacificfreeprod.html.

[3] http://rru.worldbank.org/Documents/publicpolicyjournal/275-bacon-tordo.pdf.

[4] http://www.pacificenergypier400.info/pdfs/CRUDESUP/PACIFICP.PDF.

[5] http://www.ornl.gov/sci/fossil/Publications/RECENT%20PUBS/DDSum2003.pdf.

[6] Anne Shafizadeh, Gregg McAteer, and John Sigmon, *High-Acid Crudes*, paper presented at Crude Oil Quality Group meeting, New Orleans, January 30, 2003, [http://www.cogg.org/20030130special.asp]

[7] ftp://ftp.consrv.ca.gov/pub/oil/annual_reports/2004/PR06_Annual_2004.pdf.

[8] California Department of Conservation database production files, http://www.conservation.ca.gov/DOG/prod_injection_db/index.htm.

[9] Van Vector, Samuel, *Pricing Royalty Crude Oil*, http://www.econ.com/apijan00.pdf.

[10] MMS data for 2004 is approximately 95 percent complete. December 2005 data not yet posted.

[11] Jokuty, P.; Whiticar, S.; Wang, Z.; Fieldhouse, B.; and Fingas, M.; *A Catalogue of Crude Oil and Oil Product Properties for the Pacific Region,* 264p 1999.

[12] http://www.econ.com/apijan00.pdf.

[13] http://www.energy.ca.gov/2005_energypolicy/documents/2005-0516_workshop/presentations/Baker%20&%20OBrien%20Presentation%205-16-05.pdf.

[14] OSHA Technical Manual – Section IV: Chapter 2, http://www.osha-slc.gov/dts/osta/otm/otm_iv/otm_iv_2.html.

[15] PIIRA: the Petroleum Industry Information Reporting Act, Public Resources Code 25350 et seq.

[16] Large and small refineries are defined here as refineries with crude oil receipts in 2005 greater than or less than 5 percent of the total for the state, respectively.

[17] http://www.spn.usace.army.mil/ltms/chapter2.pdf.

[18] http://www.bp.com/genericarticle.do?categoryId=2013107&contentId=2019673.

Global Trade Impacts: Addressing the Health, Social and Environmental Consequences of Moving International Freight through Our Communities

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Andrea Hricko
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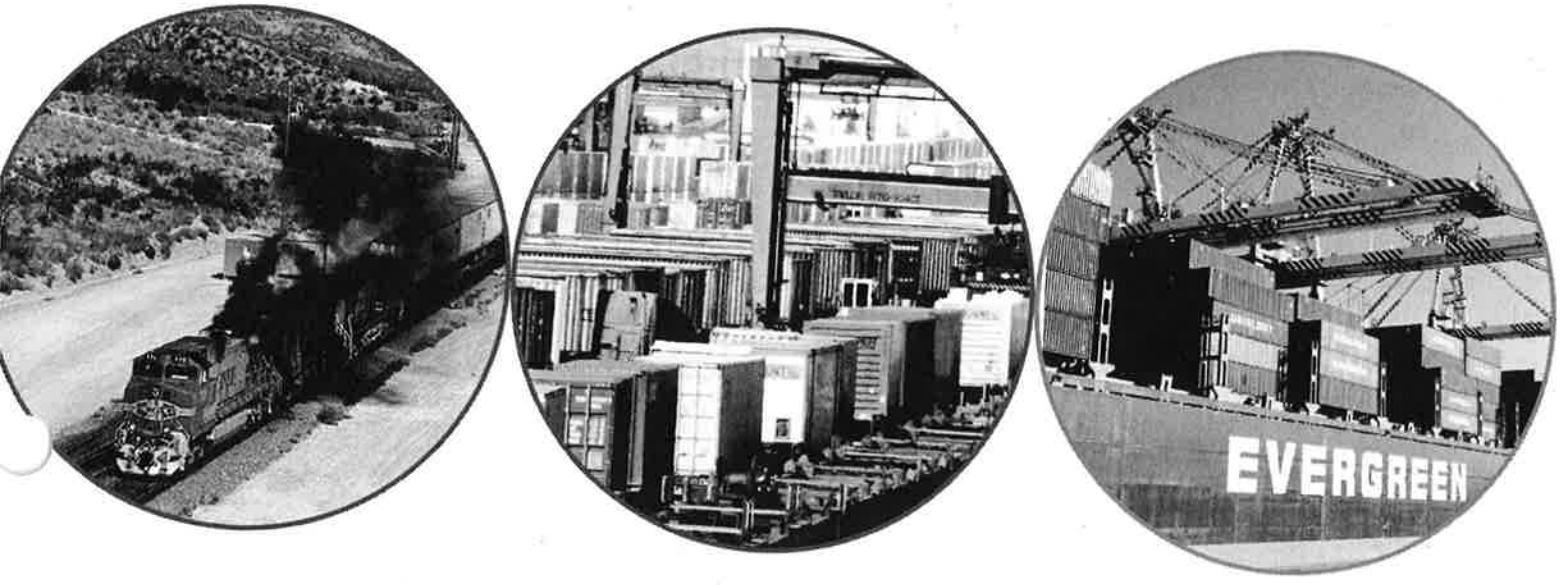
March 2011



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Globalized World, Goods Movement Impacts: An Introduction

We live today in a globalized world where products that were once manufactured locally are now being produced and delivered to us from the other side of the world. In the United States, this globalized system means that more and more products are made overseas due to lower labor costs and more lenient environmental regulations. With this shift in production, a global trade and freight transportation system has emerged to facilitate the movement of goods from the point of production to the point of consumption.

This vast and expanding network of seaports, highways, rail, distribution centers, and other cargo facilities increasingly define and impact the communities where they intersect. A growing array of ships, airplanes, trains and trucks is responsible for moving the raw materials, component parts and finished consumer products that form the backbone of the U.S. economy. These vehicles deliver their freight to a wide assortment of "goods movement" facilities that include seaports, airports, ports of entry (border crossings), rail yard and rail lines, highways, high truck traffic roads, warehouses, distribution centers, and a growing number of inland ports.[1] This distribution and logistics system, heavily influenced by the expansion of global trade, plays a central role in the evolution of global economies and its attendant impact on communities, jobs, the environment, and health.[2]

Throughout the globalization process, cheap labor combined with lax regulations enabled China, for one, to expand manufacturing production during the same period that the United States experienced a decline in manufacturing. With the reduction in U.S. factory jobs, many national, state, and local policy makers began to view global trade and goods movement industries as an economic driver that could replace millions of blue-collar manufacturing jobs lost after the 1980s. However, the shift from the United States as a producer nation to a consumer nation reliant upon distribution systems has had fundamental negative impacts on worker and neighborhood health, security and safety. Many of the new jobs created have been low-paying, with unhealthy working conditions, including use of a contingent workforce[3] of temporary workers (or "temps") who have fewer benefits and far less job security. This is particularly the case with the massive distribution centers that constitute a key part of the freight transportation system.

To facilitate such a shift, legislators have directed public dollars to subsidies and infrastructure projects (e.g., new or expanded highways, bridges, or dredging of rivers) as a means to lure business investment related to global trade. Thus, although international trade has generated new jobs, they have been accompanied by a visible price tag, including negative environmental, health, labor, and community consequences and an economic development scenario that encourages the growth of a low wage workforce.

Negative environmental impacts include air and water pollution from major shipping and freight transportation activities, with nearby community residents (or marine life, in the case of ships) exposed to diesel particulate matter and noise. The oceans are also impacted by the release of sewage and ballast water (water used to balance the ship) and the ballast water, taken onto a ship in one part of the world and released in another, can carry invasive species dangerous to plant and marine life.[4] Research findings now link air pollution to cardiovascular, respiratory and other health problems, and link noise pollution to cardiovascular illness, sleep difficulty, and anxiety. Studies show that living in close proximity to traffic-related pollution and noise brings additional risks. Air pollution and noise also affect the health of dock and warehouse workers, truck drivers, and railroad employees. Workers in distribution centers that usually have no air-conditioning also face heat stress in the warmer months, and there is a higher than average worker fatality rate. New studies also point to problems of 24-hour lighting at port and rail operations, conflicts involving incompatible land uses, the potential for contamination from hazardous spills, traffic safety problems, and hefty local costs to repair streets that are damaged by big-rig trucks. At the global level, international trade activities contribute to global warming, with significant emissions of carbon dioxide, black carbon and other pollutants. Despite the ever-growing evidence about the nature of these widespread health and workplace issues, negative health, environmental justice and labor impacts have not been widely incorporated into policy decisions.

Decisions by global retail chains such as Walmart determine how and where goods are moved. Without a national industrial policy or an effective global regulatory system to ensure that health, community, environmental and labor considerations become incorporated into such decisions, economic and political forces aligned with powerful industry advocates are able to frame the nature of the debates. As a result, the dominant narrative promoted by the freight transportation industry and most government agencies makes it appear that the goods movement system creates new jobs and cheap goods, while the downside of the shift of jobs from production to distribution and the negative external costs of the system are ignored or minimized.[5]

Despite these dominant growth and development agendas, environmental justice and community groups, health and environmental advocates, and labor unions and worker organizations have effectively challenged these agendas, and in some cases, shifted the nature of the debates. The report documents where and how this is happening, with one starting point identifying some of the steps that enable a product made in Asia (in this case, a doll) to eventually get into the hands of a child who lives near Chicago. (See box below)

The doll's itinerary is not unusual when goods made in China are shipped to the United States for sale by U.S. retailers. Today, nearly half of all imported goods sold in Chicago take a route like this from factories in Asia through Southern California ports before heading east. In fact, more than 200,000 containers of toys arrive from Asia every year, just at the Ports of Los Angeles and Long Beach.[10]

But this is not the only possible shipping route. Increasingly, new trade and freight transportation routes are expanding in port communities along the East Coast, such as Savannah GA, Norfolk VA, and Charleston SC, linked to the scheduled expansion of the Panama Canal that will enable increased size and numbers of container ships from Asia to access U.S. ports. As a consequence, a complex system of distribution centers, intermodal rail facilities, inland ports, and other logistics industry locations are expanding throughout the United States. Additionally, the doll's route illustrates the negative potential impacts on health, the environment, communities and workers. Transportation experts refer to the impacts from our doll's journey as "externalities," but to community residents and workers they are everyday factors that can directly harm health and quality of life.[11]

The health, community and other impacts that are the focus of this report fail to be depicted in an animated video by one of the country's major railroads on how goods are moved from China to the United States In "How Tomorrow Moves," by an East Coast railroad company, the freight transportation system seems simple and problem-free. See http://www.youtube.com/user/HowTomorrowMoves. Any negative impacts are invisible.[12] Our report, on the other hand,

A Doll's Journey: from China to Chicago[6]

To illustrate the global reach and local impacts of goods movement, one could follow the retail cycle of a toy doll. That doll - perhaps with a retail price of $9.97 - is an example of how the products we consume today travel long pathways from manufacturer to consumer. The dolls are likely to be made and assembled in China by workers who earn low wages and often toil under hazardous conditions.

Thousands of dolls are packed into a container and loaded by Chinese dockworkers onto a marine vessel that may be carrying a total of 4,000, 8,000 or even 13,000 + other containers filled with toys, shoes, furniture and electronics. Fueled by low-quality, high polluting bunker fuel which might have been shipped to Asia from Venezuela,[7] the ship leaves one of the world's largest ports in China, and chugs across the Pacific, discharging nitrogen oxides, sulfur oxides, particulates, black carbon and other pollutants into the earth's environment.

Arriving at the Southern California ports of Los Angeles or Long Beach (where more than 40% of all U.S. imports arrive), the container is unloaded by dock workers, who breathe exhaust from the idling ship as well as diesel emissions from a row of idling trucks, while drivers, who also breathe that exhaust, wait for their loads.

A trucker then drives the container to a nearby rail yard, located just 400 feet from an elementary school and homes, where it is loaded onto a freight train, pulled by several diesel locomotives. The container proceeds on its journey to a major retailer's distribution center hundreds (or several thousand) miles away, such as to a suburb of Chicago.

Alternatively, the container is placed onto big-rig trucks at the Los Angeles docks and sent for repackaging to a regional mega-warehouse (distribution center) some 50 miles inland from the ports. In Southern California, land that formerly had cows and dairies has given way to million-square-foot warehouses for consumer goods, drawing thousands of diesel trucks a day into formerly rural communities.

Near the distribution center, neighboring residents and truck drivers breathe air that contains diesel exhaust as truckers drive on residential streets to deliver their goods and unload their containers, including the one with the dolls. The workers often work for "temp" agencies rather than as employees of the distribution center itself, and face hazards on their job, such as heat stress from lack of air conditioning inside the warehouses.

Once repackaged, the dolls are trucked to a rail yard and reloaded onto a train that travels cross-country to an intermodal[8] rail/distribution facility outside of Chicago. There the container is put onto another truck, and it travels to its final retail destination, a big-box store in suburban Chicago. By this time, the dolls have journeyed more than 8,000 miles on diesel-burning conveyances.[9] This distance does not include the nautical miles and impact of initially transporting the ships' bunker fuel from Venezuela to China.

focuses on the hidden and not-so-hidden negative impacts on workers, community, and the globe.

As ports and goods movement activity expands throughout the United States, a major challenge is how to make the adverse impacts of freight transportation a more central part of economic development, policy and planning discussions and transportation decision-making.[13] On a human level, the challenge entails how to make the American public and policymakers understand that what appears like a streamlined system of moving goods from another continent to the United States, and from a U.S. port to a retail store, is actually anything but simple, and costs the public much more than the shipping, trucking, railroad, road-building and retail industries usually tell the public. In fact, billions of dollars of public funds are being spent to expand the country's infrastructure to accommodate ever-larger ships and ever-increasing volumes of imported containers. In addition, as the American economy continues to shift from manufacturing to a system of distributing products, major social, environmental, economic, and human health costs are created throughout the system.

A National Landscape Report: Impacts, Trends, and Organizing Responses

In 2009, faculty and staff from the Urban & Environmental Policy Institute of Occidental College and from the environmental health sciences and regional equity programs of the University of Southern California (USC) began a study of this evolving global trade and freight transportation system, focusing on areas in the United States where the system is expanding and where community, labor and social justice groups have begun to challenge the system. Funded by The Kresge Foundation, the purpose of the study - which resulted in this report - was to provide an overview of the growth and scale of the goods movement industries and the shift from a production to a distribution economy. At the same time, the study documents examples of organizing and policy approaches that have injected important considerations of health, labor, and community impacts into decision-making and identified new directions so that local and regional communities can better address what is happening in their backyards due to these shifts.

The primary authors of this report have actively participated in local and regional collaborations in Southern California and have collaborated with groups in California and across the country on issues of health, environmental justice, ports and freight transportation, and other social justice efforts. Through their involvement (along with others) in organizing the "Moving Forward Together" conferences in 2007 and 2010, the authors have begun to work with a range of organizations, many of which are referenced in this report. See inset.

Moving Forward Together National Conferences

Organized by the Southern California Environmental Health Sciences Center based at USC and THE Impact Project (a community-academic collaborative), the "Moving Forward Together" conferences have enabled local organizations across the country to meet, share their experiences and concerns about the adverse health, community and labor impacts of global trade and freight transportation and develop a platform for action and solutions.

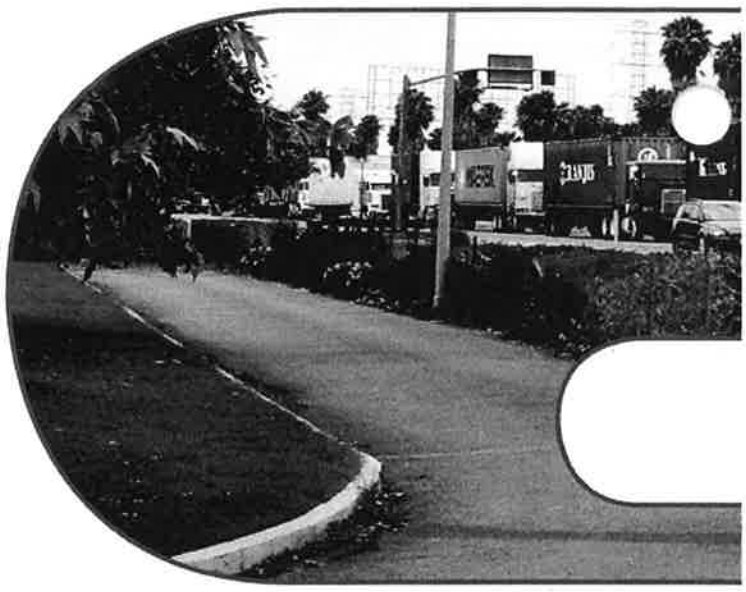
The first conference in 2007 drew 550 attendees from 16 states and four countries; three years later in October 2010, more than 600 participants from port, warehouse, and rail communities all over the United States and six other countries attended. See http://www.TheImpactProject.org.

Building on their research and experience, the authors identified 17 areas to study, including the locations of the top 10 ports in the continental United States, ports and hubs where rapid expansion and growth are occurring, and places where community efforts to address port, rail and warehouse pollution are taking place. Anchored by major seaports, goods movement relies on rail yards that are often in inland hub areas. We have included several of these hubs in the report, including the Inland Valleys of San Bernardino and Riverside Counties in Southern California, Detroit, Chicago, and Kansas City. Although we did not profile Dallas and regions in Ohio (e.g., Columbus) where new intermodal activity is concentrating, we discuss the importance of these goods movement hubs in our discussion of the selected profile regions. New rail intermodal hubs have been proposed even in agricultural areas, such as eastern Tennessee and the Central Valley of California.

The individual port and distribution hub locations discussed in the report include: Los Angeles and Long Beach, CA; New York and New Jersey, NY/NJ; Savannah, GA; Oakland, CA; Houston, TX; Norfolk/ Hampton Roads, VA; Seattle, WA; Tacoma, WA; Charleston, SC; Miami and Port Everglades, FL; Jacksonville, FL; Baltimore, MD; San Diego, CA; Detroit, MI; Riverside and San Bernardino, CA; Chicago, IL; Stockton, CA; and metropolitan Kansas City and the surrounding area. See Figure 1 next page.

For each region, this report analyzes facilities and trends in goods movement in a regional and economic

context and describes organizations and the strategies they use to address ports and freight transportation issues and solutions. We conducted research through telephone interviews, review of news articles, surveys of Port websites and Port Association information, reviews of published economic and business literature on trade and commerce and the use of U.S. Census, American Community Survey data, and the United States Bureau of Labor Statistics (BLS), Quarterly Census of Employment and Wages (QCEW). In addition, the authors conducted a review of research findings on noise, air pollution and other health impacts.



The report is organized into three sections. Part I documents the system and its trends: the continuing dominance of Asian imports, particularly from China; the impact of the expansion of the Panama Canal scheduled for completion in 2014; the growth of inland ports, intermodal facilities and related infrastructure developments; the consolidation of rail and highway expansion projects; and the role of rail and truck transport in moving goods from ports and freight facilities to their end points. The report draws on the research findings in the literature about the negative impacts freight transportation has had on health, community, the environment, and labor as well as the economic, political, and policy dimensions associated with those trends. Part II provides a snapshot of the different regions where the goods movement system will be expanding and where new movements are emerging to address system impacts. The authors point to examples of strategies that involve sharing information on health research findings, education, community organizing, legal advocacy, research, and coalition building that have informed and engaged community residents, workers, and environmental, health and labor advocates to focus on goods movement issues, public policy and decision-making. Examples of policies are provided that represent levers to ensure that health, labor, community, and environmental conditions are addressed in freight transportation planning and decision-making. In Part III, we discuss efforts to advance and build the capacity of the groups such as organizing and policy innovations that have effectively changed how the global trade and freight transportation movement issues are being framed and how decisions are being made. The recommendations reflect an urgency to "scale up" - in both knowledge about negative impacts of goods movement and in the organizing and policymaking capacity to shift debates and decision-making at the regional, national and international levels.

Figure 1. Top 10 U.S. Ports and Regional Hubs Profiled

Source: 2009 imports data from Journal of Commerce, Peter T. Leach, May 3, 2010, "Down But Not Out: North American Port Rankings Reveal Depth of Downturn, but Also Several Success Stories," p. 22
Map by Justin Seoggins at the USC Program for Environmental and Regional Equity (PERE)

Part I: Trends and Issues

Trends in the Freight Transportation Industry

The expansion of global trade has transformed the U.S. economy during the last several decades. Millions of manufacturing jobs have disappeared as companies moved their operations to countries with cheaper labor and weaker regulations. Meanwhile, U.S. consumption has grown to record levels as imported goods from China and other foreign countries have replaced American-made products on the country's retail shelves. Rising consumption and a growing import-based economy have required an extensive transportation network capable of delivering just-in-time high-demand consumer goods from Asian places like Guangdong, China, to U.S. locations like Kansas City. In the United States, this has led to an expansion in port-related infrastructure in both established and emerging gateways. Figure 2 on the next page shows how the booming import-based economy during the mid-2000s led to rapid growth in a wide range of U.S. ports including Long Beach, Seattle, and Savannah. A shift in the geography of where products are made and where they are consumed has created a new set of relationships between global economic development, commodity distribution networks, and environmental health. This section spells out some of the key trends that are taking place in that system.

Figure 3 on the next page shows the location and volume of imports at the top U.S. container ports. Half of the top 10 ports are on the West Coast; four are on the East Coast, and one is on the Gulf Coast.

Dominance of East Asian Imports

In the global production system, China has emerged as an export giant and is now at the center of the world's global manufacturing and product development. In just a decade, imports from East Asia, particularly China, have expanded significantly and are expected to continue to dominate the U.S market for years to come. China's share of U.S. containerized imports, for example, expanded from 43% to 48% between 2005 and 2009.[14]

A review of the world's top ports shows the extent to which China controls global trade. The top port in China handled 28 million TEUs (Twenty-foot Equivalent Units, a measure of container volume) in 2008, compared to 7.8 million for the largest port in the United States (Los Angeles). As the following chart indicates, some of China's key

Figure 2. Top 10 U.S. Maritime Container Ports by Annual Traffic 1995- 2008 (Thousands of TEUs)

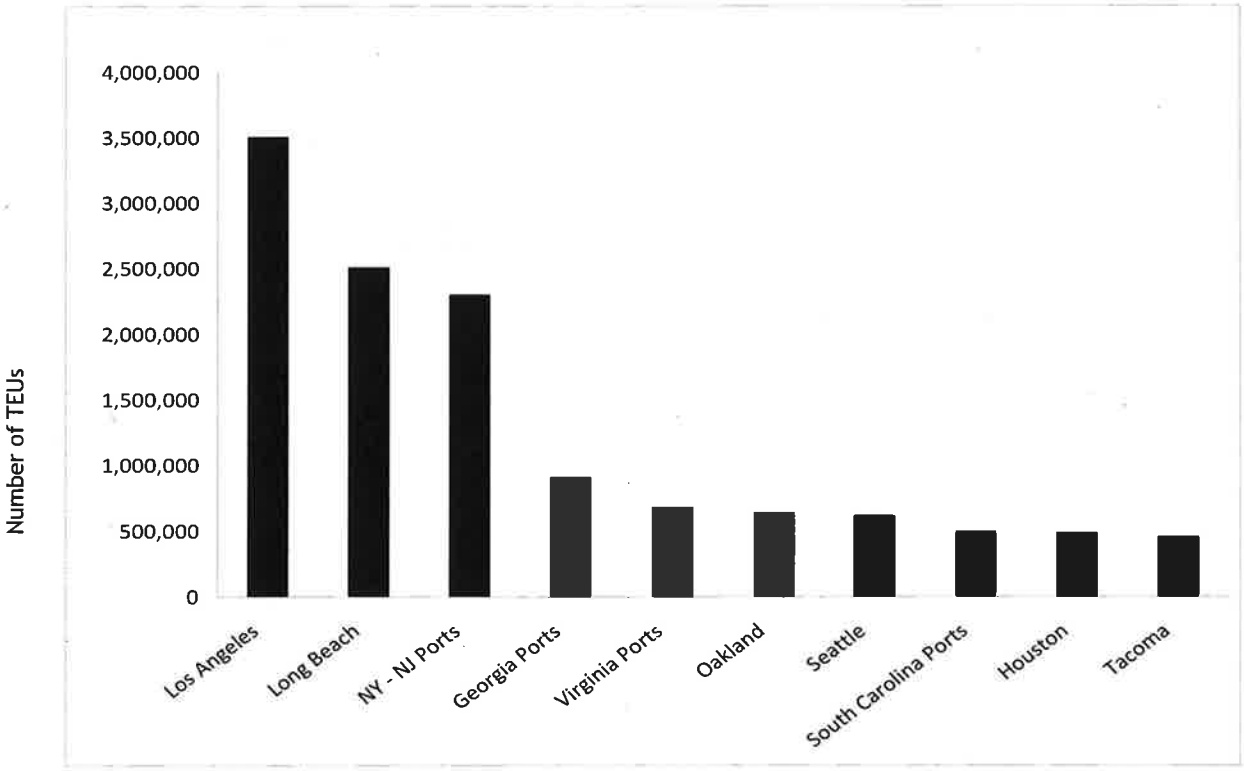
Port	1995	2008	Percent change 1995-2008	Avg. Annual Growth Rate (%) 1995-2008
Los Angeles, CA	1,849	5,671	206.7	9.0
Long Beach, CA	2,137	4,612	115.8	6.1
New York/New Jersey	1,537	3,992	159.7	7.6
Savannah, GA	445	2,116	375.5	12.7
Norfolk, VA	647	1,592	146.00	7.2
Oakland, CA	919	1,395	51.8	3.3
Charleston, SC	758	1,371	80.8	4.7
Houston, TX	489	1,331	172.2	8.0
Seattle, WA	993	1,129	13.7	1.0
Tacoma, WA	604	1,083	79.3	4.6
Total top 10 ports	10,378	24,291	134.1	6.8
Total all ports	13,328	28,190	111.5	5.9
Top 10, percent of total	77.9	86.2		

KEY: TEUs = twenty foot equivalent units. One 20-foot container equals one TEU, and one 40-foot container equals two TEUs.

NOTE: The data in this table include only loaded containers in U.S. international maritime activity and cover U.S imports, exports, and transshipments. Therefore the trade levels will be greater than those reported form U.S. international trade statistics, which exclude transshipments. The data also exclude military shipments.

Source: Chart adapted from "America's Container Ports: Freight Hubs That Connect Our Nation to Global Markets June 2009." U.S Department of Transportation, Research and Innovative Technology Administration, Bureau of Transportation Statistics, based on data from U.S. Department of Transportation, Maritime Administration, which are drawn from The Journal of Commerce, Port Import Export Reporting Service (PIERS) as of March 20, 2009.

Figure 3: Top 10 U.S. Container Ports by TEUs



Source: Data from The Journal of Commerce, May 3, 2010 Adapted by Elba Garcia, USC

ports showed an average annual growth rate of 22% to 37% between 2000 and 2008. See Figure 4.

China has been moving to extend its future trade capacity by investing heavily in transportation infrastructure. For example, the Chinese government plans on developing an intermodal rail system that will eventually connect 18 inland ports to existing ocean shipping ports. Seven have already been built and connected, with five additional terminals scheduled for completion. These projects give China the ability to maintain and extend its role as the world's leading producer and exporter of goods.[15]

Expansion of the Panama Canal

When the Panama Canal was completed in 1914, the largest container ships at that time, dubbed "Panamax ships," had the capacity to pass through its locks. As the years progressed, however, ocean-going vessels grew larger and more able to carry additional containers. By 1985, these larger ships, known as "post-Panamax" ships, were carrying a maximum of 4,500-5,000 containers, and they could no longer fit through the canal.[16] As a result, East Coast ports suffered as shippers turned to the West Coast and to inland distribution networks to deliver their goods.

In 2005, plans for the expansion of the Canal were introduced; a year later in a highly political and contested election, voters in Panama approved the project. When the $5.25 billion expansion of the Panama Canal is complete in October 2014, the Canal will be able to handle even larger ships, called "New Panamax," with a capacity of 12-13,000 TEUs.[17] [18] Most of the ships expected to transit through the locks, however, are expected to be in the 6-8000 TEU capacity.[19] For the most part, only U.S. ports that are in the range of 48-50 feet deep will be able to readily accommodate these larger New Panamax ships. As a result, many East Coast ports - including those in Baltimore, Jacksonville, Miami, Savannah, Charleston, and Norfolk are actively engaged in seeking approval to dredge or otherwise expand their port facilities. In New York/New Jersey the port authority has even agreed to raise the roadbed of the Bayonne Bridge in order to accommodate the New Panamax ships, a project that will cost up to one billion dollars.[20] Many East Coast ports have petitioned for federal dollars to underwrite expansion projects that they believe will allow them to capture an anticipated "reverse Gold Rush" of Panama Canal related imports.

Figure 4. Top 20 World Ports

Rank in 2000	Rank in 2007	Rank in 2008	Port Name	Country	2000	2007	2008	Percent Change, 2000-2008	Percent Change, 2007-2008	Avg annual rate (percents), 2000-2008
2	1	1	Singapore	Singapore	17,040	27,932	29,918	76	7.1	7.3
6	2	2	Shanghai	China	5,613	26,150	27,980	398	7.0	22.2
1	3	3	Hong Kong	China	18,098	23,881	24,248	34	1.5	3.7
11	4	4	Shenzhen	China	3,994	21,099	21,414	436	1.5	23.4
3	5	5	Busan	South Korea	7,540	13,270	13,425	78	1.2	7.5
13	7	6	Dubai	United Arab Emirates	3,059	10,653	11,828	287	11.0	18.4
65	11	7	Ningbo	China	902	9,360	11,226	1,145	19.9	37.0
38	12	8	Guangzhou	China	1,430	9,200	11,001	669	19.6	29.1
5	6	9	Rotterdam	Netherlands	6,280	10,791	10,800	72	0.1	7.0
24	10	10	Qingdao	China	2,120	9,462	10,320	387	9.1	21.9
9	9	11	Hamburg	Germany	4,248	9,900	9,700	128	-2.0	10.9
4	8	12	Kaohsiung	Taiwan	7,426	10,257	9,677	30	-5.7	3.4
10	14	13	Antwerp	Belgium	4,082	8,177	8,664	112	6.0	9.9
32	17	14	Tianjin	China	1,708	7,103	8,500	398	19.7	22.2
12	16	15	Port Klang	Malaysia	3,207	7,120	7,970	149	11.9	12.1
7	13	16	Los Angeles	United States	4,879	8,355	7,850	61	-6.0	6.1
8	15	17	Long Beach	United States	4,601	7,312	6,488	41	-11.3	4.4
113	18	18	Tanjung Pelepas	Malaysia	418	5,500	5,600	1,239	1.8	38.3
17	20	19	Bremen/ Bremerhaven	Germany	2,712	4,892	5,501	103	12.4	9.2
14	19	20	New York/ New Jersey	United States	3,050	5,400	5,265	73	-2.5	7.1

KEY: TEUs = twenty-foot equivalent units. One 20-foot container equals one TEU, and one 40-foot container equals two TEUs.

Chart adapted from: U.S. Department of Transportation, Research and Innovative Technology Administration, Bureau of Transportation Statistics. "America's Container Ports: Freight Hubs That Connect Our Nation to Global Markets" Washington, DC: 2009.

Inland Expansion of Goods Movement Hubs

Global trade shifts, including the impact of the Panama Canal expansion, directly affect the landscape of how and where freight in the United States will be transported and distributed. Innovations in retail and distribution technologies and the desire to maximize efficiencies and minimize risk have spurred investment in intermodal facilities that create more fluid connections between rail, trucks, warehouses, and distribution centers. This development pattern has created fast-growing inland hubs where trucks deliver containers from the seaports to inland distribution centers. Workers in these inland hubs then load imported goods onto trucks that are bound for regional stores or onto long-haul trains set for cross-country markets. The result is an elaborate and extensive "hub and spoke" distribution system that delivers containers from the seaports to inland rail hubs and then to distribution hubs and on to local and national retail markets.

A milestone in the shipping industry's adoption of inland distribution strategies was the 1989 opening of the Virginia Inland Port, located 220 miles from the ports of Norfolk, Newport News, and Hampton Roads. Another example of this trend is the key role that inland distribution centers and intermodal hubs in Riverside and San Bernardino counties, which are 50 miles inland from the coast, have played in the expansion of Southern California's port capacity since the late 1990s. Distribution hubs are also growing around new intermodal rail facilities, such as the $370 million, 785-acre Joliet Intermodal Terminal outside of Chicago. In conjunction with the Joliet rail terminal a 20 million square feet of container distribution facilities (warehouse, distribution, cross-dock, and transloading) will be built at the CenterPoint Intermodal Center.[21] This new complex in Will County, IL further illustrates the development of freight transportation infrastructure outside urban core areas - an increasing trend used to avoid bottlenecks and congestion. The facility is expected to address transport bottlenecks in Chicago, where it has become increasingly difficult for the trains to move through the region in less than two days, or the same amount of time required to reach Chicago from the West Coast.[22]

Similarly, just 12 miles from downtown Dallas, the 360-acre Southern Dallas Intermodal Terminal (DIT) is designed to support growing intermodal volume in the region. A new warehouse distribution and manufacturing center, the Dallas Logistics Hub, is under development and will become the largest new logistics park in North America, with 6,000 acres of land and the potential for 60 million square feet of distribution, manufacturing, and retail development.[23] Located near four major highways, intermodal rail facilities and a proposed air cargo airport, the Dallas Logistics Hub will compete with older intermodal facilities in Dallas and create new competition for other hub areas such as Chicago.[24]

Large distribution centers are a growing trend national trend. Walmart alone has 40 regional distribution centers around the United States that are larger than one million square feet each.[25] Ross Stores recently finished a 1.3 million square foot distribution center in Fort Mill, South Carolina, an important component of the goods movement route anchored by the port in Charleston.[26] A 1.8 million square foot distribution center is under construction for Skechers shoe store in Moreno Valley, CA.[27] Despite the arguments that the Skechers warehouse would bring jobs, many residents fought its approval, fearful that it would bring traffic congestion and air pollution to the area and lower their property values.[28]

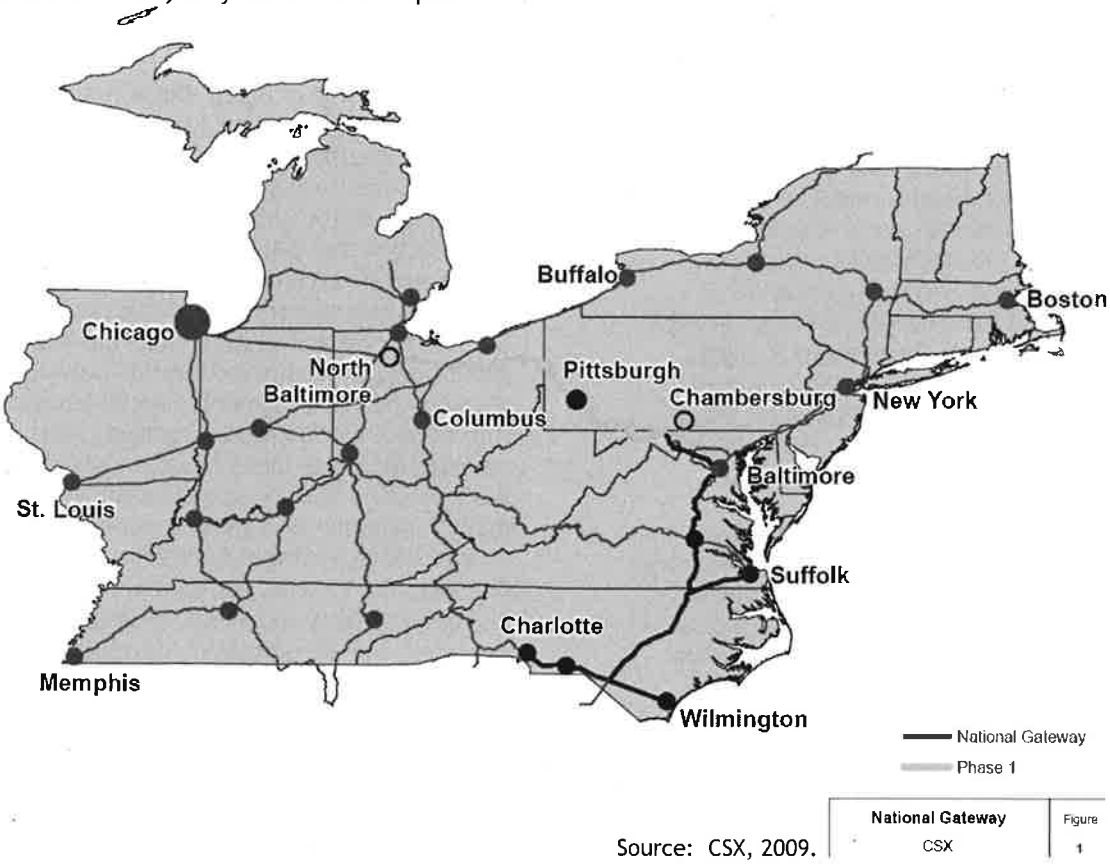
Expansion of Rail and Highway Corridors

The inland hubs are facilitated by major investments in rail and highway corridors, especially on the East Coast. Many of these rail and highway projects have been subsidized by the American Recovery and Reinvestment Act (ARRA) stimulus funds, and as a recent *Journal of Commerce* article notes, they are expected to "change the economic landscape of the country's distribution networks."[29]

Two of the nation's freight railroad companies, Norfolk Southern and CSX railroads, have capitalized on such ARRA stimulus funds (see section "Public Subsidies for Import-Based Development") and have created public-private partnerships with local, state and federal government agencies to expand and develop rail corridor projects that link established East Coast ports with Midwest markets. As an example of the project's magnitude, a Norfolk Southern project entailed blasting through 28 mountain tunnels in West Virginia to allow higher clearances for double-stacked Norfolk Southern trains, as well as laying new rail track. This project, called the "Heartland Corridor," opened in September 2010 linking the Norfolk, VA port terminals to Columbus, OH.[30] The corridor is designed to facilitate goods movement from the Virginia ports by shortening the route to the Midwest along high-speed double stack rail. Because of the Corridor, new Norfolk Southern intermodal terminals are under construction in Roanoke, VA, Columbus, OH and Prichard, WV.[31]

Competing for East Coast rail growth is CSX Corporation that has proposed a National Gateway Corridor project in an effort to build more efficient freight transportation links between Mid-Atlantic ports and the Midwest. The CSX plan includes a combination of rail infrastructure and intermodal terminal projects along three major freight rail corridors owned and operated by CSX. The proposed

Figure 5. National Gateway Project of CSX Transportation



Source: CSX, 2009.

National Gateway Corridor will also provide a more efficient north-south route between North Carolina and Baltimore, paralleling I-95. The project proposes to expand three existing rail corridors that run through Maryland, Virginia, North Carolina, Pennsylvania, Ohio and West Virginia.[32] See Figure 5.

West Coast railroads are also expanding, as are some highways in the West. Union Pacific (UP) and Burlington Northern Santa Fe (BNSF) are the country's largest railroads and operate in the Western United States. Both have used private and, recently, public funds to expand their operations or make them more efficient.[33] For example, funding is being used to make the Colton Crossing in Riverside, CA less of a chokepoint by constructing a flyover for freight trains. This crossing is currently one of the busiest at-grade rail-to-rail crossings in the nation. This is a description of the project's funding:

> *The funding comes from a combination of state, federal, and private monies from BNSF and Union Pacific. In February 2010, the project was awarded $33.8 million in American Recovery and Reinvestment Act (ARRA) stimulus funds through the Transportation Investment Generating Economic Recovery (TIGER) program. In May 2010, the California Transportation Commission approved $91 million in funding from the voter-approved California Proposition 1B Trade Corridor Improvement Fund (TCIF).*[34]

We can expect these trends (new highway corridors and expansion of distribution centers) to continue as many intermodal facilities (with related warehousing operations) are being proposed or constructed in inland areas, including in Ohio, Kansas, Tennessee and elsewhere.

The Continuing Importance of Trucks

Although the airwaves are full of commercials arguing in favor of rail transport,[35] trucks continue to play a major role in the goods movement industry. Rail companies promote a rail advantage in hauling containers on less fuel than trucks.[36] This may be true on long stretches of land across the country. But to get containerized products to and from their final destinations, multiple truck trips almost always are a necessity and can also include a rail yard where containers are transferred to trains. Few port terminals in the United States use an efficient system of placing a container onto a train directly from a ship without any involvement of a truck (although the technology exists to do this and the scheme is depicted in the CSX video). The highway trade

association American Association of State Highway and Transportation Officials provides the following example of truck involvement in "rail movement" of goods:

> *... Merchandise manufactured and packed in a container in China may be imported to the United States through the Port of Long Beach, trucked to the nearby intermodal container transfer facility, loaded onto a double-stack unit train, moved by rail to Chicago, transferred across town by truck from a western railroad to an eastern railroad, moved by rail to north Jersey, transferred to truck, taken to a nearby distribution center where the contents are transferred to smaller trucks, and finally delivered by van to a customer in Brooklyn (underlining added).*[37]

One route described above may be slightly shortened by a developing partnership between BNSF (a western railroad) and CSX (an eastern railroad) whereby a CSX crew in Chicago can relieve a BNSF crew and move the train to a proposed intermodal facility in northeast Ohio. In Ohio, the train's containers are unloaded before it travels back to the BNSF crew in Chicago. This would eliminate the need for moving containers by trains within the city of Chicago from one train terminal to another.

Trucks hauling containers are extremely heavy and can cause serious damage to bridges, highways, and local roads. The California State Department of Transportation (Caltrans) states that "trucks traveling from the ports in Southern California cause the majority of damage and stress to roadway surfaces, bridges and other infrastructure elements."[38] According to Resources for the Future (RFF), an environmental think-tank in Washington, D.C., "heavy-duty commercial trucks account for only 7.5% of highway miles, but they contribute disproportionately ... to pavement deterioration, traffic congestion, pollution, and nationwide dependence on a volatile world oil market."[39] Similarly, a study of pavement damage conducted at the University of Texas found that "one big-rig pass causes the damage equivalent to 2,000 to 3,000 cars."[40]

As the RFF study noted, to calculate the range of truck-based external costs, air pollution, pavement damage, traffic congestion, noise, road accidents, global warming, and fuel security need to be considered. Continuing truck use, along with its extensive impacts, remains a key trend that often complements rail systems as the pivotal anchor of freight transportation within the United States as well as exporting countries like China.

Public Subsidies for Import-Based Development

The influence of major global firms and their role in global markets has had a large role in shaping trends and economic development policies. Many cities and regions turned to import-based development policies in the wake of the global economic restructuring that took place in the 1980s and 1990s.[41] While most studies of globalization focus on runaway production, declining manufacturing sectors, information technology and the rise of financial sectors, little has been written about how commodity distribution networks have shaped urban development.[42] Retail innovations and shifting distribution processes have created new links between American cities and the global economy. Rising imports and an expanding shipping network have made it possible for a growing number of regions and cities, such as Southern California, Oakland, Savannah, Chicago, and Virginia, to capture a greater share of global commodity shipments by adopting port-based economic and transportation development models.[43]

This port-based development discourse became the underlying logic of regional economic policies that cleared the way for government support for goods movement projects. Los Angeles' emergence as a major gateway for Asian imports was made possible by a significant public investment in port capacity. Billions of dollars were invested in port infrastructure as a way to modernize the ports and to make them competitive in a global context.[44] Recent state funding initiatives like California's Prop 1B program collected as much as $20 billion in tax revenues to subsidize projects and programs related to transportation and goods movement in California. Although $2 billion was set aside for air quality programs, the investment of public funds is one example of how taxpayers pay for the infrastructure that enables private development, logistics, shipping and retail corporations to expand their operations without addressing the public costs of health impacts.[45]

The expansion of Southern California port activities is one example of how local policy makers have deployed an imports-based development approach and set of arguments to justify massive public spending on freight transportation projects in the 1990s and 2000s. Expansion of goods movement infrastructure serving the ports of Long Beach and Los Angeles has become the embodiment for numerous policymakers of the path to regional economic prosperity. Economic boosters - often linked to the Southern California Association of Governments, other associations of governments and the port authorities - regularly claim that expanding the region's distribution network will provide middle class jobs to local blue-collar workers with only a high school education (or less).[46, 47] Moreover, SCAG argues

that if the region builds the necessary infrastructure "to accommodate the growth of international trade and clean up its worst environmental side-effects, some 1,381,000 jobs can be created in Southern California."[48]

Meanwhile, national policy makers used the economic crisis of 2008 to call for expanded public funding of the country's transportation system. Both Republicans and Democrats argued that improving the country's aging infrastructure - through transportation funding for bridges, roads, highways, freight rail and intermodal facilities - would stimulate the economy. As part of the American Recovery and Reinvestment Act (ARRA) stimulus funds package, the U.S. Department of Transportation has funded $1.2 billion for transportation projects through the Transportation Investments Generating Economic Recovery (TIGER) program. To date, two rounds of funding in February 2010 and October 2010 have funneled more than $961.3 million in public funds to key ports and freight transportation projects. See Appendix.

In October 2010, President Obama called for a $50 billion initiative to modernize the nation's infrastructure; his 2012-13 budget proposal includes a six-year $556 billion for federal transportation funding. The wave of public investment and subsidies has fueled private enterprise but comes at a time when trust in the public sector and government's ability and willingness to increase health, labor and environmental protections is at a low point. Thus, while public investment for private economic growth appears acceptable, public protection of health, environment, and workers appears to be considered unacceptable by some, as noted in the next section.

Impacts on Workers, Health, Community, and the Environment

Evidence makes it clear that global trade and the freight transportation system causes substantial environmental, health, workplace and community impacts. These include local and regional air pollution from emissions of ships, trucks, locomotives, and yard equipment operating at ports, rail facilities, on highways, and at warehousing operations. Such impacts can disproportionately affect residents in communities near ports and in neighborhoods near freeways or where heavy truck traffic or locomotives are present, in addition to affecting the health of dock and warehouse workers, truck drivers, and railroad employees. Recognizing that port and rail communities disproportionately bear the costs of health impacts from international trade while the rest of the United States reaps benefits, Mayor Bob Foster of Long Beach CA commented in 2008:

> *Quite frankly, my first job as mayor of Long Beach is to protect the health and safety of my citizens. In my city, families that live along the trade corridors have two to three times the statewide average of asthma cases. That's not an accident. I've said it many times: we are not going to allow kids in Long Beach to contract asthma so someone in Kansas can get a cheaper television set. Those days are over.*[49]

Other neighborhood impacts from freight transportation operations include noise, round-the-clock bright lighting at port and rail operations, conflicts between incompatible land uses and public health, and the potential for contamination from hazardous spills. Goods movement communities also face traffic safety problems, unsafe streets for pedestrians, and hefty local tax charges to repair streets that are damaged by big-rig trucks.

Workers in the freight transportation industry face a number of issues. Some, such as unionized longshore workers, are able to secure living wages and benefits while others, such as warehouse workers, are part of the contingent workforce, working at the convenience of employers and often through a temp agency. Still others, such as port truck drivers, who are technically classified as "independent contractors," are left to carry the heavy burden of maintaining and operating their trucks while performing the work of private freight transport firms.

On a more global scale, international trade activities contribute to global warming, with significant emissions of carbon dioxide, black carbon and other pollutants. While the published and peer review literature has documented these community, workplace, health, and environmental impacts, findings have not been widely incorporated into policy decisions or into assessments of the comparative benefits and negative impacts from international trade and freight transportation. The section of the report below provides a brief summary of health research findings and helps situate the policy and organizing context for how best to address the underlying problems associated with international trade and goods movement traffic. See also chart at the end of this section. Additional information about air pollution and health impacts can be found in a searchable U.S. EPA database of scientific citations.[50]

Impact on Workers

Labor impacts include how the freight transportation and logistics industries that manage the flow of goods from overseas to U.S. consumers have produced a new generation of contingent workers. These workers have low wages and are often used seasonally or hired periodically as the

economy ebbs and flows. Many of the jobs involve temporary workers who have fewer benefits and far less job security, particularly those associated with the massive distribution centers that constitute a key part of the freight transportation system. Port truck drivers are misclassified as independent contractors and lack basic worker protections such as hourly wage, overtime, health insurance, unemployment benefits, OSHA protections, and the right to organize. The global shift of production away from higher paying manufacturing jobs in the United States to this contingent or "flexible" labor work force has therefore emerged as a key outcome of the rapid growth of the global freight transportation system.

Impacts on temp workers and independent contractors

One of the ways that shippers have reduced their costs in the development of inland hubs is by creating a distribution system that relies on a large army of low-wage, temporary warehouse workers. Evidence from Chicago and Southern California reveals that many of the workers who are employed as "flexible labor" (or temps) by the goods movement industry's massive distribution centers are paid poverty-level wages.[51] A survey in Chicago found that the majority of warehouse workers were temps earning wages below the federal poverty level. [52]

It is important to note that the freight transportation and logistics industries have historically used inland distribution centers and workers as a cost-saving and anti-union strategy. For example, while shippers claim that a decentralized system is part of their move towards more efficient and flexible distribution of goods, it is also an attempt to circumvent the type of labor power displayed by the West Coast port lockout of 2002.[53]

One has to search carefully to find where these temporary warehouse workers show up in employment statistics, and not all economists appear to include them when they calculate the average wages of warehouse workers. Our analysis shows that increased numbers of temporary workers appear in annual average employment growth under the category of Professional and Technical Services.[54] For example, from 2000-2009 annual average employment growth in goods movement industries rose 3.1% and 5.3% respectively in San Bernardino and Riverside Counties. Meanwhile, there was additional growth in the category called Professional and Technical Services (the classification for temp workers) of 5.5% and 7.3% respectively. In Will County, Chicago, where warehouse activity has expanded, average employment growth in the Professional and Technical Services grew 4.2%, in addition to 8.2% annual growth in goods movement employment.[55]

Truck drivers who move cargo to and from the ports reflect another set of workers in the freight transportation system earning poverty-level wages and serving as flexible labor for freight shipping firms. With the deregulation of the trucking industry in the 1980s, independent truck drivers were forced to bear the burden of all maintenance and upkeep for trucks that cost over $100K, along with Port fees, licensure, fuel and other costs of doing business at the Port. Truck drivers who own and operate their own trucks and must compete individually for hauling jobs, and net less than $30,000 annually.[56] In a survey of 2,183 workers at seven major ports, researchers from National Employment Law Project, the union federation Change to Win and Rutgers University found that 82% of the nation's 110,000 port truck drivers are treated as independent contractors.[57] This misclassification of drivers as independent contractors prevents them from securing basic worker protections, such as hourly wage, overtime, health insurance, unemployment benefits, and the right to organize.[58]

The interlocking factors of employment status, poor wages, lack of benefits and health insurance, combined with exposure and increased health and safety risks as described below, pose employment, workplace, and residential impacts to this growing sector of workers.

Health and safety of workers in the freight transportation industry

Many workers in the goods movement industry also face significant health and safety hazards on their jobs as truck drivers, warehouse workers, and railroad workers. For example:

- **Worker fatalities.** In 2009, workers in the "transportation and warehousing" industry had the second highest number of worker fatalities in the United States, with #1 being "construction." Workers in this industry had the third highest work injury fatality rate of all industries, exceeded only by the category of "agriculture, forestry, fishing, and hunting" (#1) and by "mining" (#2).[59]
- **High hazard jobs.** In California, both warehousing and truck transportation were on the "Highest Hazard Occupation" list for OSHA inspections (2009-2010).
- **Exposure to heat stress.** Workers in non-air conditioned truck and locomotive cabs and in warehouses are subject to potential heat stress. Warehouses in the Inland Valleys of Southern California, where summer temperatures are often above 100 degrees Fahrenheit, seldom have air-conditioning.

- **Lung cancer and heart disease deaths, accidents - truck drivers.** A study of causes of death among unionized workers in the trucking industry (compared with the general U.S. population) found an excess of lung cancer and heart disease deaths among drivers, dock workers and shop workers at trucking terminals, and higher than expected deaths from transportation-related accidents.[60] Another scientific study surveyed 31,000 union workers in the U.S. trucking industry exposed to higher levels of diesel exhaust on a regular basis as part of their jobs. It found that they were more likely to develop lung cancer with increasing years of work.[61] According to the California Air Resources Board, lung cancer rates among workers in the trucking industry are among the top five highest rates of all industries surveyed in the state.[62, 63]

- **Lung cancer deaths - railroad workers.** A study of nearly 55,000 U.S. railroad workers (who worked in the industry from 1959 - 1996 found that exposure to diesel exhaust was linked to lung cancer deaths among these workers.[64]

- **COPD - railroad workers.** U.S. railroad workers hired after the introduction of diesel locomotives had a 2.5% increase in chronic obstructive pulmonary disease (COPD) mortality risk for each additional year of work in a diesel-exposed job.[65]

- For those workers who live in the neighborhoods surrounding ports, the health risk is compounded. In Los Angeles, for example, 50% of port truck drivers live in low-income or poverty level neighborhoods near the ports and along the port trucking corridors,[66] where levels of localized air pollution are elevated.

Exposure to Air Pollutants

Air pollution is a mixture of gases, such as carbon monoxide and nitrogen dioxide, and particles of different sizes. The particles usually come from vehicle emissions, factories, wood or gas stoves, and/or wildfires.

Goods movement activities provide multiple sources of air pollution, much of which relate to diesel emissions. Freight transportation can add to "regional air pollution" or, in cases where freight facilities are close to homes and schools, they can create "localized pollution." Thus, ship and vehicle emissions near a port can add to the regional pollution that residents in a wide geographic breathe. But for those residents who live in close proximity to a marine terminal with ships, yard equipment and idling trucks waiting for containers, it can mean additional "local pollution." This is also true for residents living near rail yards, highways, and distribution centers. In these cases, not only are the residents exposed to regional pollution similar to all residents in their region, but they are also exposed to additional pollution because of their closeness to truck, locomotive and ship exhaust.

Diesel exhaust consists of gases and particles. Some of these particles are in the $PM_{2.5}$ range (that is they are smaller than 2.5 microns in diameter). Within that size range are extremely small particles called "ultrafine particles," tinier than 0.1 microns in diameter. Automobile exhaust also contains particles in these sizes. Both cars and diesel trucks emit "black carbon" in their exhaust, although much higher levels of black carbon (or "elemental carbon") are found when sampling air pollution levels on truck-congested highways than on highways with mostly gasoline vehicles.

Diesel particulate forms a large part of the fine particulate matter (PM) in urban air.[67] Because studies of PM seldom differentiate the source of the particulate matter (that is, whether the PM comes from gasoline vehicles, diesel vehicles, power plants, refineries, etc.), we present first the information on health effects of PM and then the specific studies addressing diesel PM. We also note that diesel exhaust contains gases as well as both fine and ultrafine particles (UFPs).

Below, we describe some of the research findings on health effects of exposure to air pollution.

Health effects of exposure to $PM_{2.5}$

Studies demonstrate that exposure to $PM_{2.5}$ (particles) increases the risk of cardiovascular disease and reduces life expectancy. That is, those exposed to higher levels of particulate matter are more likely to develop cardiovascular disease or die earlier than expected from heart disease (such as heart attacks and coronary artery disease, according to a number of studies published during the past 20 years.[68 69 70] The American Heart Association recently published a statement on the role of air pollution in heart diseases, stating that:

> *Numerous epidemiological studies conducted worldwide have demonstrated consistent associations between short-term elevations in PM and increases in daily cardiovascular morbidity [illness] and mortality [death]…. Several studies have also reported adverse cardiovascular outcomes in relation to long-term PM exposure. Elderly patients, those with underlying coronary or pulmonary disease, lower socioeconomic populations, and diabetics may be at particularly increased risk… Pope has*

estimated an average loss of life expectancy directly related to chronic air pollution exposure from between 1.8 and 3.1 years for those living in the most polluted cities in the United States. Cardiovascular causes account for the majority (69%) of the overall excess in morbidity and mortality.[71]

Globally, concerns have also been raised about shipping emissions, because ships have diesel engines that run on bunker fuel - a low-grade fuel that is considered "dirtier" than diesel fuel. A landmark study by a University of Delaware scientist noted that those living near coastlines are at particular risk from particulate matter in shipping emissions; he and his team calculated that 60,000 persons a year die prematurely from particulate matter released by ocean-going ships involved in international trade.[72]

Diesel particulate emissions in the community: impacts on residents' health

Concerns about diesel exposure are highlighted in this report because the ships, trucks, locomotives and much of the yard equipment used along the entire goods movement supply chain are diesel-fueled and emit a significant amount of air pollutants - and they create significant risk for residents in goods movement communities.

Diesel emissions cause some of the most extensive impacts from freight transportation. The California Air Resources Board estimates, for example, that goods movement activities in that state each year cause 3,700 people to die prematurely - earlier than they would have if they had not been breathing high levels of particulate matter.[73]

To calculate the anticipated output of particulate matter (PM) and other air pollutants, a number of ports have conducted emissions inventories. One of the first such studies was conducted in Southern California and published in 2007, estimating that the Port of Los Angeles in 2005 was contributing 12.5% of the PM air pollution in the Southern California region (and that the Los Angeles and Long Beach Ports combined were contributing some 25% of the region's PM).[74] This inventory determined that ships were the largest source of diesel pollution from the ports, producing 54% of the ports' diesel emissions in 2006, with freight trucks being second.[75]

Another study of the Port of Los Angeles was a Health Risk Assessment (HRA), conducted by the California Air Resources Board. It found an elevated diesel cancer risk in those living close to the Port, but also found that even 15 miles away residents had a higher risk than others in Southern California did. The HRA calculated that emissions from the two Ports combined were estimated to cause annually 120 premature deaths and 750 asthma attacks.[76] Another study by the South Coast Air Quality Management District took air pollution samples and modeled air pollution risks. It, too, demonstrated elevated diesel cancer risks in and around the Port of Los Angeles, leading one resident - an emergency room physician - to proclaim that those in the Harbor area were "living in a diesel death zone."[77]

Emissions inventories have also been conducted at 18 major rail yards in California, as part of a series of Health Risk Assessments for those yards prepared by staff of the California Air Resources Board. The inventories were used to estimate the tons of particulate matter in diesel exhaust emissions at each yard per year. For example, a large BNSF rail yard in Barstow, CA was estimated to emit 26 tons of diesel particulate matter per year.

Above, we described some of the known health impacts of diesel exposure on workers, including lung cancer. Exposed community members are also considered at risk of lung cancer from diesel emissions in and near their communities. Some of the other known health impacts for residents exposed to diesel include:

- **Reduced lung function in children exposed to diesel while growing up.** According to investigators at USC, children who grow up in more polluted communities with high levels of elemental carbon or EC (indicating diesel particle pollution) are more likely to have reduced lung function.[78]

- **Effects on lung function in adults with asthma, exposed for a brief period to diesel exhaust.** A study in London demonstrated that short-term acute exposure to diesel exhaust in adults who already had asthma could impact lung function; the study compared persons with asthma who walked in a park with no diesel traffic and then several weeks later walked on a London street with high volumes of diesel taxis and buses. Reduction in lung function and an increase in markers of inflammation were seen when the group was exposed to diesel exhaust - and the changes were associated with elevated levels of elemental carbon and UFPs.[79]

- **Reduced sperm production and endocrine disruption in laboratory animals.** In a series of Japanese studies of laboratory animals, prenatal (in *utero*) exposure to diesel exhaust particles were found to reduce sperm production in adulthood.[80] The Japanese team concluded that exposure to diesel exhaust particles disrupts endocrine (testicular) function in the male mouse reproductive system.[81] In many of these

studies, filtered air caused the adverse effects, suggesting that the gaseous phase of diesel exhaust appears to be the cause.

Health impacts of exposure to ultrafine particles

New concerns have been raised in the past 10-12 years about the potential health effects of ultrafine particles (UFPs) from combustion processes. These UFPs have been studied less extensively than $PM_{2.5}$ or larger PM_{10} particles. UFPs do not weigh much because of their size, making up only 10% of the total mass of $PM_{2.5,}$ and they have a large surface area, to which harmful chemical constituents from the exhaust can adhere.[82]

Recently, fourteen European scientific experts collaborated on reviewing the scientific literature on UFPs. Most of the experts concluded that:

> *The likelihood of an independent causal relationship between increased short-term UFP exposure and increased all-cause mortality, hospital admissions for cardiovascular and respiratory diseases, aggravation of asthma symptoms and lung function decrements was rated medium to high.*[83]

The group stressed the importance of considering UFPs in future risk assessments and the need for further research on UFP exposure and health effects. Similar suggestions were made at a symposium in California sponsored by the South Coast Air Quality Management District, at which several scientists who spoke concluded that that if UFPs cause health effects, an air pollution standard for UFPs is needed.[84]

Some of the studies show that:

- **More than 90% of particles in diesel exhaust are actually ultrafine particles UFPs**, smaller than 0.1 micron in diameter, and the **tiny particles can be easily inhaled into the lung.**[85] Laboratory studies increasingly show that these UFPs are more toxic and have a greater ability to cause lung inflammation than larger sized particles.[86]
- **Ultrafine particles appear to possess the most toxic potential of various size particles**, according to Los Angeles researchers.[87]
- **Ultrafine particles translocate (migrate) to the brain[88] and to promote early atherosclerosis** (hardening of the arteries) in exposed laboratory animals.[89]

Exposure to traffic-related pollution: living close to busy road and highways

New studies over the past decade have examined the levels of traffic-related pollution at different distances from homes and schools, the volume of nearby traffic, and the nature of land uses in the area. These studies have shown that the levels of several traffic-related pollutants (ultrafine particles, nitrogen dioxide, and elemental carbon, a marker for diesel) are high in close proximity to roadways, especially within the first 150 meters from the road[90] and that living or going to school in close proximity to busy roads and highways with significant traffic-related pollution is linked to adverse health effects. Studies involving children exposed to traffic-related pollution have shown the following effects:

- **Reduced lung function.** Children who live near traffic-related air pollution are more likely to suffer reduced lung function as they grow up.[91]
- **Increased risk of asthma.** Children living in homes within 225 feet of a highway have an increased risk of asthma.[92] Children are more likely to develop asthma when exposed to traffic pollution at school.[93]
- **Increased wheezing, use of medication.** Asthma exacerbation such as wheezing and use of more asthma medication occurs more often among children living closer to highways.[94]

Many recent studies involve adults exposed to traffic-related pollution. The research findings have shown the following adverse effects:

- **Low-birth weight babies.** Women who live near busy highways while pregnant are more likely to have babies with low-birth weight or who are premature.[95]
- **Miscarriages.** California women who live within 50 meters of a road with daily traffic of 15,200 or more (compared to women living further away and exposed to less traffic) were more likely to suffer miscarriage. This finding was especially true among African-American and nonsmoking women.[96]
- **Pregnancy complications.** Exposure to traffic-related air pollution during pregnancy increases risk of preeclampsia and preterm birth.[97]
- **Women undergoing in-vitro fertilization** who were exposed to higher levels of traffic-related air pollutants, particularly nitrogen dioxide, had an increased chance of in-vitro fertilization failure.[98]
- **Breast cancer.** The risk of breast cancer among post-menopausal women is higher in areas where there are higher levels of NO_2. In this Canadian study, there was an increased risk of ~25% for every increase of 5 ppb in exposure to NO_2 (as a marker for traffic-

related pollutants) when the levels in one area were 5 ppb higher than in another area, the risk of breast cancer went up by 25%.[99]

- **Atherosclerosis.** Adults living within 328 feet of a Los Angeles freeway have twice the average progression of atherosclerosis, the thickening of artery walls that can lead to heart disease and stroke.[100]

- **Cognitive impairment.** Women living within 50 meters of roads with at least 10,000 vehicles a day on them are more likely to develop mild cognitive impairment as they age.[101]

- **Diabetes.** In a study of German women living near busy roadways, women who were exposed to traffic-related pollution were more likely to develop new cases of diabetes than those who were not exposed.[102]

- **Heart/lung disease deaths.** Living near a major road is strongly associated with deaths from cardiopulmonary (heart/lung) disease. Adults who lived near a major road in the Netherlands were found to have twice the risk of mortality from heart/lung disease as adults in the same city living further from roads.[103]

- **COPD.** Long-term exposure to traffic-related air pollution may contribute to the development of chronic obstructive pulmonary disease (e.g., emphysema), and this may be more likely to occur in people with diabetes and asthma.[104]

In addition, an analysis of cancer by census tracts in Los Angeles County found elevated rates of throat, mouth and tongue cancers and certain types of lung cancer in close proximity to a truck-congested I-710 freeway in Los Angeles County,[105] one of the most heavily used highways for movement of goods in the country.

Regulation of diesel emissions

Vehicle emission standards in the United States are promulgated by the U.S. EPA. The State of California has authority under the Clean Air Action to adopt more stringent standards, which are set by the California Air Resources Board. Other states can adopt the California standards or the national standards.

The U.S. EPA has studied the health effects of diesel exhaust and concluded that diesel is a "likely" human carcinogen, that is, a toxic likely to cause cancer in humans who are exposed over time. The agency regulates diesel exhaust as a Mobile Source Air Toxic (MSAT).

In California, diesel particulate matter is regulated more strictly than in the rest of the United States. In 1998, diesel particulate was designated a Toxic Air Contaminant (TAC) in that state, based on more than 30 studies showing that worker exposure to diesel exhaust is linked to lung cancer and other health effects.[106] This designation, along with hundreds of scientific studies showing the health effects of particulate matter (especially $PM_{2.5}$) provides authority for California to strictly regulate diesel emissions.

Community groups have raised serious concerns about locomotive emissions, especially from idling locomotives operating at rail yards that are near homes and schools. U.S. EPA has primary regulatory authority over locomotive emissions and has issued regulations. Arguing that "Federal law preempts California from setting emission standards for new locomotives and new engines used in locomotives," CARB has instead negotiated voluntary agreements with BNSF and Union Pacific, the two major freight railroads operating in the State. The South Coast Air Quality Management District attempted to limit idling of locomotives, efforts that have ended up in litigation. These voluntary efforts have disappointed and angered environmental justice organizations that represent residents who live near rail yards and have resulted in protests in both Los Angeles and Sacramento.

Exposure to Noise

Port and intermodal rail operations are noisy by virtue of the locomotives and yard equipment being used and the handling of heavy containers by cranes and other equipment. A common negative impact in freight transportation communities relates to the high volume of heavy-duty trucks - a major source of noise. Community and occupational health studies show that noise can affect health and quality of life.

Transportation noise research, largely conducted in Europe, shows three main types of impacts from noise:

- **Psychological (e.g., annoyance).** A number of studies show significant annoyance from exposure to high levels of transportation noise above 60 dB.[107] Children studied in The Netherlands were found to be seriously annoyed by both aircraft noise and road traffic noise at school.[108]

- **Physiological (e.g., hearing loss, increase in blood pressure).** Published studies show that workers are at risk of noise-induced hearing loss from noise at rail yards.[109] In addition, chronic noise exposure may contribute to the progression of heart disease.[110] In a recent study in the Netherlands, adults living near roadways who were exposed to noise were found to have higher rates of stroke in relationship to higher levels of noise, after accounting for air pollution.[111]

- **Mental health (e.g., anxiety).** Elevated noise levels from road traffic and airports affect children's mental health and classroom behavior.[112] [113] [114] Excessive noise from traffic can disturb restorative sleep.[115]

Figure 6 shows the noise levels in decibels for common outdoor and indoor noise activities. Note that quiet urban daytime noise is usually slightly above 50 dBA and that a diesel truck 50 feet away, going 50 miles per hour, may register between 80 and 90 decibels.

Figure 6. Noise levels for common activities

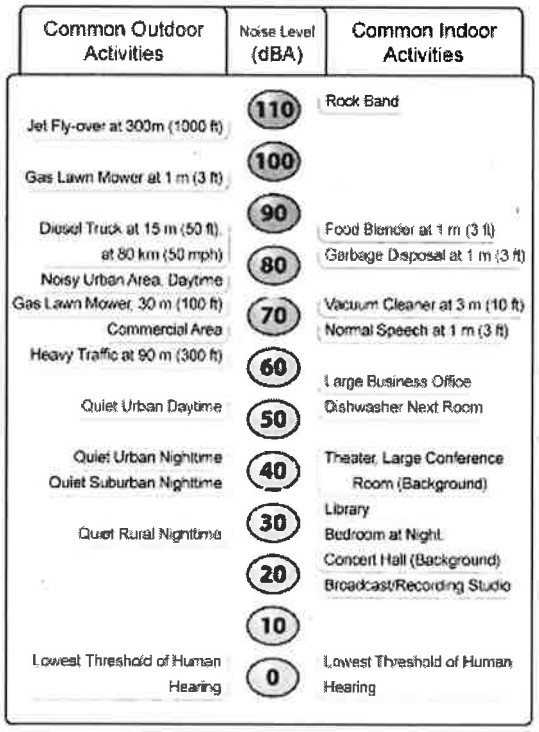


Exhibit 2.2.6-1
Typical Sound Levels from Indoor and Outdoor Noise Sources

Source: Final Environmental Impact Report for the Gerald Desmond Bridge, Port of Long Beach. 2010

Figure 7 shows estimated noise levels at certain distances from busy roads and at certain speeds, with varying levels of traffic. It helps to illustrate how much the number of trucks and the speed that the traffic is moving impacts the noise levels. The more trucks there are and the faster the traffic is moving results in higher noise levels.

Figure 7. Levels of noise at 150 feet from busy roads

Vehicles per Hour				
Automobiles	Medium Trucks	Heavy Trucks	Speed, mph	Leg (h) dB (A)
1,500	100	200	65	67
1,500	100	200	50	64
1,500	100	0	65	63
1,500	0	0	65	62

Source: Transportation Research Board of the National Academies, TR News. Transportation noise. Sept-Oct 2005, Number 240.

According to Southern California Association of Governments, complaints about noise vary according to the decibel level.[116] As Figure 8 shows, widespread complaints begin when noise gets into the 60 dB range, with serious annoyance between 60 and 70 dB.

Figure 8. Impacts of various noise levels on sensitive uses

Noise level	Where the noise is	Impact
55 dB	Outdoors near homes and sensitive uses	Sporadic complaints and community annoyance start
DNL 55-60 dB	Outdoors and near sensitive uses	Upper range for intelligible speech indoors at a typical home
55-60 dB range	Homes and sensitive uses	Widespread complaints
60-70 dB	Homes and sensitive uses	Threats of legal action begin
70 dB and above	Homes and sensitive	Strong appeals to local officials to stop the noise and threats of legal action

Source: Adapted from text, SCAG Regional Transportation Plan, Proposed EIR, 2004.[117]

Noise from locomotives is particularly high. A Southern California study showed that the noise impact caused by 40 freight trains per day generates approximately 75 decibels at 200 feet from the tracks and that freight trains also generate substantial amounts of ground-borne vibration near the tracks.[118] Community residents near rail yards in Los Angeles have complained about noise for decades.[119] A recent study in Teaneck, NJ by Rutgers investigators estimated that 40% of residents living near train tracks where locomotive horns sounded at night had a high probability of waking from the horn noise.[120]

Despite documentation of excessive noise levels and the presence of federal noise limits, many proposed rail yard projects claim that they will generate no significant noise impacts. For example, in 2010, McCalla, AL, residents expressed concerns about potential noise from a proposed Norfolk Southern Railroad intermodal hub,[121] but a consultant hired by the railroad dismissed the noise concerns even before finishing an environmental review.[122]

Neighborhood Impacts - Lighting, Traffic Congestion, Truck and Rail Accidents

When faced with new or expanding freight transportation facilities, concerns of residents often focus on the proximity of neighborhood schools and homes to ports, rail yards, warehouses, and highways. While residents in these communities highlight potential health effects of traffic-related

air pollution and noise, they also express deep concern about their "quality of life." Ports, rail yards, and many distribution centers operate round-the-clock operations, often employing bright lights, often referred to as "stadium lighting." New interest in the effects of bright nighttime lighting has recently emerged, with human studies looking at the effect of bright light and disturbances in sleep, hormones, immune function, and circadian rhythm.[123] A laboratory study in mice has shown that nighttime exposure to artificial light stimulated the growth of breast tumors by suppressing the levels of a key hormone called melatonin.[124] In addition, night lighting (or "bright skies") creates serious negative consequences for animal and bird life.[125]

Occasional train derailments and frequent truck accidents are a common occurrence in goods movement communities. Nationwide, in 2008, more than 4,000 persons were killed in big-rig truck accidents, with 69% of them being occupants of passenger vehicles.[126] Also in 2008, more than 200 persons died at highway-rail grade crossings involving freight railroads.[127] Traffic congested with big-rig trucks is also a common complaint and safety hazard in communities with ports and rail yards. Big-rig trucks constitute 20-25% of the volume of vehicles on the Long Beach Freeway in Southern California, the conduit from the Ports to the major downtown rail yards.[128] The truck route already has 35,000 trucks a day on it, and there is a proposal to triple that number by widening and double-decking the freeway in some sections of its 20-mile route.[129]

Race and Place Issues

According to the U.S. EPA National Environmental Justice Advisory Council report on goods movement:

> *The environmental, public health and quality-of-life impacts of goods movement on communities are more pronounced in areas with major transportation hubs and high traffic roads. Minority and low-income communities near these hubs and throughways bear disproportionate impacts because of their close proximity to multiple pollution sources.*[130]

A screening analysis done for U.S. EPA determined that at least 13 million people, including a disproportionate number of low-income, African-Americans and Latinos, live in close proximity to these facilities and are exposed to higher levels of diesel particulate matter than other residents in their region.[131]

Incompatible Land Uses

As in many communities across the country, land use decisions have resulted in homes, schools, and even parks being located near ports, highways, rail yards, and warehouses. Despite the growing amount of scientific research that shows the direct correlation of health risk with proximity to freeways, rail yards, and diesel emission sources, health considerations typically are not integrated into land use decision-making. In Southern California for example, 65 schools are located within one mile of the I-710 Freeway,[132] a major highway connector from the Ports of Los Angeles and Long Beach for which government officials have proposed an expansion. More than 600,000 residents, including 212,000 under age 18, live within 1,500 meters of the freeway.[133]

Another example of incompatible land uses concerns rail yards in Southern California. In the Los Angeles area, BNSF is proposing to develop a new intermodal rail facility four miles from the ports,[134] and Union Pacific has proposed expansion of its adjacent UP Intermodal Container Transfer Facility (ICTF).[135] Many neighboring residents oppose both projects because they are located in close proximity to schools and established residential communities.[136] Draft environment impact reviews for the two projects are expected in 2011.

At the urging of community and environmental justice groups in California, the California Air Resources Board adopted guidelines for the siting of schools near sources of pollution, such as rail yards, ports, warehouses, and busy highways.[137] In addition, the U.S. EPA recently published draft guidelines on school siting, which suggest buffer zones to protect students from the pollution of highways, rail yards, and ports.[138] These proposed guidelines do not have legal authority, however, and few local governments responsible for land use planning have actively pursued implementation of such approaches. See land use and health section below.

Health Impacts in Asia from Manufacturing Products for Export

The dramatic increase in international trade, especially between developing countries in Asia and the United States, has also resulted in major impacts for the exporting countries as well, where the system of trade is inextricably linked to manufacturing processes that compound negative impacts to worker, health, and communities.

As China's economy has changed to focus on manufacturing and on international trade, occupational hazards have become a major concern. Lead poisoning

(and more recently cadmium poisoning) among workers, for example, has been a ubiquitous problem in - and near - metal smelters in China. Children in Chinese provinces have also suffered. For example, children living near factories that produce car batteries (which contain recycled lead) have suffered from elevated levels of lead in their blood.[139] Protests have broken out in a number of Chinese cities when children have been found with lead poisoning or when authorities have suggested siting new chemical plants there.[140]

Growing awareness and concern focuses on air pollution. A finance organization called 24/7 Wall St. recently performed an analysis to determine the 10 cities with the world's worst air. They reviewed studies on air quality, government data, and information about sulfur dioxide, nitrogen dioxide, and particulate matter. Among the top 10 were the following four cities in China: Beijing (tied with New Delhi, India), Chongqing, Guangzhou and Hong Kong.[141]

A 2006 scientific study investigated high levels of air pollution in the Pearl River Delta region of China, home to hundreds of manufacturing operations and found that the region "produces more than $100 billion of goods annually for export to North America, Europe, and other parts of Asia [and that] 10-40% of emissions of primary SO2 [sulfur dioxide], NOx [nitrogen oxides], RSP [respirable suspended particulates], and VOC [volatile organic chemicals] in the region are caused by export-related activities."[142] This funding has spurred environmental and public interest groups such as Civic Exchange, who advocate for reduction of sulfur content in fuels burned by ships.[143]

Climate Change/Global Warming/ Natural Resource Impacts

Beyond the enormous and multi-dimensional health impacts, international shipping and freight transportation have begun to be identified as major contributors to greenhouse gas emissions and climate change. In fact, a recent study by NASA's Goddard Institute for Space Studies identifies the transportation sector, including trucks, ships, and rail that rely on diesel fuel, as "the greatest contributor to atmospheric warming now and in the near term."[144] Trucking and rail freight in the United States alone accounts for 1.5% of global emissions,[145] and shipping alone accounts for approximately 3-3.5% of total global emissions and 13% of global emissions from transportation.[146] According to the International Maritime Organization (IMO), the shipping industry releases more greenhouse gases than the global aviation industry.[147] On its own, a single container ship is said to emit more greenhouse gases than two thousand diesel trucks[148] and its emissions may contribute to pollution hundreds of kilometers inland.[149]

Shipping releases more carbon dioxide (CO_2) than all but six of the world's nations - more, for example, than is released by Germany.[150] Due to the anticipated growth in world trade, CO_2 emissions from global shipping are expected to rise by 126-218% by 2050 if a business-as-usual approach is followed.[151]

Much of these greenhouse gases come from the shipping industry's use of bunker fuel, a "bottom of the barrel" high sulfur petroleum product. Ships burn bunker fuel in diesel engines and the engines are among the world's highest polluting combustion sources per ton of fuel consumed.[152] Not only is bunker fuel dirty, but much of it is actually imported by China from Venezuela making the transport of the dirty fuel even more polluting in the process of transporting it. California officials are worried about increased concentrations of nitrogen oxides from burning bunker fuel, stating that they are "causing a rise in the acidification of the ocean, since the oceans are the "sink" into which about one-third of all NOx emissions are eventually deposited.[153] Burning bunker fuel in ships also produces harmful black carbon, a carbonaceous (carbon rich) aerosol considered by many to be responsible for climate change, second only to CO_2.[154] A December 2009 study in the scientific journal *Lancet* pointed to the importance of black carbon, which, in combination with ozone, "could together exert nearly half as much global warming as carbon dioxide."[155]

In yet another piece of the fuel supply chain, exports of low-sulfur coal from Montana to China are raising concerns and protests among residents in Oregon, not far from a coal export terminal in Longview, Washington, from which the coal would be shipped to Asia. Residents point out the irony that they are trying to close their own coal-burning generating plants and meanwhile may become the export location for millions of tons of coal each year to China.[156]

Selected Health, Worker & Community Impacts - Freight Transportation

	Hazard	Where it is found, who is at risk	Illness or condition that the long-term exposure or impact can cause
REGIONAL AIR POLLUTION	Particulate matter	Elevated levels of $PM_{2.5}$ in the air	Cardiovascular disease, COPD (e.g., emphysema)
	PM and elemental carbon (EC)	In vehicle exhaust; EC is a marker for diesel	Chronic exposure leads to reduction lung function in children
	Ultrafine particles (UFPs)	In vehicle exhaust; considered very toxic	When lab animals breathe UFPs, some end up in the brain; UFPs can cause artery hardening in lab animals
	Nitrogen dioxide - precursor to ozone	Diesel emissions contains high levels of NO_2	Increase in school absences is linked to increases in ozone levels
LIVING NEAR TRAFFIC-RELATED AIR POLLUTION	Living close to highways	Children	Increased asthma; exacerbation of asthma (e.g., wheezing) and use of more asthma medication
	Living or going to school near a busy road	Children	More likely to develop new cases of asthma
	Living near busy roads	Pregnant women	More likely to have premature or low birth weight babies or miscarriages, or develop preeclampsia
	Living near a freeway	Adults	Thickening of the artery walls that can lead to heart disease and stroke
	Living within 50 meters of a busy road with more than 15,000 vehicles/day	Women	More likely to develop mild cognitive decline as they age
	Living near busy roadways	Women	More likely to develop new cases of diabetes
	Living near busy roads	Men and women	More likely to develop stroke and new cases of heart disease
HIGH NOISE LEVELS	Community noise pollution	At risk: those living near busy highways, marine terminals, airports, rail yards, and train tracks, and/or construction of the above	Residents near airports and highways show (for adults) an increase in cardiovascular disease and stroke, sleep difficulties and anxiety; and (for children) problems with school behavior and anxiety
	Elevated levels of noise in workplaces	At risk: dock workers, railroad workers and truck drivers	Long term exposure can cause hearing loss, stress and high blood pressure
LABOR ISSUES; WORKER HEALTH AND SAFETY	Contingent employment - e.g., warehouse workers	Workers often hired by agencies as temporary workers with low-pay and no benefits	Stressful, insecure jobs without benefits
	Misclassification as independent contractors rather than employees	Port truck drivers	Lack of basic worker protections, such as hourly wage, overtime, health insurance, unemployment benefits, right to organize and OSHA protections
	Diesel exhaust	Dock workers, railroad workers, truck drivers and workers at trucking operations	Increase in lung cancer in all three occupations; increase in COPD (e.g., emphysema) among railroad workers
	Heat	Lack of air conditioning in cabs of trucks and locomotives and inside huge distribution centers	If outdoor temperatures are extremely high and there is no relief or mitigation, workers can suffer from heat stress illnesses
	Injuries/fatalities	Some parts of the freight transportation industry are considered "high hazard"	E.g., The 2009-2010 California OSHA highest hazard industry list included warehousing and truck transportation
COMMUNITY IMPACTS	Industrial blight	Empty containers in lots near homes; views of industrial cranes	Decreases home values and quality of life
	Traffic congestion	Cars must travel with big-rig trucks; expanding number of heavy duty trucks hauling containers; truck driving schools operating in neighborhoods	Stress from congestion; increased commuting time means longer times on the road breathing air pollution in exhaust from cars and trucks
	Cars traveling in same lanes and on same highways	Expanding number of heavy duty trucks	Injuries and fatalities in car-truck accidents
	24-hour lighting	Lights shine in windows	Difficulty sleeping at night
	Road repairs	Highways, truck routes, residential streets near rail yards, ports and warehouses	High cost to local and state taxpayers to repair the roads and highways from big-rig truck damage
	Eminent domain	Exerts the right of railroads or governments to appropriate private property (e.g., to build a highway)	Community residents can lose their homes

Part II. The Changing Landscape: Organizing Strategies and Policy Innovations

As the global system of trade expands and becomes more flexible in its operations, disparities in health, work, and community quality of life are clearly visible at the local, regional, national, and international levels. While the freight transportation industry uses its political muscle to push forward with its publicly subsidized plans for expansion, communities are trying to reframe the debate about responsible and equitable development. Across the country in places like Southwest Detroit, the Harbor area of Los Angeles, West Oakland, Gardner, KS, Newark, NJ, and Gulfport, MS, communities have responded by organizing and advocating for policies that encourage clean air, good jobs, and livable neighborhoods. Despite the limited focus on community, environmental, health, and labor issues in how the dominant global trade and goods movement politics are framed, this organizing and advocacy around the country has resulted in important policy innovations. Increasingly, local groups have begun to work at the regional level and build state and national coalitions and networks to address what is a non-transparent, massive global system of trade and distribution of products. In doing so, these organizations have staked out a role in decision-making that affects their health and long-term well-being.

The emerging and increasing influence by health, labor, environmental justice and community organizations suggest that future political debates over freight transportation may become increasingly contentious. This section of the report provides an overview of freight transportation activity, organizing, and advocacy across the country in 17 locations where seaports and/or freight distribution hubs are located. Regions were selected because of the large size of their ports and freight transport activity, others because they have significant organizing efforts to prevent/reduce diesel emissions and community/ worker impacts, and still others because expansions are planned and few residents or community groups seem to be aware or engaged in evaluating these potential impacts. Through review of published sources, interviews, and analysis of socioeconomic data sources we profile existing and proposed port and freight transportation projects, document negative local and regional impacts, and identify visible and emerging examples of organizations and strategies specific to ports and freight transportation. See Inset on pages 26-27 for a list of groups identified in the study. See Appendix for contact information.

California

Los Angeles/Long Beach, California

Issues

The adjacent ports of Los Angeles and Long Beach make up the San Pedro Bay Ports complex, the nation's largest and 7th largest port facility in the world. Within the national landscape of freight transportation, the San Pedro Bay Ports represent a strategic hub not only for the West Coast but also for the nation. More than 40% of the nation's imports come through these two ports, making Los Angeles and Long Beach critical to the system of trade nationally and globally.[157]

The Port of Los Angeles encompasses 7,500 acres including 27 cargo terminals and includes on-dock intermodal facilities and rail yards.

Despite their already large size, the Ports of Los Angeles and Long Beach continue exploring ways to expand. Governed by separate and often competing Harbor Departments in the adjacent cities of Los Angeles and Long Beach, the two Ports handled 11,816,592 TEUs in 2009;[158] that number is expected to more than double by 2030.[159] The Long Beach Port, for example, is moving ahead with its $750 million Middle Harbor Project that would allow the port to replace three smaller container terminals with one larger facility including an on-dock rail yard. Once complete, the project will increase the size of the two existing terminals from 290 acres to 345 acres and double the cargo-handling capacity of the three piers.[160] In addition, the Port will modernize its ITS container facility and expand on-dock rail operations on Pier G.

The Port of Long Beach also has begun development of its $1.1 billion project to replace the 40-year-old Gerald Desmond Bridge with a larger, higher bridge that will accommodate more traffic and allow passage underneath of the largest vessels in operation today.[161] Currently, about 15% of total U.S. containerized imports move across the bridge.[162] Other proposed projects include a new terminal (Pier S) on existing vacant land at the port with an estimated project cost of $650 million, and expansion of on-dock rail at Pier B.[163]

The Port of Los Angeles has also embarked on a five-year, $245 million expansion of the TransPacific (the TraPac project) that will deepen the waterside at Berths 144-147, upgrade approximately 50 additional acres of existing land to modern container handling standards, and construct a new on-dock rail facility.[164] Another project involving the Terminal Island facility will provide for the redevelopment and expansion of the existing container terminal by 56 acres, for an approximate total of 346 acres.[165] And finally, the China Shipping container terminal will be expanded from an existing 72 acres to 142 acres of backland and 2,500 feet of wharves served by 10 Post-Panamax A-frame cranes, the new equipment required to unload the massive Post-Panamax ships. Expansion is expected to increase the terminal's capacity to handle 1.4 million TEUs a year.[166]

Although the ports are developing new and expanded rail yards on-dock, they rely heavily on a regional intermodal train network to access other rail yards throughout Southern California - some the largest in the country. The major rail corridor is the Alameda Corridor, a $12 billion, 24-mile stretch of rail that links the Port of Los Angeles to the massive rail yards in East Los Angeles and Commerce. The corridor is governed by a joint powers authority that involves both cities as well as the regional transportation agency, Los Angeles County Metropolitan Transportation Authority ("Metro").

Four miles north of the Ports is a proposed expansion of an existing intermodal facility, the Union Pacific Intermodal Container Transfer Facility, which currently handles 750,000 containers a year. Next to that rail yard is proposed a new rail yard - the Southern California International Gateway (SCIG) facility, which would be built and operated by BNSF. If the ICTF is expanded and the SCIG is built, the two yards together would handle three million containers a year. Residents adjacent to the two yards have been voicing strong opposition since 2005 specifically targeting the Port of Los Angeles that owns the land for both of these projects. Meanwhile, in Commerce, CA alone there are four yards, with the BNSF Hobart Yard being one of the largest intermodal rail facilities west of the Mississippi River. In San Bernardino, the rail yard owned by BNSF produces the highest diesel cancer risk of any rail yard in the state of California, due to the way the wind blows and the proximity of homes to the rail yard.

Both Ports support expansion of the I-710 Freeway, a truck-congested highway running from the Ports to the Commerce/East Los Angeles rail yards. The project proposes to add lanes and double-deck the freeway in some sections, tripling its capacity. The proposed project would affect an 18-mile stretch of freeway that goes through 15 cities in close proximity to schools, hospitals, daycare and senior centers.

Groups

The scale and negative impacts of port operations and goods movement in Southern California are the subject of numerous scientific studies as well successful organizing, advocacy and policy campaigns in Los Angeles which have changed the way the ports and freight transportation industries do business throughout the country. For goods movement activists, what happens in Los Angeles/Long Beach has become

a benchmark for opportunities and challenges for organizing and policy change. Historically, the International Longshore and Warehouse Workers Union (ILWU) launched statewide actions by focusing on the Los Angeles/Long Beach Ports to secure contracts for its dockworkers along the West Coast. Today, there are more than 15 organizations (community-based, environmental, academic, and labor) with full-time staff working on port/goods movement issues in the Los Angeles/Long Beach area.

Community-based and Environmental Justice Organizations

Community-based and fenceline environmental justice community organizations near Los Angeles ports and highway and rail corridors have engaged in direct organizing and resident education and the need to prevent new, and reduce existing, negative health and environmental impacts to residents and workers.

- **Communities for a Better Environment (CBE)** is an environmental justice group that works to protect communities suffering disproportionately from environmental pollution caused by refineries, power plants, and mobile sources such as diesel trucks. CBE's team of researchers, lawyers, and organizers focus strategies in the communities of Wilmington, Huntington Park and South Gate.

- **East Yard Communities for Environmental Justice (EYCEJ)** was formed in the City of Commerce (20 miles north of the Ports of LA and Long Beach) to provide a resident perspective in decisions related to four freight rail yards in the community. In addition to involvement in THE Impact Project, EYCEJ is also a key organization addressing the negative impacts of the expansion of the I-710 (Long Beach) Freeway that divides the communities in Southeast Los Angeles County. In addition to community base building strategies, EYCEJ has led statewide advocacy efforts to strengthen regulation related to rail yards and has begun work with other health and environmental justice organizations in the state to focus on health considerations in state level policies related to federal Transportation Reauthorization.

- The **Coalition for a Safe Environment (CFASE)** is based in Wilmington, adjacent to the Port of Los Angeles. CFASE regularly responds to draft environmental impact review documents, an arduous, technical and time-consuming effort because many of the documents are 2000+ pages long. CFASE is a leader in advocating for alternative, zero emissions technologies to move containers. CFASE has also been involved in precedent setting legal actions to secure community benefits from port expansion projects. See discussion of electrification of the China Shipping Terminal and the fund settlement negotiated from the TraPac expansion.

- The **Long Beach Alliance for Children with Asthma (LBACA)** is a project of Memorial Care Hospital in Long Beach. LBACA educates residents about the connection between environmental factors (including air pollution) and asthma. LBACA works closely with mothers in the impacted neighborhoods to conduct traffic counts and engage in research and advocacy on behalf of their children who suffer from asthma. In addition, LBACA provides education and training to improve the skills of physicians when they provide care at community provider sites.

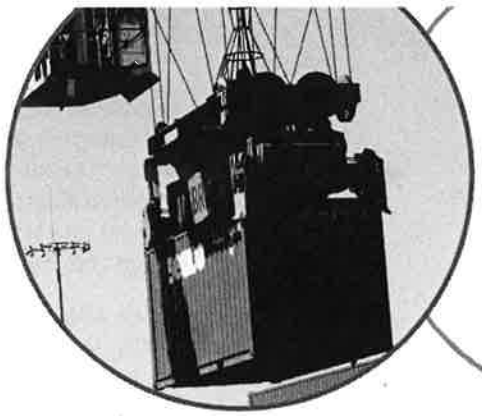
- The **Long Beach Interfaith Community Organization**, a PICO-affiliated faith-based organization, educates congregational and school leaders in Long Beach to become active in ports and goods movement discussions.

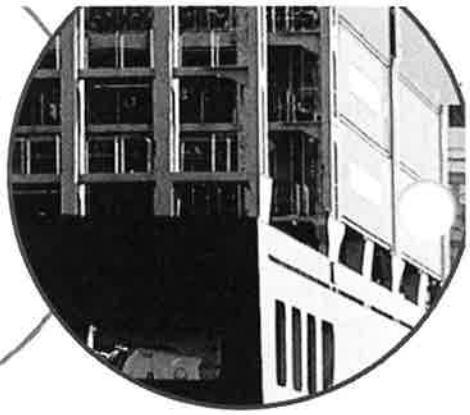
These groups have educated and organized residents to share important community perspectives and knowledge, inform scientific studies on community health risks and exposure, and mobilize residents to participate in policy debates. Several of the above grassroots organizations have been bolstered by funding and programmatic support from The Liberty Hill Foundation, a progressive foundation dedicated to social change and building grassroots leadership.

Environmental groups

Environmental organizations have played key roles in advocating for policy changes to achieve cleaner air at the Ports and in goods movement operations.

- The **Natural Resources Defense Council (NRDC)** has played a leadership role in Southern California on issues of air pollution, ports, and freight transportation through its technical and legal expertise. In 2002, for example, NRDC filed a lawsuit on behalf of community and environmental plaintiffs against the City of Los Angeles and its Harbor Department for failing to comply with the California Environmental Quality Act (CEQA) when it reviewed the proposed expansion of the China Shipping terminal at the Los Angeles port. The result: a settlement that included electrification of the China Shipping Terminal and a shift in the shipping industry to implement health protective technologies. The precedent has pushed the Port of Los Angeles to advance





Select Groups Engaged in Ports and Freight Transportation*

Los Angeles/Long Beach

Community-based Organizations
Coalition For A Safe Environment
Communities for a Better Environment
East Yard Communities for Environmental Justice
Communities for Clean Ports/End Oil
Long Beach Alliance for Children with Asthma
Long Beach Interfaith Community Organization

Local and Regional Coalitions
Coalition for Clean and Safe Ports
Port Work Group, Green LA Coalition
The Trade, Health and Environment Impact Project (THE Impact Project)
Coalition for Environmental Health and Justice

Environmental Groups
Coalition for Clean Air
Natural Resources Defense Council

Homeowner Associations
Peninsula-San Pedro Homeowners
West Long Beach Neighborhood Association

Research and Academic Institutions
Southern California Environmental Health Sciences Center, based at University of Southern California
Southern California Particle Center, based at UCLA
Urban and Environmental Policy Institute, Occidental College

The Inland Valley: San Bernardino and Riverside
Center for Community Action and Environmental Justice
Clergy and Laity United for Economic Justice
Inland Valley Action Network
Partnership for Working Families
Warehouse Workers United/Change to Win

Oakland/East Bay
Ditching Dirty Diesel Collaborative
East Bay Alliance for a Sustainable Economy
Pacific Institute
Regional Asthma Management and Prevention
West Oakland Environmental Indicators Project

Central Valley, CA
Central Valley Air Quality Coalition
Natural Resources Defense Council
Center for Race, Poverty & the Environment
Greenaction
Fresno Metro Ministry

San Diego
Environmental Health Coalition

California Statewide Coalitions
California Diesel Rule Work Group
Statewide Environmental Justice, Health and Freight Movement Policy Project

Pacific Northwest: Seattle and Tacoma
Puget Sound SAGE
Washington CAN
Washington Coalition for Clean and Safe Ports
University of Washington, School of Public Health

The Gulf Coast
Air Alliance Houston
Clean Economy Coalition
Community In-Power and Development Association
Gulfport Community Land Trust
Mississippi Center for Justice
Sealy Center for Environmental Health Sciences at the University of Texas Medical Branch

Southwest Network for Economic and Environmental Justice
STEPS Coalition / Partners for Safe & Healthy Port Campaign
Texas Environmental Justice Advocacy Services
Turkey Creek Community Initiatives

Detroit
Detroit Community-Academic Urban Research Center (University of Michigan, Schools of Public Health, Nursing and Social Work and Detroit Department of Health and Wellness Promotion)
Detroit Hispanic Development Corporation
Detroiters Working for Environmental Justice
East Michigan Environmental Action Council
Southwest Detroit Community Benefits Coalition

Chicago
Center for Urban Economic Development, University of Illinois at Chicago
Citizen Action, Illinois
Greater Englewood Community and Family Task Force
Illinois Campaign to Clean up Diesel Pollution
Respiratory Health Association of Metropolitan Chicago
Warehouse Workers for Justice
Warehouse Workers United/Change to Win
Will County Residents for Responsible Intermodal Development

Kansas City
Hilldale Environmental Loss Prevention
Johnson County Intermodal Coalition
Natural Resources Defense Council
Sierra Club

New York/New Jersey
Center for the Urban Environment, John S. Watson Institute for Public Policy, Thomas Edison State College
Coalition for Healthy Ports
Environmental and Occupational Health Sciences Institute, University of Medicine and Dentistry of New Jersey
Ironbound Community Corporation
Garden State Alliance for a New Economy
New Jersey Environmental Federation
New Jersey Environmental Justice Alliance

Baltimore
Environmental Justice Partnership
NIEHS Center in Urban Environmental Health, Johns Hopkins Bloomberg School of Public Health

Philadelphia
Clean Air Council

Hampton Roads and Southwest Virginia
Citizens for the Preservation of our Country

Savannah, Georgia
Citizens for Environmental Justice/Harambee House
First African Baptist Church
Southern Environmental Law Center

Charleston, South Carolina
Department of Epidemiology and Biostatistics, University of South Carolina
The Lowcountry Alliance for Model Communities
South Carolina Coastal Conservation League
New Rosemont Homeowners Association
Southern Environmental Law Center

Jacksonville, Florida
Mayport Village Civic Association

* These are examples of groups identified in regions profiled in this study

electrification projects at the port including at the proposed China Shipping expansion project.

- The **Coalition for Clean Air** (CCA) is a statewide environmental advocacy organization focused on a broad set of issues related to air quality in California. CCA's "Preventing Port Pollution" project focuses on securing policies that reduce pollution from ports and freight transportation throughout the state. Working with coalitions such as the Port Work Group of the Green LA Coalition and the Coalition for Clean and Safe Ports (see below), CCA's has provided leadership roles in efforts to adopt the Clean Air Action Plan, implement the Clean Trucks Program at the Ports, and ensure health and air quality protections with the I-710 Freeway expansion. Its advocacy work also includes efforts to reduce air pollution in the Central Valley of California.

Local and regional coalitions

Some of the ability of these community-based groups to influence policy is due to effective local and regional coalitions that serve to bring diverse organizational missions together for long-term work or for specific issue campaigns. These include:

- The **Port Work Group**, a working group of the citywide (Los Angeles) Green LA Coalition, that brings together environmental justice, environmental, and public health organizations, with support from academic researchers.[167] The Port Work Group worked with groups throughout the state to come up with a set of health and environmental justice criteria to apply to projects funded by the $2 billion Trade Corridors Improvement Fund (TCIF).[168] The PWG also pushed for the passage of the Clean Air Action Plan (CAAP), adopted in 2006 by the Ports of Los Angeles and Long Beach, the first air emissions policy to be developed jointly between the two port authorities in the United States (and one of the first times that the two competitive ports in Southern California have ever collaborated). Currently, the PWG along with community organizations in Long Beach are engaged in a campaign to convince the Ports of LA and Long Beach to adopt on-dock rail, alternative technologies, and health protective land uses instead of approving the SCIG and the ICTF rail yard expansion projects. See above. The Port Work Group includes all of the groups listed above as well as Communities for Clean Ports, a Long Beach organization focused on clean ports and alternative energy.

- The **Coalition for Clean and Safe Ports**, made up of labor, community, environmental, environmental justice, faith and community organizations (including many of the groups mentioned above), forged a successful campaign to push the Harbor Department and City of Los Angeles to adopt a Clean Trucks Program at its Port. The program calls for replacement and retrofits of approximately 16,000 trucks in order to meet the 2007 federal EPA emissions standards by 2012. To achieve these goals, the program featured a "concessionaire model" that requires trucking companies who service the Port to hire truck drivers as employees in return for securing concession contracts with the Port. Currently, truck drivers are misclassified as "independent contractors" even though they lease a truck from their employer and have to report to their employer. Although the Coalition also pushed for a similar program at the Port of Long Beach, decision-makers there decided to pass a plan that required retrofits without addressing the issue of employee status facing port drivers. Through the Coalition, organizers have connected resident concerns about the neighborhood impact of goods movement and ports operations, e.g., trucks parking in residential areas to issues of drivers and the port trucking industry. The Coalition is staffed by the Los Angeles Alliance for a New Economy (LAANE), an organization with strong relationships with labor, including the Los Angeles County Federation of Labor, and links to similar coalitions in other port and goods movement communities.

- The **Trade, Health and Environment Impact Project (THE Impact Project)**,[169] a community-university collaboration that brings together scientific research from USC and UCLA (see below), global trade analysis from Occidental College (see below), and a community perspective from four of the environmental justice organizations discussed above (CCAEJ, CFASE, EYCEJ, and LBACA). This collaborative functions as a hub for information and resources as well as local and regional campaigns. USC and THE Impact Project have hosted three major conferences on the impacts of port and goods movement that have drawn scientists and advocates from across the United States and internationally. THE Impact Project provides important technical assistance and peer-to-peer support to the growing number of participants of these conferences. Members of THE Impact Project have visited Kansas, South Carolina, Washington, and Georgia to give talks about the health impacts of ports and goods movement or provide technical assistance to community and environmental justice

groups. Each group in THE Impact Project has a Neighborhood Assessment Team ("A-Team") composed of members who have been trained to count traffic and measure air pollution.[170]

- **Coalition for Environmental Health and Justice (CEHAJ)** works to ensure that health issues are the number one priority in plans to expand the Long Beach (I-710) Freeway, which runs from the Port of Long Beach to the rail yards in East Los Angeles/ Commerce. Involved groups include EYCEJ, CBE, CCA, and NRDC. Organizing by these groups, along with testimony provided by USC and UCLA faculty and staff resulted in the I-710 Project Committee voting to support conducting a HIA as part of the environmental review for the proposed expansion of the I-710. The HIA will help to measure comprehensive impacts that may not be addressed in traditional EIRs/EISs. For the I-710 expansion project, the HIA will focus on additional areas of: air pollution, noise, light, congestion and mobility, displacement, economic effects, access to community resources and segregation and isolation.[171]

Homeowner Associations

In Los Angeles and Long Beach, homeowner associations have worked in coalition with environmental, health, labor and other community organizations to link homeowner concerns with broader concerns of public health. During the 1990s and until 2001, the **Peninsula-San Pedro Homeowners Association** was one of the only groups regularly attending Port of Los Angeles Harbor Commission meetings and hearings, demanding more accountability, and engaging in lawsuits to ensure that environmental health impacts were considered in port expansion projects. Since then the Association has worked with other groups, including the coalition that appealed the TraPac decision and negotiated a community benefits settlement from the Port. See discussion of the TraPac community benefits agreement in the Policy section later in the report. The **West Long Beach Neighborhood Association** educates its members around the expansion of rail yards in that community and organizes residents to attend hearings.

In addition to the range of organizations focused specifically on ports and goods movement, Los Angeles is home to a robust set of social, environmental, and economic justice organizations that have won significant policy campaigns. These include a citywide living wage policy, community benefits agreements from major development projects, prevention of siting of polluting industries, and public funding for innovative workforce development programs. Because of this broader and established organizing and advocacy infrastructure, individuals or groups organizing around ports and goods movement are able to draw from experience as well as political influence of these organizations, helping to build a progressive movement through a focus on ports and goods movement.

Academic research centers

A number of academic and research centers in Southern California focus on the health, labor, and community impacts of freight transportation.

- The **Urban & Environmental Policy Institute (UEPI)** at **Occidental College** is a community-oriented research and advocacy organization with projects that include food justice, globalization and trade, the built environment, and transportation. As a partner of THE Impact Project, UEPI has brought the food supply chain into focus as a global goods movement issue.[172] A UEPI faculty member also facilitates the Los Angeles Port Work Group and is part of a facilitation team for the Statewide Freight, Health and Environmental Justice Project (see below).

- Scientific investigators from both USC and UCLA participate in two research centers: the **Southern California Environmental Health Sciences Center,** funded by the National Institute of Environmental Health Sciences (NIEHS) and the **Children's Environmental Health Center,** funded by U.S. EPA and NIEHS. USC scientists conduct the Children's Health Study of the effects of air pollution on children's respiratory health as well as other studies related to health effects in Los Angeles' Harbor Communities. The Centers' Community Outreach and Engagement Program seeks to ensure that public health becomes an integral part of transportation decisions, including ports, rail yards, highways, and warehouses. The outreach program coordinates THE Impact Project and a key organizer of the Moving Forward Together conferences.

- **Investigators** at the **Southern California Particle Center**, based at the University of California, Los Angeles (UCLA) have been conducting exposure assessment and toxicologic studies of ultrafine particles for the past 11 years, and are conducting a study of particle toxicity at rail yards.

- **USC & UEPI** are taking the lead in developing a communications network that can help those involved in freight transportation and public health campaigns around the country to share information and strategies for change.

The impact of these organizations working independently and also collaborating in various coalitions has resulted in a growing capacity of groups able to work with and directly challenge port and freight transportation decision-making that does not place a high priority on community, worker, and environmental impacts.

The Inland Valleys: San Bernardino and Riverside Counties, California

Issues

As one of the fastest growing metropolitan areas of the country prior to the Great Recession, the Inland Valleys are home to the nation's boom-and-bust housing market as well as the nation's largest conglomeration and most rapidly expanding group of warehouses and distribution centers. Between 2000 and 2005, the influx of new residents to the region represented one-fifth of California's population growth.[173] In 2008, more than 80% of Southern California industrial space under construction was located in these two counties; approximately 159 million square feet of industrial space entered the Inland Valley's market between 2000 and 2008.[174] Local boosters and policymakers have pushed a pro-growth agenda that encouraged the construction of speculative housing and large scale warehouses along the region's major transportation corridors

These warehouse, some larger than 1.5 million square feet,[175] are critical to freight transportation and logistics industries that require the vast buildings to store, load and distribute goods. In Southern California, 44% of the state's existing warehouse space, or approximately 36.5 million square feet is located in San Bernardino and Riverside Counties; 60.4% of the region's available (empty) warehouse space and 62.4% of available industrial land parcels that could be used for warehousing are located in these two counties.[176]

Similar to other goods movement communities, the combination of warehouse construction, increased port-related truck and train trips, and a growing number of low-income communities of color have led to heightened economic and environmental health disparities. People of color represent 65% of San Bernardino County's population and 58% of Riverside County's residents; in 2009 poverty rates were 17% and 14% respectively.[177]

Freight transportation and logistics industry activities, particularly the construction of massive warehouses and intermodal rail facilities, have turned parts of the region into environmental justice hotspots that threaten the health and quality of life of local communities. Pollution from trucks traveling to these warehouses, commuters driving cars far distances to work, chemical reactions that create smog from vehicle and industrial pollution blown in from Central Los Angeles, railroad operations and other industrial practices - all contribute to making the Inland Valleys one of the most polluted areas in the country. In 2000, the South Coast Air Quality Management District found that Mira Loma Village, a low-income Latino community in Riverside County had the highest levels of particulate pollution in the nation.[178]

The conversion of former military bases into public-private redevelopment projects has opened up large tracts of land for further goods movement development with government agencies providing subsidies for these new projects. For example, George Air Force Base (located in Victorville, San Bernardino County) is being developed into Global Access, a 8,500 acre airport, rail and logistics complex; March Air Force Base as the 1,290-acre logistics-focused Meridian Business Park (Riverside County); and Norton Air Force Base as a logistics hub developed by Alliance*California (San Bernardino County).[179] Building upon existence of air fields, these inland hubs now include facilities such as free trade zones and U.S. customs offices designed to facilitate foreign imports by air as well as facilitating freight transport from the ports of Los Angeles and Long Beach.

Until recently, local and regional planning agencies in the Inland Valleys have been able to advance goods movement industrial growth without accounting for the low wages and increased health risks the industries produce. Industrial and residential developers are the largest contributors to local electoral campaigns and have launched successful recall efforts against local officials who oppose new warehouse construction.[180] The power of these developers over many of the region's political jurisdictions leaves many communities with no political recourse and/or political power to influence debates on growth and development. The region's large populations of young people of color and immigrants who do not or cannot vote further concentrate political power in the hands of pro-developer interests. In 2008, 37% of the population in Riverside County and 40% of San Bernardino were 24 years or younger.[181] Community and labor organizers recognize shifting trends in demographics and see the potential to develop this new electorate into a more progressive force in the political base of the Inland Valleys in the future.

Groups

Environmental justice organizing in these two counties has been anchored and led by the **Center for Community Action and Environmental Justice (CCAEJ)** since the 1970s when community activists targeted the largest Superfund site in the country. Combining grassroots organizing and

leadership development with research and policy advocacy, CCAEJ supports grassroots leaders to push back against the expansion of massive retail corporation distribution centers near homes and to reduce the diesel emission impacts of rail yards in Mira Loma, Colton and San Bernardino.

Organized labor has struggled to build significant bargaining power in the region's primary logistics and residential construction industries. Other labor and worker justice organizations such as **Clergy and Laity United for Economic Justice (CLUE), Partnership for Working Families** (PWF) and the **Inland Valleys Labor Action Network (IVLAN)** have facilitated relationships and potential partnerships between organized labor and community organizations. Unions affiliated with **Change to Win** have turned attention to goods movement industries. The **Warehouse Workers United** campaign, for example, has focused attention on the growing temporary, contingent workforce related to the expansion of warehouse and distribution centers in the Inland Valleys. The **United Food and Commercial Workers** union is engaged in a worker organizing campaign focused on major food retailers such as Walmart and Tesco (Fresh & Easy).

These new collaborations, along with shifting demographics and electoral campaigns, seek to build more progressive politics in the Inland Valleys. Organizing around the major presence of the goods movement industry has galvanized residents, developed relationships and set in motion a shift in the way that health and environmental justice organizing and policymaking occurs. Demographic changes in the Valleys also suggest that pro-development elected officials may shift under the weight of shifting demographics. Young Latinos for example will age into a potentially powerful voting block. The community, labor, health, and environmental organizations will play an important role as these young leaders and voters begin to exert electoral influence over the political and policy landscape in the Inland Valleys.[182]

Oakland/East Bay, California

Issues

The Port of Oakland, the 6th largest port in the country, is located along 19 miles of waterfront on the San Francisco Bay and includes 20 deepwater berths and 35 container cranes, 29 of which are Post-Panamax size.[183] The Port of Oakland handles nearly all of the containerized goods moving through Northern California[184] that are transported from the port through the low-income neighborhood of West Oakland and onto the region's major highways and rail lines. According to the Alameda County Public Health Department, West Oakland has cancer risk rates twice as high as other Bay Area residents[185] and the highest rates of asthma hospitalization in the county.[186]

The Port has completed a $600 million expansion that includes two new marine terminals, an intermodal rail terminal, realignment of roadways, dredging, and a 38+ acre public waterfront park for education and wetland estuary. The design vessel for the project is a container ship that transports more than 6,500 TEUs. It has a design draft (depth in the water) of 46 ft. that is 1,139 ft. long and 140 ft. wide. The -50 foot dredging project will also support transitioning four military bases to civilian use. The Port argues that it is essential for this project to occur if it is to remain internationally competitive. The dredging project is designed to maintain and improve Oakland's position as an international cargo gateway.[187]

Groups

In response to this expansion, environmental justice and health advocates along with neighborhood activists and a labor community coalition have begun to coalesce and have elevated issues of worker, health, and community participation in ports and freight transportation discussions. Some of this organizing has occurred in West Oakland, the neighborhood along the fence line of the Port of Oakland and the former Oakland Army Base currently under redevelopment. The West Oakland neighborhood is utilized as the major truck route to and from the port and for freight rail traffic to and from the UP and BNSF rail terminals. Organizing around the impacts of freight transportation in the East Bay Area is represented by:

- The **West Oakland Environmental Indicators Project** (WOEIP), a community-based research project developed between community residents and the Oakland-based Pacific Institute, a research institute that works to advance environmental protection, economic development, and social equity. The community knowledge and research work of the WOEIP has established a scientific framing of the issues related to diesel, air pollution, and health for the community-based campaigns. With research support from the Pacific Institute, WOIEP published a report, "Clearing the Air: Reducing Diesel Pollution in West Oakland" which documents the diesel emissions and the impact pollution has on West Oakland.[188]
- **The Pacific Institute**, a research intermediary based in Oakland provides research support, targeted technical assistance and capacity building with Bay Area community organizations to develop capacity to engage in planning and policies that affect them. The Institute helps convene and organize scientific and popular education resources to support

groups. For example, along with community-based and health organizations in the East Bay, the Institute is working on issues of diesel and rerouting truck routes that run from the Port north toward Sacramento. The Institute published a report on the cost of the impacts of freight transport and has developed popular education materials on goods movement and health.[189]

- The **Regional Asthma Management and Prevention** (RAMP) program works to reduce the burden of asthma by facilitating collaboration among a diverse group of stakeholders focused on activities ranging from clinical management to environmental prevention, with a particular emphasis on those communities inequitably burdened by the disease. RAMP's freight transportation advocacy includes work to monitor the California Air Resources Board in order to prevent or minimize any rollbacks of diesel reduction regulations as well as track freight transportation and climate change policy at the state level. At the regional level, RAMP focuses on incorporating freight transportation and health issues into the development of the regional plans under the implementation of SB 375, California's land use policy to address climate change.

Several campaign-specific coalitions have formed that focus on freight transportation and ports and that investigate the root causes of negative health, labor, and environmental, and community conditions. WOIEP, RAMP, and Pacific Institute are core members of the **Ditching Dirty Diesel Collaborative** (DDDC) a Bay Area-wide coalition of individuals, community based organizations, public agencies, and health organizations focused on reducing diesel and its negative impacts, particularly in low-income communities of color. Through advocacy, education and community mobilization, the DDDC focuses on truck idling and freight transport through the Bay Area.

The **East Bay Alliance for a Sustainable Economy** (EBASE) a social justice organization based in Oakland coordinates the Oakland campaign of the Coalition for Clean and Safe Ports, the labor, community, environmental justice, and faith coalition to implement a Clean Trucks Plan based on the employee concessionaire model passed in Los Angeles (discussed later in this report). The Coalition includes member organizations from the DDDC, and DDDC staff serves on the CCSP's executive committee. In 2007 EBASE wrote a report, produced for the CCSP titled that documents the negative impacts of industry trucking operations on workers and public health in Oakland and the region.[190] With the Pacific Institute, EBASE conducted research in 2009 and found that health impacts from truck pollution in the Oakland region total $153 million a year.[191]

Similar to Los Angeles, ports and goods movement-focused organizations in Oakland also draw on a broader landscape of social justice, labor, public health, and environmental organizations.

Central Valley: Stockton, California

Issues

The Port of Stockton in the Central Valley of California positions itself as a key facilitator of the movement of bulk cargo serving the agriculture and construction industries of the Central Valley. A recent Brookings Institution report noted the importance of San Joaquin County where the Port of Stockton is located, as a key to exporting California's agricultural products.[192] The port is approximately 75 miles inland from San Francisco Bay and the Pacific Ocean. Inbound ships sail past San Francisco, Oakland, Redwood City, Richmond, Benicia, Pittsburgh, and Sacramento to dock in Stockton to take advantage of the direct access to Interstate 5 - the north-south corridor in California.

In 2004, the Port of Stockton expanded by incorporating and assuming control over Rough and Ready Island, a 1,561 acre area once part of the United States Navy Supply Depot. The redevelopment project area, now designated for port related uses, is being developed into industrial, warehousing, and other Port-related uses. In 2010, the Port received a $30 million federal ARRA/TIGER grant to develop its "highway to sea" waterborne shipping route between Oakland, Stockton and West Sacramento, which will move goods between the Port of Oakland and the Port of Stockton and West Sacramento via waterways instead of trucks having to rely on the Altamont highway.[193] This "Green Trade Corridor" project seeks to capitalize on the waterways that link the three ports to facilitate goods movement from Oakland through Sacramento and Stockton.[194]

Groups

Concerns over Port operations and redevelopment projects have emerged among established homeowner associations and environmental organizations concerned about the ecosystems surrounding the deep-water channel and the delta region. The Deep Water Ship Channel, the Port's primary waterway, is on the federal list of highly polluted waterways, contaminated by toxic chemicals from Navy operations, large-scale agriculture, and gold mining activities as well as by port ships.

Homeowner groups who live across the channel from the Port and Rough and Ready Island along with environmental organizations (including NRDC) have stopped port projects and pushed the Port to agree to mitigations for dredging and development projects. That included two major lawsuits. The first, filed in 2004, challenged approval of a massive 1400 acre port expansion project that would have tripled the size of the port to include a marine terminal, commercial and industrial development and result in more than 13 million additional vehicle trips annually.[195] A second, in 2007, was against the Port of Stockton and the Army Corps of Engineers to halt the dredging in the San Joaquin River due to inadequate review of the impacts the dredging and port expansion would have on people living in the surrounding communities and the river. A settlement was reached in the 2004 lawsuit that requires the Port to establish a $5 million air quality mitigation fund, an amount that will be reduced if the port requires its tenants to utilize clean trucks in their operations. The port also agreed to reduce dock fees to ship operators using cleaner fuels, impose mandatory idling restrictions on tugboats and trucks, and require use of electrical hook-ups for tugboats.[196]

Despite port expansions and significant recent public investment in port projects, there is no visible organized community-based environmental justice and health advocacy focused on the Port of Stockton. Stockton resident Maya Abood, who organizes for Pesticide Watch in Stockton, suggests that although there are no actively involved groups focused on the Port, other organized groups may be poised to move that direction. "No one has made the link between food, imports, and the Port and how it relates to low-income immigrants who make up much of Stockton's population. A community group has worked hard to establish a community garden on county land close to the Port but has little idea about the hazards and toxics that might exist."[197]

Increasing ports and freight transportation related organizing and advocacy of environmental justice and health organizations in other parts of the Valley might hold promise for Stockton. For example, the **Central Valley Air Quality Coalition (CVAQ)**, a coalition organization based in Fresno, includes more than 70 member organizations focused on air quality in the Central Valley, including curbing diesel emissions from agricultural trucks. CVAQ has played an important role in creating linkages between air quality and environmental justice advocates in the Central Valley with other health and labor organizations in Northern and Southern California. CVAQ priority areas include a focus on the relationship between air quality, regional transportation, and land use planning required under SB 375 (passed in 2008) that requires the state's Air Resources Board to develop regional greenhouse gas emission reduction targets for passenger vehicles. Working in collaboration with groups in the Bay Area (Pacific Institute, RAMP, Greenaction, and Ditching Dirty Diesel Collaborative), CVAQ sees regional planning under SB 375 as important process through which emissions reductions can be reduced in the heavily impacted Central Valley.

Member organizations within CVAQ have also organized their respective campaigns in the Central Valley to address air quality and environmental justice. The **Center on Race, Poverty & the Environment** (CRPE) uses organizing, legal, policy and advocacy strategies to focus on improving air quality by holding mega-dairies and government accountable for environmental pollution, policy discussions related to the state compliance with the federal Clean Air Act, issues of climate change and land use planning, and eliminating health impacts from agricultural pesticide use. As a member of the Central Valley Air Quality Coalition (CVAQ), CRPE provides legal, policy and organizing knowledge and experience to the discussions about air quality, health, and freight transportation in the Central Valley. **The San Joaquin Valley Environmental Health & Justice Project of Greenaction** focuses on reducing diesel emissions due to goods movement and waste movement in primarily low-income communities who suffer disproportionate rates of disease. **Fresno Metro Ministry's Environmental Health Program** focuses on air quality and improving environmental health in the Fresno area. It is a member of CVAQ and the San Joaquin Valley Cumulative Health Impact Project (SJV CHIP), a community-university partnership founded by environmental health and social justice organizations representing low-income and communities of color in the San Joaquin Valley and UC Davis.[198]

San Diego, California

Issues

For the border region of San Diego and Imperial Counties, trade via the San Diego seaport combines with the increasing numbers of trucks and trains that travel to and from Mexico. The Port consists of two cargo terminals: the 10th Avenue Cargo Terminal located next to the Barrio Logan neighborhood of San Diego and the National City Cargo Terminal. While there appear to be no expansion projects proposed for the port itself, there is tremendous investment in off-port transportation and highway improvement projects to facilitate the transport of Port cargo and also cross-border trade. In April 2010, the California Transportation Commission approved the allocation of $400 million of primarily Prop 1B monies to the San Diego region to fund goods movement infrastructure projects including: completion of SR 905 between the Otay Mesa border crossing and Interstate 805, a third

border crossing at East Otay Mesa with connecting SR 11, freeway access improvements at the Port of San Diego and capacity enhancements at the National City Marine Terminal, and improvements to rail corridors.

With the new investments in infrastructure expansion, communities in San Diego will be faced with increased truck traffic. New housing developments along the freeways have intensified the negative health and environmental impacts of truck traffic. For example, the new residential communities in San Ysidro along the I-905 Freeway will be directly impacted by truck traffic moving from the Otay Mesa border crossing (near a maquiladora industrial zone) and Interstate 805. The project also received $20.2 million in TIGER funds granted in February 2010.[199]

Groups

For the **Environmental Health Coalition** (EHC), organizing around ports and goods movement has focused on the local and regional impacts of the San Diego Port and the regional truck traffic that heavily affects neighborhoods like Barrio Logan. Organizing began in the 1990s when residents began taking action on the increased truck traffic in the Barrio Logan community emanating from the 7th Avenue Marine Terminal. In addition, EHC organized to curtail the use of methyl bromide in fumigation plants on port property affecting Barrio Logan or, depending on the wind direction, across the San Diego Bay to Coronado. EHC has waged community plan campaigns that have institutionalized the separation of industrial and residential uses in National City; an approach they are using to address the negative impacts of the ports in San Diego's neighborhoods. Through this combined set of strategies, EHC has successfully pressured the city to ban trucks weighing more than five tons from surface streets in Barrio Logan. EHC's successful campaign to shut down the South Bay Power Plant, a peaker plant owned by the Port of San Diego, illustrates a strategy to rid the Chula Vista neighborhood of a polluting facility and to push the Port to adopt an alternative energy strategy in order to come up with 50 megawatts of power by either reducing demand and/or creating distributive generation, e.g., cogeneration, solar, and wind energy. Toward this goal, EHC has worked with the International Brotherhood of Electrical Workers (IBEW), the International Longshore and Warehouse Workers Union (ILWU), the Sierra Club, the Apollo Alliance and the San Diego Community College District to successfully push the port to commit to $7.5 million infrastructure for shorepower to service cruise and cargo terminals at the Port. The next challenge: ensure that cargo shippers comply with the new shorepower rule.

In 2009, recognizing the influence of the Harbor Commission, EHC Executive Director, Diane Takvorian, also campaigned for a seat on the Port Commission. She was unsuccessful in being appointed, leaving the Commission strongly in control of growth and business interests.

The strategies used by EHC focus on the conditions specific to neighborhoods while linking these issues to broader trends such as the relationship between regional housing and increased truck traffic between United States and Mexico.

Statewide Coalitions and Initiatives

Within California, several statewide coalitions and initiatives focus attention on the negative health and environmental justice impacts of ports and goods movement. A statewide coalition of environmental justice and health advocates formed to influence state policy related to the siting, expansion and operations of rail yards in California, particularly in Wilmington, Commerce, the Inland Valleys, and Oakland. The coalition is led by community groups whose members live next to rail yards: **East Yard Communities for Environmental Justice, Communities for a Safe Environment, Center for Community Action and Environmental Justice (CCAEJ), and community group members of the Ditching Dirty Diesel Collaborative.** In addition, the **California Diesel Rule Work Group** works to ensure stricter rules on the use of diesel. In 2009 as part of its phase of funding for freight transportation work, The California Endowment provided resources to its goods movement grantees to engage in an 18-month **Statewide Environmental Justice, Health and Freight Movement Policy Project** to develop strategies to achieve health and environmental justice in goods movement projects and policies. The Project decided to focus on three areas: 1) movement building; 2) policy research and education; and 3) communications and media. Based on this work, several of the groups have begun new collaborations to integrate health and environmental justice considerations into decision-making related to land use planning to address climate change (SB 375) and inject health and environmental considerations into state level planning related to the Federal Transportation Reauthorization Act.

Pacific Northwest: Seattle and Tacoma, Washington

Issues

The Port of Seattle is a deep-water port located in Puget Sound, and is the 10th largest seaport in North America. The Port cargo facilities include four container terminals, 11 container berths that support two million TEUs annually.[200] Located along

Interstate 5, the port is situated within one mile of two major rail hubs operated as well as four railroad routes that link the Port to inland markets. In a push to expand the port facilities to accommodate larger vessels, more container traffic, and cruise ships, the Port of Seattle proposes to enlarge its Terminal 18 to double capacity,create a rail yard, develop on-dock rail structures, upgrade the cruise terminal, and undertake a $7.5 million project to dredge the Duwamish River.[201] According to the Port, factors influencing expansion plans include the expansion of the Panama Canal and competition with the Canadian ports of Vancouver and Prince Rupert and with the Port of Tacoma, 30 miles south[202] [203]

The Port of Tacoma is situated in a deep water harbor of Commencement Bay in Puget Sound and is linked to two transcontinental railroads, Interstate 5, Interstate 90, SR 509 and SR 167, creating a hub of warehouse and distribution activities. It is a major entry point for goods moving east; more than 70% of the Port's international imported container cargo heads east via rail to major markets, such as Detroit, Chicago, New York and Boston.[204]

At the Tacoma port, the Port's four on-dock or near-dock intermodal rail yards are served by the BNSF Railway Company and UP Railroad. Within three miles of the port are 15 transload warehouse facilities and distribution centers with over 2.8 million square feet of storage space.[205] The Port of Tacoma, like Seattle, is gearing up for competition for the large-scale post-Panamax ships by expanding its Washington United Terminal wharf and extending the berth to support two cranes to accommodate the larger ships.[206]

Groups

Seattle-based **Puget Sound SAGE** (SAGE) focuses work primarily at the Port of Seattle on issues of economic and environmental justice. Local and regional organizing currently focuses on organizing port truck drivers in the Tacoma/Seattle region, 40% of whom are immigrants from East Africa; 20% Latino and 20% white (including Russian and Eastern European immigrants).[207] An 8-week long training curriculum developed by SAGE includes segments on labor rights, power analysis, organizing, pollution and health.

SAGE, along with other labor, environmental and community organizations, including **Washington CAN**, a statewide grassroots lobbying organization, constitute the **Washington Coalition for Clean and Safe Ports**, the regional effort of the national Coalition for Clean and Safe Ports. Led by SAGE, the coalition works to win approval and implementation of a Clean Trucks Program modeled after Los Angeles' Clean Trucks Program (described more fully in the next section). The Port of Seattle approved a clean trucks program in April 2009; however, the program bans only pre-1994 trucks from the port docks (Los Angeles' plan bans pre-2004 trucks) and lacks the important employee concession requirement, leaving drivers with few resources to be able to retrofit their trucks in order to meet the new regulations. A loan program exists to support qualified truck drivers but no driver has applied or qualified for the loans.[208]

The Coalition works closely with other clean trucks campaigns to build a national strategy that has focused on passage of the Clean Ports Act, proposed federal legislation that would give greater regulatory and planning authority to local governments in order to address the negative impacts of port trucking.

SAGE and Washington CAN are also exploring collaboration with the **University of Washington School of Public Health** researchers on a health impacts study of diesel trucks and exposures to air emissions. The findings from this study will help lay the groundwork for possible organizing and activism at the Port of Tacoma.

As an entry point for goods shipped east to the Midwest and onward to the East Coast, Seattle and Tacoma play critical roles in the global freight transportation system. The state lacks the same sort of organizing focus on impacts of goods movement and ports that exists in California, although building on the national infrastructure of the Coalition for Clean and Safe Ports helps provide traction for shifting the policy debates in the region.

The Gulf Coast

Houston, Texas

Issues

Known as The Bayou City, Houston is home to ten waterways that flow through the surrounding area connecting Houston to the Gulf of Mexico for the transport of petrochemical products and grain from the Midwest. The port complex, located along the Houston Ship Channel, is made up of more than 150 private companies that operate their own private terminals and facilities that handle 85% of the total cargo moving through the port. The Port of Houston Authority (PHA) handles just 15% of the total cargo in the Ship Channel. In 2004, the PHA began construction of its Bayport Container and Cruise terminal that has catalyzed the construction of large scale distribution centers for companies such as Walmart. The terminal is slated for completion in 2022 and will have seven container berths with capacity of handling 2.3 million TEUs, tripling the Port's container handling capacity[209]

Public health studies by the University of Texas School of Public Health in Houston have documented

the health risks in the Houston area and measured the Ship Channel's pollution (which includes not only the port activities but also chemical plants and oil refineries) and its impact on children living within two miles.[210] Despite these demonstrated health impacts, the Texas Commission on Environmental Quality (TCEQ), which regulates the state's pollution permitting, has some of the most lax restrictions on air pollutants in the country. For example, the median concentration of benzene found in Houston's Manchester neighborhood was 12 times the highest acceptable levels in New Jersey, but well within Texas' limits.[211]

Groups

Organizing and advocacy groups face considerable challenges in the face of powerful oil interests who define the political economy of Houston and Texas. The combination of powerful oil industries along with the lack of state regulatory standards and enforcement for air pollution has meant that community-based and environmental justice organizations must find common ground with environmental and health advocacy organizations at the regional and state levels.[212]

In spite of the heavy influence of private industry in the Ship Channel and the lack of public participation in decision-making, several organizations have begun to increase their organizing and advocacy efforts focused on air pollution and have begun to address port issues. These groups include:

- The **TEJAS (Texas Environmental Justice Advocacy Services)**, a grassroots environmental justice organization formed in 2006 to address issues in the low-income Latino communities in Houston by offering technical assistance and information to the community and helping organize community members on issues related to air quality and community health. Organizing communities adjacent to the Houston Turning Basin (where ships turn around to head back to the Gulf), TEJAS has focused campaigns on ship idling, hazardous waste transport, air monitoring, and abandoned barges along the ship channel.
- TEJAS partners with the **CIDA (Community In-Power and Development Association)**, the **Clean Economy Coalition** in Corpus Christi, and the **Southwest Network for Environmental and Economic Justice** (SNEEJ) based in San Antonio, a regional environmental justice network of grassroots environmental justice organizations. It also works closely with **Air Alliance Houston,** an organization that formed when the Galveston-Houston Association for Smog Prevention (GHASP) and the Mothers for Clean Air organization merged to create a joint mission to reduce air pollution in the Houston area through research, education, and advocacy.
- **The University of Texas Medical Branch, Galveston**, houses the Sealy Center for Environmental Health Sciences, which has a Community Outreach and Engagement Program. The program has begun working with TEJAS and other community groups around port air emission issues. The Center's coordinator for public forums and toxics assistance has attended the Moving Forward Together conferences along with community leaders from TEJAS and other groups.

The expansion of ports and freight transportation infrastructure in the wake of Katrina and the BP oil disaster pits low-income and communities of color against tremendously powerful industry leaders without strong regulatory policies in place to protect air quality and community health. The expansion of Walmart's distribution center along the Channel Harbor, for example, reflects the scale and pressure of goods movement development in Houston and the Gulf. Breaking the status quo and forging health and environmental protective policies is necessary to redefine development and ensure health in economic development strategies in the gulf communities.

Gulfport, Mississippi

Issues

Four hundred miles due east of Houston, the community of Gulfport faces the pressure of port expansion in the form of a proposed highway project that connects Gulfport with inland distribution centers, a proposed inland port to be created by filling in 160 acres of landfill, and large scale railroad projects. The Port expects to spend $40 million to expand its west container terminal by 60 acres and is investing $570 million to increase growth to 700,000 TEUs.[213] The proposed "port connector road," SR 601, consists of a six-lane highway connecting the Port of Gulfport to inland ports along the I-10. According to residents, the highway and other port projects will negatively impact wetlands, residents' health, and historic African American communities.

Like other communities along the Gulf Coast, Gulfport must deal with the impacts of the longtime presence of industry, toxics, poverty, and disinvestment in addition to the recent devastation by Hurricane Katrina and the BP oil spill. The dominant pro-business agenda has pushed for rebuilding efforts that focus on the Port and large-scale infrastructure projects. In August 2010, Governor Haley Barbour and the Mississippi State Port Authority signed a memorandum of understanding with the Panama Canal Authority

to share information and technology and engage in joint marketing ventures in order to increase trade as the Panama Canal expands.[214] A public-private partnership between the Mississippi State Port Authority and the Kansas City Southern (KCS) Railway Company, recently received $20 million of TIGER funds to upgrade the KCS rail line in order to connect the Port of Gulfport to Chicago and Canada as well as to New Orleans and the East Coast.[215]

In 2007, the State announced it was redirecting $600 million of federal Post-Katrina housing funds to expand the port instead of meeting the needs of low-income homeowners to rebuild their homes. Civil rights and public interest attorneys led the **Mississippi Center for Justice**, with assistance from **NRDC**'s Santa Monica office, filed a suit on behalf of low-income residents in Mississippi contesting the U.S. Department of Housing and Urban Development's approval of the state's plan to spend funds on port expansion. The lawsuit was settled in November 2010 with HUD agreeing to provide funds to a state program that would reach out and help low-income homeowners rebuild.[216]

Groups

Community-based, environmental justice groups are actively involved in campaigns to address environmental racism and economic disinvestment long before Katrina and face the challenges of redirecting economic development in the process of rebuilding. Members of the **Gulfport Community Land Trust** in North Gulfport have been actively opposing the port expansion. Using land use and land trust approaches, the Land Trust seeks to protect wetlands and natural flood control areas in their historic African American community.[217]

In Turkey Creek, the community formed the **Turkey Creek Community Initiatives**, a community development organization engaged in the revitalization of coastal Mississippi's low-income, historic Turkey Creek community and watershed. They, along with the Land Trust and other grassroots organizations and environmental groups face significant challenges as they take on pro-business and pro-development interests in order to inject health, community, environmental, and cultural considerations into policies related to environmental, housing, transportation, and local and state economic development.

The **Mississippi Center for Justice** has represented a wide range of community, environmental and social justice organizations and has facilitated public interest legal assistance to address the range of civil rights, housing, environmental, and economic justice issues in the Gulf communities.

The **Steps Coalition**, a group of community leaders and social justice advocates formed in 2006 to identify ways to strategize about post-Katrina development in the Gulf Coast. Coalition members have launched the **Steps Coalition Partners for Safe & Healthy Port Campaign** that focuses on educating and organizing residents in the many neighborhoods that will be affected by port expansion and freight transportation projects. These include port fenceline communities, neighborhoods located near highway and rail construction projects that will serve the expanded port, and residents near an inland storage yard where containers would be evacuated for hurricanes.[218]

The Midwest

Detroit, Michigan

Issues

The Port of Detroit, located across the Detroit River from Windsor Canada, encompasses 35 acres of dock and shipping facilities. Three interstate highways, I75, I94, and I96, as well as six railroad companies service the Port. The port, along with bridges, highways, and rail yards facilitate the flow of goods between North America and Canada. These freight transport facilities contribute to the Windsor-Detroit corridor's ranking as the busiest commercial land border crossing in North America.[219] Detroit, Wayne County, and the State of Michigan eye the port and freight transport to bring jobs and development to the region.

Among the proposed projects are:

- The **Detroit Intermodal Freight Terminal (DIFT)** is a 595-acre expansion of the 300-acre Livernois Junction Yard intermodal freight terminals in Southwest Detroit. The project is a public private partnership between the Michigan Department of Transportation and the following railroad companies: Norfolk Southern, Canadian Pacific, CSX, and Canadian National. The Federal Highway Administration has issued the final environmental clearance for the project.

- The **Detroit River International Crossing (DRIC), now referred to as the New International Trade Crossing (NITC),** will connect Detroit/I-75 to Windsor, Ontario, Canada/Highway 401. Bridge advocates note that the project would help facilitate trade between the United States and Canada, now about $44 billion annually.[220] The project administered by a partnership between the U.S. Federal Highway Administration, Transport Canada, the Ontario Ministry of Transportation and the Michigan Department of Transportation, includes a customs plaza, and will increase

freight transport, directly impacting the Delray neighborhood in Southwest Detroit where four railroads operate. The project, a direct competitor to an existing privately owned Ambassador Bridge, was initially stalled because of influential pressure by Ambassador Bridge owners. However, in early 2011 Detroit Governor Snyder and U.S. Transportation Secretary Ray LaHood announced support for the NITC giving the project new momentum.

- The **Detroit River Rail Tunnel (DRTP)** would connect Detroit and Windsor underground and allow double stack cargo trains for freight transport between the two countries.

In addition to the proposed new projects, residents in Southwest Detroit face the challenge of already existing polluting facilities such as a regional wastewater treatment plant, the largest rail yard in Michigan, three major highways and heavy industry.[221]

Groups

These various projects have a direct impact on Southwest Detroit where community-based and environmental justice organizations develop strategies to influence these projects. These include **Detroiters Working for Environmental Justice (DWEJ),** an environmental justice organization, and **Detroit Hispanic Development Corporation (DHDC).** These two community-based organizations address ports and freight transportation concerns along with other community-based issues. DWEJ formed in the early 1990s in the swell of the emerging environmental justice movement to focus on pollution, health, and jobs. The DHDC organizes around environmental issues but also provides direct services and advocacy on other issues such as immigration, education, and juvenile justice issues that face the Latino immigrant community. Both groups rely on organizing and leadership development strategies to strengthen relationships within the community in order to build grassroots influence. DWEJ and DHDC are partners with the **University of Michigan School of Public Health** to produce community-based research and develop educational tools for community residents to understand the health impacts of proposed projects and policies. Through a long-term university-community partnership called Community Action Against Asthma (funded through NIEHS), DWEJ and other community organizations focus attention on the development of the DIFT.[222]

The **Southwest Detroit Community Benefits Coalition**, a community benefits agreement coalition that is supported by the Los Angeles Alliance for a New Economy and the Detroit office of LISC (Local Initiative Support Corporation), has begun organizing around the development of the NITC. Formation of the coalition brings together labor and community organizations for project-specific campaigns and builds long-term relationships between these groups. A related citywide coalition, **Doing Development Differently in Detroit (D4)**, has also helped convene and strengthen relationships labor, community, environmental justice, environmental, community, and other social justice interests in Detroit.[223] See also discussion of community benefits agreements below.

The **East Michigan Environmental Action Council (EMEAC)** is a Detroit-based environmental organization that has focused its air quality work on issues of diesel pollution. Actively engaged in state level coalitions and the national **Clean Air Task Force**, EMEAC also focuses on local level diesel education in Southwest Detroit and serves as a community representative on the Southwest Detroit Environmental Vision, a business and community collaboration closely associated with the Port Authority. Within EMEAC, an emerging environmental justice focus has begun to link air pollution work to ports and freight transportation issues.[224]

Organizing in Detroit also means organizing in an international context. Canada's push to build its trade corridor capacity illustrates how local projects in Detroit are inherently international ones. The international context also illustrates the limited role the City of Detroit has in local development planning. For example, the City of Detroit's Department of Environment, faces budgetary as well as internal political challenges leaving much of the power to direct local development in the hands of Canada and the private sector. Established and emerging organizing in Detroit, however, points to the ways that global framing and expanded collaborations address goods movement and incorporate health considerations into development debates.

Chicago, Illinois

Issues

Chicago is the only place in the nation where six of the seven major North American railroads (BNSF, UP, Canadian National, Canadian Pacific, CSX, and Norfolk Southern) converge, making the entire region (Will, Cook and Page counties) a "switching yard for America's eastern and western railroads."[225] Today one-third of all freight traffic in the nation passes through Chicago[226] and is expected to double over the next 20 years.[227] The region's newest intermodal, UP's Joliet Intermodal Terminal, is a 785-acre facility developed by CenterPoint Properties, a company that also created the CenterPoint Intermodal Center in Elwood, a $1 billion, 2,500-acre logistics park billed as "one of the largest private developments ever undertaken in the United States."[228]

The Chicago area is home to at least 25 intermodal facilities - making it also the largest concentration of intermodal rail yards in the United States.[229] [230] Chicago also has the second largest amount of warehouse space in the western hemisphere, after California's Inland Valleys.[231] Its more than 300 warehouses employ about 150,000 warehouse workers, a number likely to continue to grow.[232] Much of this warehouse activity is located in Will County where annual freight transportation employment grew on average 8.2% between 2000 and 2009.[233] Mirroring the warehouse growth in the Inland Valleys in California, Will County is one of the fastest growing counties in the United States due to the logistics industry's presence.[234]

Many of the region's older rail yards are located adjacent to low-income communities of color in Chicago. These include neighborhoods such as Back of the Yards, Brighton Park, Roseland, Bedford Park, and Englewood.[235] Residents who live near these yards not only suffer from higher asthma rates, but from higher rates of respiratory disease, cardiac disease, and premature death as well.[236] The average cancer risk of residents living within ½ mile of the 15 largest Chicago-area intermodal facilities has been estimated at more than 10 times that of people living just four miles away.[237] According to a report by the Chicago Reporter, about 57,000 people, the majority of them people of color, live within one-half mile of these 15 rail yards alone.[238]

Groups

Organizations working in the Chicago region to address the impacts of truck and rail pollution include:

- **Warehouse Workers for Justice** (WWJ) is an independent workers' center founded by the United Electrical Workers (UE) that was established in 2009 following a successful plant occupation at Chicago's Republic Windows and Doors. The new WWJ was founded realizing that the union's traditional base was shrinking as electrical manufacturing jobs continued to be shipped overseas. UE wanted to try to reach out to logistics industry workers, where it saw jobs that cannot be outsourced and saw warehouse workers with the "potential to build power."[239] WWJ provides a venue to educate warehouse workers on their rights and fight for policy change to secure greater workplace protections.[240] WWJ has established a partnership with **Warehouse Workers United,** a Change to Win project. WWJ worked with the **Center for Urban Economic Development (CUED) at University of Illinois at Chicago** (UIC) to conduct a survey of warehouse workers in Will County and produced a detailed profile of the new Will County logistics industry jobs and the impacts of the expanding warehouse industry has on workers.[241] Through the Warehouse Workers United campaign, WWJ has begun collaborating with CCAEJ in California to organize warehouse workers, many of whom are temporary workers and residents of environmental justice communities in the California's Inland Valleys and Chicago.[242]

- The **Will County Residents for Responsible Intermodal Development** is a coalition in Will County that formed to oppose the proposed development of the CenterPoint Intermodal facility in Crete Village.[243]

- The **Greater Englewood Community and Family Task Force** formed to stop the development of the CSX Englewood intermodal facility, located in the South Side African-American community of Englewood. Organized opposition by the Task Force resulted in a community benefits agreement in which the railroad pays the city of Englewood $300,000/year, allocated to residents "for home repairs and other projects."[244] A class action lawsuit was filed against CSX in 2005 where homeowners sought to limit the yard's operations to daytime and early-evening hours. Although the rail company acknowledged the negative impact on the neighborhood, their lawyers successfully argued that the company was protected by a federal law that leaves regulation of rail operations to the federal government.

- The **Respiratory Health Association of Metropolitan Chicago** (RHAMC) performs research, advocacy, and outreach to fight asthma, lung cancer, chronic obstructive pulmonary disease (COPD), emphysema and other respiratory illness and disease.[245] As part of RHAMC's work combating diesel pollution, the organization has secured funding for and implemented - often in tandem with Illinois EPA - "more than 100 clean diesel projects throughout the state." US EPA recognized that work with a leadership award earlier this year.[246] Other than its successful work lobbying for idling limits, much of its focus on diesel pollution involves coal power plants, public transportation and construction, and private passenger rail - rather than freight rail and trucks.[247]

- **Citizen Action/Illinois** is the state's largest public interest group. Tackling diesel pollution is among its program areas.[248] [249] Along with RHAMC, Citizen Action/Illinois actively advocates for the use of ultra-low sulfur

diesel (ULSD), particulate filters or diesel oxidation catalysts (DOCs) in trucks, buses, and construction equipment, anti-idling, automatic shut-off technologies in locomotives, and the replacement of old switcher engines with Gen-Set or hybrid diesel-electric models.[250] [251]

- Citizens Action/Illinois and RHMAC founded the **Illinois Campaign to Clean Up Diesel Pollution** in 2007, a coalition comprised of over 80 health, environmental, labor, faith and community organizations.[252] It is also part of the National Partnership to Reduce Diesel Pollution, a nationwide coalition of organizations "committed to reducing direct diesel fine particulate matter emissions by 40% by 2012, 55% by 2015, and 70% by 2020."[253] The Illinois Campaign applies pressure to elected representatives to secure state and federal dollars for retrofitting and replacing diesel vehicles and equipment, as well as encourages government and industry to apply for these funds.

Increasingly, organizations in Chicago are starting to consider how to make the links between issues and organizing in the urban core neighborhoods of Chicago with outer ring neighborhoods facing new freight transportation expansion projects. The intensification and expansion of freight transportation in the Chicago region illustrates the need for regional scale analysis and organizing and the challenges facing community and workplace-based organizers and health advocates.

Kansas City, Kansas

Issues

Kansas City has served as one of the nation's primary logistics hubs since the construction of the transcontinental railroad in the 1860s. The City's Argentine Yard, a 780-acre freight car yard is the second largest rail yard in the world, representing one of three terminal points for BNSF to service diesel locomotives. More than 1,800 train cars run in and out of the yard every day.[254] Several years ago BNSF announced its plans to build an intermodal logistics park to include a rail yard, a large trucking distribution center and warehousing in the area of Gardner, KS, about 30 miles southwest of the Argentine yard. The railroad wanted the City of Gardner to annex land on which to build the rail yard, but significant controversy arose over the plans, involving residents and political leaders. As a result, the small city of Edgerton decided to annex the 1,100-acre site, which is located in Johnson County, KS, less than a mile from two schools and homes in Edgerton. The 24-hour a day facility is expected to generate more than 5,000 new truck trips per day and an estimated 17,080 trips by 2030.[255] Residents of Edgerton and Johnson County are also concerned about potential water contamination because of the proposed intermodal facility's proximity to the Hillsdale Watershed that provides drinking water for the surrounding areas.

Groups

Because of concerns about the intermodal development and proposals to build large distribution centers in the city of Gardner, the **Johnson County Intermodal Coalition** was formed and included homeowner Eric Kirkendall, whose house was threatened by the prospect of development. He and his wife lived in an old farmhouse in Gardner, where they had hoped to retire. However, the developers wanted to build 12-acre warehouses to support the intermodal rail yard on three sides of his 4-acre homestead. The Intermodal Coalition, along with the **Hillsdale Environmental Loss Prevention (HELP)** organization, stepped up its efforts to protect the community, raising particular concerns about whether possible pollution from the proposed facility could harm the Hillsdale watershed. HELP filed a lawsuit, which was joined by NRDC in Santa Monica, CA, against the Army Corps of Engineers focusing on the project's water quality and air quality impacts. The suit asked the Kansas District Court to freeze a permit issued by the Army Corps of Engineers and require a full Environmental Impact Statement (rather than the simpler Environmental Assessment that had been conducted) before allowing BNSF to move forward with the project.

Several Johnson County officials, as well as Eric Kirkendall, attended the 2007 Moving Forward Together conference in Los Angeles, where they met USC faculty members, environmental justice activists, and lawyers from NRDC. When a member of the City Council in Gardner, along with the Coalition and HELP, decided to sponsor a community forum on the rail yard and distribution centers, they invited three representatives from Los Angeles (USC, East Yard Communities for Environmental Justice, and NRDC) to speak at the meeting, along with representatives of HELP, the Sierra Club and the American Cancer Society. More than 100 residents attended, and significant local press was generated. Subsequently, Kirkendall was a featured speaker at the 2010 Moving Forward Together conference. He told attendees that his fight to stop the distribution centers from construction was unsuccessful. His home was demolished and he and his wife are now residents of Lawrence, KS. The lawsuit against the intermodal rail yard has not yet been decided in court.

As is the case in other communities with few organizations focused on ports and freight transportation, local organizers and campaigns in Kansas City drew upon the capacity and expertise of

other regional campaigns, resources and experiences in order to build stronger organizational and movement capacity to shift the terms of the goods movement debates. In the Kansas City region, the primacy of rail and goods movement industries and employment has begun to bring together interests focused on water quality and community quality of life. Connecting these new emerging constituencies to a broader network of similar communities and interests will be important in building a stronger base of organizing in Kansas so that residents have a stronger voice in decisions that are being made about their own community's future.

Northeast/Central Atlantic Coast

New York/New Jersey

Issues

The Ports of New York and New Jersey make up the 3rd largest port complex in the nation and handle the highest volume of shipping containers on the East Coast. The Port Authority of New York and New Jersey (PANYNJ) runs the Port Newark and the Elizabeth Marine Terminal (Port Elizabeth), adjacent ports that include six container terminals, four auto processing terminals, and cargo berths. The PANYNJ is also responsible for other port terminals: APM Terminal, Maher Terminal, Global Terminal, New York Container Terminal, Red Hook Container Terminal, Brooklyn Port Authority Marine Terminals, South Brooklyn Marine Terminal, and the Port Jersey-Port Authority Marine Terminal.[256] In addition, the PANYNJ governs the regional airport system, the PATH rail transit system, six tunnels and bridges between NY and NY, the Port Authority Bus Terminal in Manhattan, and the World Trade Center.

In 2008, the port handled over 377,000 intermodal containers, more than double its volume in 2000.[257] With hopes of attracting trade from the expansion of the Panama Canal, the PANYNJ has invested in a number of port expansion and improvement projects. $3 billion has been committed to terminal expansions and improvements alone.[258] These projects include costly alterations to the Bayonne Bridge in order to accommodate Panamax ships, the expansion of the Port of Newark Container Terminal and the construction of a permanent rail facility that could increase capacity to 1.2 million boxes, deepening two of its berths, and the creation of a fourth container berth at the New York Container Terminal.[259] Once complete the Corbin Street Intermodal Support Yard, part of the $600 million Port Authority investment in the ExpressRail, will be able to move 1.5 million cargo containers annually.[260] Several other rail expansion projects have been completed initial phases and will facilitate distribution of goods from the port to inland Midwest distribution hubs. In October 2010, CSX Corporation opened a double-stack rail line at the Port of New Jersey/New York, a $20 million project to raise clearances on a five-mile track linking port terminals with CSX's main route between New York and the Midwest. The initial phase of the Liberty Corridor line features the enlargement of two tunnels, including a 4,400-foot tunnel under Jersey City.

The New Jersey neighborhoods of Ironbound and South Ward are surrounded by these existing and expanding port, rail, and highway corridors. In December 2008, activists and residents stood at six intersections, four in the Ironbound neighborhood and two in the South Ward, counting port trucks passing through their community. Although they were aware of the large truck volume in their community, in just two hours they counted over 630 trucks, or 5 trucks per minute, passing directly through their neighborhoods.[261] In fact, approximately 7,000 diesel trucks make an estimated 10,000 trips to and from the ports every day.[262]

Using scientific testing methodology of the U.S. EPA, the Clean Air Task Force conducted research on diesel exposure in the areas around the ports and found cancer risks to be 486 times greater than U.S. EPA's acceptable cancer level of one in a million. [263] [264]

Groups

Several well established organizing and coalition groups are involved in organizing around ports and goods movement. These include:

- The **New Jersey Environmental Justice Alliance**, founded in 2003, is am umbrella organization of more than 40 environmental justice organizations in Jersey working on issues related to particulate matter air pollution, climate change, cumulative risk and impacts, and the siting of schools on contaminated land. The NJEJA works with community-based environmental justice organizations as well as advocacy and research institutions, to develop environmental justice legal strategies and increase the capacity of the environmental justice community to address these and other issues. A recent conference featured a founding member of NJEJA who raised the imperative of "environmental health justice."[265]

- **New Jersey Environmental Federation** (NJEF), a statewide organization with over 100,000 members and almost 100 affiliated groups, has been working since 2003 to combat the health and environmental impacts of diesel emissions in vulnerable communities. Working with the New Jersey Environmental Justice

Alliance, NJEF has won legislation to publicly fund the retrofitting of diesel-powered vehicles and a statewide idling law. NJEF has a long history of working with labor, such as chemical workers and refinery workers. In 2006, NJEF released a report detailing the port-related health impacts from diesel emissions in the Ironbound, a port-adjacent community in Newark with an extreme legacy of environmental injustice and pollution.[266]

- **Ironbound Community Corporation** (ICC), a social service organization that worked with NJEF to produce the 2006 report, had been fighting for years against the harmful impacts of toxic emissions from a waste plant, an international airport, a Superfund site, chemical plants, interstate freeways, more than 100 brownfields, and the Ports of Newark and Elizabeth. Prior to the ports campaign in New Jersey, the ICC was fighting against the construction of a proposed incinerator in their community, which was already wrought with diesel hotspots and other polluting facilities. In March 2010, ICC received a U.S. EPA CARE grant to survey the neighborhood and develop a cumulative analysis of toxics and health effects in a community with high poverty rates and where 75% of residents speak languages other than English, primarily Portuguese or Spanish.[267] 55% of those over 18 do not have a high school diploma. Census tracts in the neighborhood range from 25% to 55% of households below the poverty level.

- The **Coalition for Healthy Ports** is a bi-state coalition of community-based organizations, labor, faith based and environmental organizations are involved in the campaign for clean trucks. The Coalition for Healthy Ports is the East Coast campaign of the national Coalition for Clean and Safe Ports. The coalition is staffed by the **Garden State Alliance for a New Economy** (GANE), the region's equivalent to LAANE (Los Angeles), SAGE (Seattle) and EBASE (Oakland). Members include the organizations discussed above as well as **GreenFaith,** an interfaith organization of religious leaders that have engaged in environmental justice campaigns. Using the Los Angeles model for a Clean Trucks Program, the Coalition for Healthy Ports pushed for a similar clean trucks program that would meet community needs and address emissions through an employee concessions model. See full discussion on the Clean Trucks Programs below. The Coalition has produced a series of important reports that document the health, labor and community issues faced by port truck drivers and community residents who live adjacent to the ports and freight movement corridors.[268]

Collaboration with academic researchers has produced important community-based research and industrial analyses. The director of the **Center for the Urban Environment at the John S. Watson Institute for Public Policy at Thomas Edison State College** has been conducting surveys to determine the level of exposure of communities of color to the diesel emissions from port commerce [269] and has helped Coalition members conduct truck counts near the Newark port. The Coalition for Healthy Ports also draws upon research by **Rutgers University's School of Management and Labor Relations** on the low wages and poor working conditions of port truck drivers in the trucking industry.[270]

Organizing and activism around the New York/ New Jersey ports and freight transportation reflects a robust labor, community, and environmental sector that recognizes the power and negative impacts of freight transportation at the local, state, and national level. Organizing and advocacy strategies by these groups illustrate the ability to build local level work into effective state level influence able to impact decision-making by the state governed Port Authority of New York/New Jersey.

Baltimore, Maryland

Issues

The Port of Baltimore, situated on Chesapeake Bay, is a major auto-handling port and the East Coast port closest to the Midwest cities. Although only 21st among U.S. ports, the city of Baltimore has become focused on freight industry expansion related to the Panama Canal. In 2010, Baltimore signed a deal with Ports America, the nation's largest terminal operator to take over expansion and operation of the Seagirt Container Terminal, the state's primary container terminal. The $105 million expansion includes building a fourth berth, dredging the harbor, and purchasing four super-post-Panamax cranes.[271]

In order to accommodate the doublestack container rail transport which are unable to travel through the existing Howard Street Tunnel, CSX Corporation is seeking a new location for a proposed intermodal facility. Several possible 100-acre rail adjacent sites are in consideration, including a location in Elkridge adjacent to a proposed new school site.[272]

The new intermodal project is but one project of the proposed CSX rail expansion strategy to expand rail transport and distribution along the East coast and to Midwest markets.[273] As noted earlier, CSX

has entered into a public private partnership to develop the National Gateway Corridor, a north-south rail expansion effort between North Carolina and Baltimore, paralleling I-95. The project proposes to expand three existing rail corridors that run through Maryland, Virginia, North Carolina, Pennsylvania, Ohio and West Virginia. The project received $98 million in TIGER funds in February 2010.[274]

Groups

While there are no visible community-based organizations or campaigns within the City of Baltimore organized explicitly around ports commerce or goods movement, there are a number of environmental justice organizations in Baltimore concerned and engaged in health and environmental justice issues, particularly in the East Baltimore community. Many of these groups are involved with the **Environmental Justice Partnership (EJP)**, a community-university partnership between the **Johns Hopkins Bloomberg School of Public Health (JHSPH)**, the Maryland Institute College of Art (MICA) and community-based organizations in East Baltimore.[275] Formed in 2003 with start up monies from the National Institute of Environmental Health Sciences (NIEHS), the EJP connects academics with East Baltimore residents to build capacity for environmental justice groups to engage in discussions with governmental agencies, academic institutions, and the private sector.[276] The EJP's accomplishments include winning paid relocation of residents in the Wagner Point neighborhood, which heavy industry has made unlivable, and the dissemination of newsletters and educational materials produced with the help of graphic design students at MICA. Pat Tracey of Johns Hopkins states that the EJP had mostly been focusing on "the impacts of urban demolition, the number of industries in low-income neighborhoods, and bus depots situated so close to residents that it feels as if they are in their backyards."[277] To date, there has been little community demand to investigate port-related community issues.

Port industry stakeholders on the other hand are organized through the Baltimore Port Alliance, an organization of about 150 public and private maritime business interests.[278] The industry stakeholder group has its own environmental committee that received $3.5 million in stimulus funding in 2009 from the U.S. EPA to help industry retrofit its diesel-powered locomotives, short-haul trucks, and cargo-handling equipment at the port with clean-diesel technology.[279]

Although the Port's Green Port Initiatives (including electrification of cranes, reducing idling time on-dock, and installing dust emissions systems) have not yet intersected with concerns and needs of the environmental justice community in Baltimore, the active set of environmental justice organizations working with public health researchers and scientists, suggests a potential alignment and community-based capacity to develop health and justice strategies in port and freight transportation policy and decision-making.

Norfolk/Hampton Roads and Inland Virginia

Issues

The Port of Hampton Roads is the oldest continuously operating port system in the United States.[280] Many consider the Port of Hampton Roads the economic engine of the region and indeed, all of the area's biggest industries are port-related: shipbuilding, ship repair, naval installations, cargo transfer and storage, and manufacturing related to the processing of imports and exports.[281] Presently, the Port consists of four terminals operated by the Virginia Port Authority (VPA): Norfolk International Terminals (NIT), Portsmouth Marine Terminal (PMT), Newport News Marine Terminal (NNMT), and APM Terminal Virginia (APMT). The APM, or Maersk Terminal, was the United States's first privately developed container terminal and is among the most technologically advanced and automated cargo-handling facilities in the world.[282] It also used to be in competition with the Port of Hampton Roads until the Port acquired it for a 20-year $1 billion lease in July 2010.[283] Additionally, the VPA owns and operates an inland intermodal facility, the Virginia Inland Port (VIP) in Front Royal, 220 miles inland and connected to the Port by rail.[284]

While all four terminals have on-dock rail, the majority (about 2/3) of the freight leaving the port is carried on trucks and just 30% via rail.[285] This truck-to-rail ratio - as well as the Port of Hampton Roads' attractiveness as a port of call in general - likely has something to do with the fact that a full two-thirds of the U.S. population can be said to be located "within a 750-mile, 2-day truck drive" of the Port,[286] making trucking relatively easy.

Notably, Hampton Roads is a deepwater port and one of the world's largest natural harbors. This has meant that that unlike all other Eastern seaports except for Baltimore, the Port of Hampton Roads requires no additional dredging, which puts it in a prime position to begin accepting - without delay and absent many costly infrastructure improvements - the expected arrival of Post-Panamax ships in 2014. In fact, Norfolk, the largest terminal, already boasts the world's largest "Suez-class" container cranes equipped to handle ships larger than any yet built.[287] A fifth terminal, Craney Island is scheduled to open in 2017 to accommodate the doubling and tripling of container traffic expected by 2015 and 2020, respectively.[288] Goods movement infrastructure projects such as the rail-based Heartland Corridor, capitalize upon the expanded infrastructure and the linking of the Port to

Midwest intermodal facilities and markets. In March 2010, the Virginia Department of Environmental Quality (VDEQ) granted the Port of Virginia and the U.S. Army Corps of Engineers permit approval to start construction of the Craney Island Eastward Expansion.

In terms of air quality, the air shed that includes the Port of Hampton Roads had been in violation of the federal '8-hour ozone standard' in the past (from 2004 to at least 2006), but appears to be in compliance at present.[289] In response to the Port of Hampton Road's past noncompliance with National Ambient Air Quality Standards (NAAQS), the VPA has taken several steps to address air pollution.[290] This includes the development and implementation of its "Green Operator" Voluntary Diesel Truck Retrofit Program, which encourages trucking companies to voluntarily retrofit and replace their aging trucks. The port-initiated effort, like many other clean trucks programs at other ports however, lacks the employee concession element necessary to address the negative impact of clean trucks programs on port drivers.

In 2005, the Virginia Port Authority initiated its Comprehensive Air Emissions Inventory and Integrated Planning program as a way to track the emissions produced by all port operations (i.e., from ships, trucks, trains, and equipment).[291] While the monitoring plan is in place, policies that other ports utilize to cut emissions such as cold ironing capability, speed reduction regulations, emissions regulations for trucks or rail, and any significant alternative fuel programs have not yet been implemented at VPA. Most recently, the VPA issued its Craney Island Eastward Expansion Environmental Mitigation Plan, which focuses on minimizing the environmental impacts of the expansion.

Groups

There does not appear to be any engaged health, environmental justice, or community-based group actively working on freight transport and port-related issues in this region of the country. By contrast, in Southwest Virginia, Norfolk Southern and business leaders are pitted against farmers and community members over a proposed intermodal facility in Elliston (Montgomery County) that would convert 65 acres of pristine farmland to a Norfolk Southern rail yard for transferring freight between trucks and trains. The proposed rail expansion will require state funding to facilitate increased freight on the new Heartland Corridor, which provides shorter, double-stacked service between the Port of Hampton Roads and Midwest markets.[292] Montgomery County residents, along with the Montgomery County Board of Supervisors, are concerned about the impacts the facility would have on their rural communities - and specifically about the effects of unprecedented increases in traffic and pollution and the construction of new roads and warehousing. A number of Montgomery County residents formed **Citizens for the Preservation of Our Country** and have been pursuing legal means to halt construction.[293] The residents group faces strong opposition in the form of the Roanoke Regional Chamber of Commerce and the Virginia Department of Rail and Public Transit (DRPT) which cites the Virginia Department of Transportation (VDOT)'s assessment that "truck traffic generated by the intermodal facility should present little to no impact."[294] The Montgomery County Board of Supervisors has also come out in opposition to the proposed intermodal, anticipating that the environmental and health impacts from the large increase in truck traffic through their communities would far outweigh any economic benefits.[295] The body passed four resolutions to oppose the proposed facility and filed a lawsuit to block its construction. The resolution is now before the Virginia Supreme Court.

Southeast

Charleston, South Carolina

Issues

The Port of Charleston ranks 13th of North American ports and handles approximately 8,400-TEU vessels annually at its five marine terminals.[296] It is located in the Charleston Harbor and with access to interstate highways 526 and 26 and two state highways. Norfolk Southern and CSX rail lines link the port to cities in the South, Mid-West, and Mid-Atlantic.

Like other East Coast ports, the Port of Charleston is positioning itself to attract trade from the widening of the Panama Canal. The Port has developed a 10-year, $1.3-billion capital plan in order to increase container capacity in the port 50% by 2018.[297] This includes the $21.7 million expansion of the Columbus Street Terminal that added more than 70 acres of storage yard and additional rail infrastructure to the auto terminal.[298]

Several other port projects as well as goods movement infrastructure projects are also in the works. The South Carolina State Port Authority (SPA), the state agency responsible for the Port of Charleston and Port of Georgetown, has assumed authority over the 280-acre former Navy base in North Charleston, and is in the process of developing a new three-berth, 280-acre container terminal that is expected to increase port capacity by 1.4 million TEUs.[299] The South Carolina SPA had also developed a cooperative agreement with the Georgia Ports Authority (GPA) to develop a new marine terminal on the Savannah River

in Jasper County, SC, and had therefore supported the Savannah Harbor dredging plan. However, the SPA has raised some objections to the GPA dredging plan and is threatening to withdraw from the project.[300]

Port communities in the densely populated downtown area of Charleston and North Charleston face harmful health and environmental impacts of existing port commerce.[301] Industrial and port-related activities, particularly that of ship traffic, port trucks, trains, and other diesel-powered vehicles, contaminate the surrounding neighborhoods with toxic chemical emissions.[302] Specifically, in 2005, operations at the Port of Charleston, according to its own emissions inventory, released 175 tons of $PM_{2.5}$ (particle pollution).[303] Although the Port said that this was less than 5% of Charleston's particulate pollution, the Coastal Conservation League, a local environmental advocacy organization, calculated that it would make up about 70% of Charleston's permitted industrial emissions, most of which remains in downtown communities.[304]

A study commissioned by the **Coastal Conservation League** concluded that the cost of port pollution from the port expansion in Charleston could be up to $81 million every year.[305] However, Nancy Vinson, the League's program director for Water, Air, and Public Health, contends this is actually a conservative estimate, so the real costs will be even higher if the port does not take measures to reduce diesel emissions as it expands.[306] A lawsuit filed by the CCL charged that SPA approved the port expansion before receiving the results of at least three environmental studies and illegally issued environmental permits for the port expansion. As part of the recent settlement in August 2010, the SPA acknowledged the health and environmental impacts of port-related diesel emissions and vowed to clean up port activities. In the settlement, the SPA agreed to install new air pollution monitors, switch to cleaner port trucks, and begin relying on post-1994 trucks that are less polluting. Although the replaced trucks will only make up about 15% of port traffic, emissions from older engines currently comprise one third of air pollution from port commerce.[307]

Prior to this settlement, there were no local policies regulating the emissions of port-related vehicles. Since the SPA is a state-owned agency where the governor appoints SPA board members, it is difficult to establish policies to mitigate port-related diesel emissions at the local level because the issue is part of the statewide agenda and involves a more robust and complex decision-making system.[308] Local government, therefore, has no power to govern the ports and cannot monitor emissions or mandate emissions reducing measures.

Groups

The **South Carolina Coastal Conservation League** (CCL), a nonprofit environmental organization working to protect South Carolina's natural resources and communities along the coast, originally got involved in port issues about a decade ago when the South Carolina Port Authority was planning on expanding the port to a peninsula called Daniel's Island adjacent to Charleston. The Port Authority was planning to build a new 12-berth port terminal that would have generated as much port traffic as the Ports of New York and New Jersey, which is the second largest port facility in the country.[309] In response to the threat of environmental degradation along the South Carolina coast, the Conservation League led a campaign to stop the planned port expansion, and succeeded. The Port Authority then proposed a smaller expansion on the old naval base north of downtown Charleston, which is currently underway. The naval base is, however, close to a number of African-American communities.

The Coastal Conservation League originally agreed with moving the port expansion away from Daniel Island to the old naval base, because it had a rail infrastructure to move containers. However, the SPA decided to use trucks instead of rail, which would bring 10,000 additional diesel-powered vehicles per day to I-26, the already burdened main line into Charleston.[310] In response, the Conservation League formally decided to take action against the proposed port expansion and sought out community partners.

Since then, the CCL and the **New Rosemont Homeowners Association,** representing the community closest to the proposed port expansion, have formed a strong environmental-community partnership fighting for the port to take measures to reduce diesel emissions. The CCL has also developed an environmental-medical partnership with the Charleston County Medical Society to work together to highlight the health effects the port expansion will have on port-adjacent communities, as well as the associated health costs. In March 2010, the CCL and the New Rosemont Homeowners Association hosted a community meeting, with panels including experts from Charleston as well as from California, to educate local community members and elected officials about the devastating impacts of port commerce. As a result, a new coalition, CLEAN, was formed to address environmental health and environmental justice issues in their port communities.

The Lowcountry Alliance for Model Communities (LAMC) is a non-profit community-based organization in North Charleston, SC that is organized to address environmental justice and health issues in seven African-American neighborhoods near the Port of Charleston. It formed in 2005 to unite the voices of

seven economically disadvantaged neighborhoods in North Charleston and became involved in port issues when they learned that the environmental review of the proposed port expansion had completely ignored the impacts that the project would have on their port-adjacent communities, namely: diesel truck traffic, Superfund sites, an incinerator, brownfields, heavily-trafficked roads, and chemical plants.[311] The neighborhoods experience high levels of poverty, segregation, crime, and drug use.[312]

Starting in 2005, LAMC, the SPA, and the City of North Charleston began to negotiate a community Mitigation Plan Agreement (MPA) to address LAMC member concerns. In the MPA, the SPA granted the communities $4 million to address important community needs such as funding for local scholarship programs, affordable housing, environmental monitoring, healthcare and fitness initiatives, and other programs.[313] The SPA also made a separate, voluntary, agreement with the South Carolina Department of Health and Environmental Control, pledging to address pollution and health issues associated with the Port and other local industries.[314] The SPA reports that it is using cleaner engines and including emissions-reducing guidelines in its construction bid documents, and that it has switched all of its terminal equipment to ultra-low sulfur diesel (ULSD)[315]

LAMC has also formed a community-academic partnership with the Department **of Epidemiology and Biostatistics at the University of South Carolina.** With a four-year grant from the NIEHS, the university and LAMC are conducting an environmental health study in the City of North Charleston to determine if there is a link between multiple sources of pollution and the health of the residents living in the seven neighborhoods that comprise LAMC. The partnership utilizes a collaborative problem-solving model to address cumulative impacts. Working with LAMC, researchers are using "training and community outreach techniques to educate residents about local environmental health risks and education to teach ways to reduce their exposure."[316]

Important community, health, and environmental organizing focused on the Port and freight transportation has taken place in Charleston. Even with important gains for the community, the scope and scale of proposed port expansion points to gaps between health, environmental justice, labor, and community development issues and health protective policies. Specifically, since South Carolina is a right-to-work state, organized labor (other than the International Longshore Association) does not have as strong a presence as in some other states and therefore does not play a major role in the debate surrounding the port expansion in Charleston. Finding strategies that integrate health and worker considerations across the range of organizing, advocacy, and policy solutions will be critical as the port moves forward with its plans to expand.

Savannah, Georgia

Issues

The Port of Savannah, the 4th largest in the United States by container volume, is a major gateway for imports from South and Central America and the Caribbean and for exports to Asian countries. As with many of the Eastern and Southern ports, the widening of the Panama Canal has become a major focus for expansion. These include plans to deepen the Savannah harbor, by up to six feet (for a depth of 48 feet) to accommodate post-Panamax and Super post-Panamax cargo ships expected to start using the Panama Canal. The dredging project is estimated to cost $588 million and to be completed by 2015.[317]

Federal funding has been a major contributor to Port expansion. $21.7 million, for example, was approved by Congress to fund the deepening and maintenance of the Savannah and Brunswick harbors, run by the Georgia Ports Authority (GPA).[318] "The approval of $1.49 million in construction funds is another solid vote of confidence by the Congress in support of Georgia's effort to prepare the Savannah Harbor channel for the new generation of larger ships that represents the future of ocean commerce," said Steve Green, GPA Chairman of the Board.

The Draft Environmental Impact Statement (DEIS) for deepening the Savannah River was released on November 16, 2010.[319] The DEIS minimizes air pollution issues that will result from port expansion. Unlike the San Pedro Bay Ports in Southern California, the expanded port in Savannah would not require any ships to plug in to electricity while in harbor. The DEIS actually claims that such electrical shore power is "experimental," despite the fact that ports in Los Angeles, Long Beach, and Tacoma are already plugging in ships at some terminals. Recently the Port of Charleston, SC announced that it is going to replace pre-1994 trucks[320] but there are no plans to replace old trucks at the expanded Port in Savannah, according to the DEIS.[321]

The Port relies on the expansive network of warehouse and distribution facilities. There are more than 20 Savannah-area distribution centers that combine to generate 500,000 TEU annually.[322] In addition, there are 200 more distribution centers within a five-hour drive. These include major retailers such as Walmart (3.3 million sq. ft.), IKEA (1.7 million sq. ft.), Target (2.1 million sq. ft.) and Home Depot (1.4 million sq. ft.).[323]

In relation to freight transportation corridor activity, Georgia is also expected to be the first state in the Southeast to finish its expansion of I-95, which will accommodate three lanes of traffic in each direction from South Carolina to Florida. Overall, the expansion is expected to facilitate port growth and tourism in the area. Specifically, the I-95 expansion is crucial to the port because Port of Savannah officials estimate that 80% of the port's containers end up on I-95.[324]

Groups

A number of community-based environmental justice, neighborhood, and faith-based organizations target the port and its proposed expansion projects. Citizens for Environmental Justice/Harambee House is a longtime environmental justice organization, along with the First African Baptist Church represented Savannah at the 2010 "Moving Forward Together" conference in Los Angeles. Other attendees included Operation Street Harvest, from Decatur Georgia and the Center for Sustainable Coast from Saint Simons Island. Through contacts made at the conference, Harambee House developed a working relationship with the Coalition for a Safe Environment in Wilmington (Los Angeles), California. Jesse Marquez of CFASE has travelled twice to Savannah to provide technical assistance. In January 2011, he joined with groups in Savannah to sponsor a community meeting to discuss potential impacts of the proposed dredging of the Savannah River and review the draft EIS released in late 2010. As a result of technical assistance provided to the Savannah organizations, six community groups signed onto a letter and submitted it to the Army Corps of Engineers, asking for a more thorough evaluation of the impacts of the river deepening, including health and environmental justice concerns.[325] USC's community outreach program also submitted comments on the river deepening project, including lengthy documentation of the latest research findings on air pollution and noise that it argued must be considered in the Corps' evaluation.

Together with technical experts, environmental justice activists have been able to engage in formal environmental review processes. Like other community-based organizations however, the groups in Savannah engage in the port expansion debates by necessity rather than choice. Harambee House/ Citizens for Environmental Justice was founded in 1991 after studies showed the overwhelmingly disproportionate number of hazardous waste facilities located in African American communities in the south.[326] Organizing has focused not only on the Port of Savannah but also on the Savannah River Site, a Department of Energy facility and former nuclear reactor, waste management, risk assessment toxic use and public participation to involve local residents in environmental decision-making.[327]

Environmental organizations have weighed-in on the dredging project; seven groups oppose the dredging: Savannah Riverkeeper, Center for a Sustainable Coast, Coastal Conservation League, Altamaha Riverkeeper, Ogeechee Riverkeeper, the Georgia chapter of the Sierra Club, the South Wildlife Federation, and the South Carolina chapter of the Southern Environmental Law Center. One organization, the Georgia Conservancy however, has taken a neutral stance on the important project.[328]

Miami/Port Everglades, Florida

Issues

The Port of Miami is the largest container port in the state of Florida and ninth in the United States.[329] The port handles a mix of containerized cargo and passenger cruise ships and is located just off I-95, the major north-south artery between Florida and the northeast states. Known also as the "Cruise Capital of the World," the ports feature four passenger terminals. Like many other East Coast ports, the Port of Miami is gearing up for the expanded Panama Canal with large-scale projects that include:

- A $1 billion tunnel project under Biscayne Bay to facilitate the truck traffic from the port's eight terminals on Dodge Island to I-395 via Watson Island. When complete, the tunnel will connect the port's eight terminals on Dodge Island to I-395 via Watson Island.[330]
- Deepening of the Miami Harbor to accommodate the largest Panamax vessels. Estimated total cost of the project is $171 million with local funding sources contributing approximately half the cost.[331] The Port originally sought federal funds for the project and the County committed $150 million of local funds toward cost of the project. However, the President's proposed budget plan for 2012-13 did not include the $75 million sought Port officials.[332] The State Department of Transportation announced in early March 2011 that it would fill the funding gap.[333]
- Construction of on-dock rail and rehabilitation of a rail connection to the port. Once complete, the rail line, which was damaged by Hurricane Katrina, will link the port to the nearby Hialeah rail yard operated by the Florida East Coast Railway. The $50 million project, partially funded through TIGER federal stimulus funds, will contribute to Port and rail plans to expand Hialeah into an inland port and logistics hub to stage and shuttle rail cars to and from the port.[334]

Just over 20 miles north of Miami, Port Everglades is located across from the three cities of Ford Lauderdale, Hollywood, and Dania Beach. It is the 12th largest container port in the United States with 5.2 million tons of containerized cargo that move through Port Everglades annually.[335] Port Everglades also has proposed expansion projects, which include a dredging project that is part of the 2006 Master/ Vision Plan approved by the Broward County Board of County Commissioners in December 2007; a $2 billion expansion plan was developed which projects an annual 4% growth of port cargo volume over the next 20 years. A new integrated fumigation plant was opened in January 2011 to facilitate the transport of fresh food products and a new 41-acre terminal Southport Container Yard is in the final phase of completion.

Groups

In Port Everglades, environmental organizations including the **South Florida Audubon Society** pushed for negotiation and a compromise with port officials in February 2010 to adhere to the 20-year old conservation easement in which the port promised to protect a stand of mangroves. As a result of negotiation, the Broward County Board of Commissioners approved a plan that would eliminate 8.5 acres of mangroves in order to expand berth activity and replace it with 16 acres of mangroves at an alternative adjacent location.[336] The **Coalition for Clean and Safe Ports** has also begun to explore a campaign at the Port of Everglades but no active campaign has begun yet. In Miami, environmental groups such as the **Sierra Club** have focused on addressing the environmental impacts of port projects but there is only a limited health and environmental justice focus on the Port and freight transportation.

While environmental organizations have actively engaged in to ports and freight transportation, other well-established and successful organizing groups in South Florida that focus on issues of displacement, housing, gentrification, and economic justice have not yet focused on these issues explicitly.[337] The strength of environmental organizations along with progressive health, economic and environmental justice groups represents a potential environmental and social justice base for building new port and goods movement organizing and advocacy campaigns.

Jacksonville, Florida

Issues

Similar to other regions, increased investment in the seaports of Jacksonville, FL, reflects the competition among local ports for anticipated increased trade through the Panama Canal. Commenting on the Panama Canal expansion's potential impact on the state of Florida, Joe Mazurkiewicz, a political consultant and member of the state's transportation commission, said: "With strategic investment we could position the state of Florida to be the kingpin for everything east of the Rockies and south of Canada."[338] The 2009 Master Plan for the port focuses on the development of port terminals as well as the acquisition of additional property, primarily parcels along the western and northern shores of the shipping channel, a 2-mile stretch of the St. Johns River between the mouth of the Atlantic Ocean and the Talleyrand Marine Terminal, the ports largest terminal.[339] These projects include a terminal expansion project sponsored by Hanjin Shipping company that was original set to open in 2014 to make Jacksonville its hub for East Coast Operations,[340] but construction recently was delayed by two years so that the harbor would have enough time to complete its 48-foot deepening project. The new terminal will not be ready until 2016.[341]

Groups

Specific organizing to inject health, labor, community, environmental and environmental justice considerations have begun to address this growth. In Jacksonville, the **Mayport Village Civic Association** waged fierce opposition to oppose potential cruise ship terminal expansion, arguing that cruise ships would take away important dock space for the long-time shrimp trawlers and expand cruise terminals closer to residential areas, in some cases just 60 feet from residences.[342]

After the Association filed a lawsuit seeking an injunction against the Jacksonville Port Authority (JaxPort) in 2009, JaxPort announced that it would explore other expansion options for the cruise terminal. Nonetheless, a news story in January 2011 stated that Mayport is still in consideration by JaxPort.[343] In the process, the northeast Florida Sierra Club began advocating for use of ultra-low sulfur diesel fuels and electrification of the new terminal, if and when it is built. There are tremendous political obstacles for organizing and advocacy: a review of the JaxPort Master Plan published in March 2009 makes no reference to health risk or air quality standards or diesel emissions despite the expansion proposed at its terminals. Its description of environmental impacts calls them a "reasonable consequence" of growth. The plan states:

> *It is the desire of JaxPort to grow in an orderly manner, carefully addressing the impacts of its growth. As this growth occurs, there will no doubt be some environmental impacts as a reasonable consequence of the industrial processes to port operations.*[344]

While there may be a wide range of opportunities to raise health, community, labor and environmental

considerations in policy debates, JaxPort is overseen by a seven-member Board of Directors: four are appointed by the Mayor and three by the Governor; six of seven represent the private sector with three having direct ties to logistics and trade firms.

Strategies and Organizational Issues

From the description of the issues and organizing in the regions, we find that while communities share similar negative impacts of freight transportation, organizing capacity and political conditions in each region determine the appropriate corresponding strategies. For example, in Los Angeles/Long Beach (home to the country's largest ports) there are numerous organizations and experienced, multi-sectoral coalitions engaged in successful campaigns. Advocates have sought out key allies and formed partnerships with academic institutions, illustrating how the merging of community organizing, advocacy and science can influence policy. In regions with fewer organizations and/or less developed attention to health, ports, and freight transportation, strategies are geared towards education and identifying opportunities for establishing coalitions and networks. Despite the wide variation between groups and strategies across the United States, examples point to the need for capacity in three areas: increasing knowledge and awareness of issues, strengthening organizational effectiveness, and building political power sufficient to influence policy and decision-making.

Education and Direct Organizing

Increasing awareness of the negative impacts freight transport has on community, worker, and environmental health, particularly in directly impacted communities, serves as the necessary baseline for informing the public as well as engaging the range of necessary stakeholders in freight transport planning and decision-making.

Peer to Peer Learning and Training

Peer learning and one-on-one trainings have clearly helped to educate, mobilize, and connect communities across the country on this issue. Popular education materials such as the development of "Goods Movement 101" (a project developed by East Yard CEJ along with USC) serves as a popular education training tool (also available in a customizable PowerPoint format for other goods movement communities) that presents the system of ports and goods movement, the negative impacts, and examples of solutions that improve community health and quality of life. The Pacific Institute's popular education training manual, "Gearing Up for Action: A Curriculum Guide for Freight Transport Justice," also provides important educational tools for community groups.

Moreover, peer-to-peer learning and training can help address the tensions that emerge within and between organizations. Building coalitions and developing long-term trust among labor, community, environmental, environmental justice, and health interests is a necessary but challenging effort. When left unaddressed, tensions between labor and community, mainstream environmental and environmental justice goals and process may result in instances where environmental and conservation goals conflict with broader community, environmental justice, and health and community development goals. Without opportunities to dialogue and learn from each other groups may ultimately cut individual deals to mitigate some off port impacts and miss addressing others. Similarly, tensions between community and labor interests may slow or undermine campaigns. Identifying opportunities and deploying appropriate and relevant methods for facilitation and conflict resolution to address these tensions is critical to short-term campaign success as well as longer-term movement building.

Coalitions and Networks

Building the relationships between a wide range of stakeholders and building capacity for the groups to take action together requires organizational structures that are able to bridge interests, bring together resources, and facilitate shared agenda setting and action. For many regions, coalitions have served important roles in bridging interests and helped build political power that individual groups may not achieve on their own.

These coalitions have been key engines for progressive policy change in ports operations and goods movement. Tackling the freight transportation system and forging solutions to address negative impacts of projects and operations require command of a broad range of issues - e.g., air and water quality, health risk, and land use along with policy and technical capacity that often no single organization is able to tackle alone. Instead, freight transportation-related coalitions of organizations bring together the necessary health, labor, community and environmental perspectives and organizational resources to focus on influencing specific goods movement related decision-making. Whether labor-community coalitions or coalitions made up of broad stakeholders, such collaborations enable single issue and community-specific groups to work toward a common goal.

Coalitions across the country have helped ensure that industry is not able to promote economic development and job growth without necessary health and environmental protections. In Southwest Detroit, for example, the formation of the Southwest Community Benefits Coalition is an important step. However, given the influences of industry, high unemployment rate, and low capacity of local government to direct private sector development projects, securing permanent and long lasting benefits will require deep relationships and attention to longstanding issues facing Southwest Detroit's low-income, communities of color. In Gulfport and other right-to-work states where labor has less influence, freight transportation organizing and coalitions rely heavily on environmental justice, community, and other worker rights organizing as well as community-based research and legal strategies.

New coalition organizations such as worker centers are emerging in Chicago and the Inland Valleys that serve to bridge community and labor union interests and focus on workers as community residents as well as laborers. This draws on the resources of nonprofit organizing institutions as well as labor unions. National networks such as the Coalition for Clean and Safe Ports and the Clean Air Task Force also serve as important resources that provide opportunities for capacity building, linked policy development, and organizing strategies.

An overall national network focused on the broad efforts around the goods movement sector can additionally play a role in strengthening local campaigns, providing information sharing tools, such as an interactive web site, providing links to researchers, and enhancing outreach to new constituencies while also expanding media coverage of key issues. Such a network has been requested by participants at the "Moving Forward Together" conferences and efforts to build it are underway. Ultimately, a national network can play a role, with its regional and state counterparts, in changing the discourse around freight transportation to make community, health, environmental, and labor impacts more central in project planning policy.

In addition, through coalitions, community-based organizations have gained access to and engaged support from important technical, legal, and scientific resources. It was a coalition that filed the successful lawsuit against the City of Los Angeles' failure to provide public environmental review over the construction of the China Shipping (North American) Holding Co. Another coalition used a legal challenge to the Los Angeles Harbor Department's limited environmental review of the TraPac terminal expansion and as part of the settlement, secured a $50 million mitigation fund (see above). In Edgerton, Kansas, NRDC's lawsuit filed on behalf of the Intermodal Coalition along with the Hillsdale Environmental Loss Prevention (HELP) organization has temporarily slowed the proposed project giving community residents an opportunity and time to educate and organize themselves. NRDC also helped make arguments on behalf of the City of Los Angeles in legal challenges filed by the American Trucking Association to block implementation of the City's Clean Trucks Program.

Coalitions and collaborative organizations are also a vehicle for community organizations to gain access to important scientific research and to inject community knowledge into research. While the complexity and scale of goods movement and port operations require academic scientists, trained lawyers, and the resources of mainstream labor organizations and advocacy groups, grassroots organizing and leadership development must be at the core of building long-term capacity and leadership focused on freight transportation. The policy successes in Southern California reflect how experienced base building strategies tied to coalition building result in strong campaigns and policy change.

Ensuring Health in Decision-making: Power and Policy Innovations

Despite the ever-growing evidence about the nature of these widespread health, environmental and workplace issues, these findings have not been widely incorporated into policy decisions about expanding the size of ports and the freight transportation system in the United States. Powerful economic and political actors continue to push for expansion of the global trade and goods movement system even as the negative community, environmental, health, and labor costs mount. Decisions by global retail chains such as Walmart are able to dictate the scope and scale of how and where goods are produced and moved. These retailers work closely with and influence powerful shipping, logistics, and other freight companies that in turn influence developers and government decision-makers about expansion of ports and infrastructure. In addition, fragmented regulatory authorities mean that state and local agencies are unable to effectively address ship emissions (regulated at the international level) or locomotives (regulated at the federal level). Interstate commerce laws prevent local and state governments from exercising authority over freight transportation, although numerous legal challenges are underway. Lobbying by the freight transportation stakeholders often results in less strict regulatory enforcement and the use of public subsidies for new infrastructure projects.

Industry's power to frame the debate about freight transportation as an economic engine that creates new jobs has provided policymakers with political cover to override community public health, and workplace concerns about further expansion. With the needs of private interests treated as a public good, the government ends up underwriting goods movement infrastructure at the local, state, and national levels. Lobbyists for the freight industry are especially effective in pressing national legislators into funding new transportation projects. The Center for Public Integrity (CPI) in Washington, D.C. has singled out the work of a group called the Freight Stakeholders Coalition, with 17 national members, to document how this industry maneuvered to influence federal transportation spending.[345] This Coalition includes members from the trucking, ports, rail, road building, retail, export/import, and shipping industries. CPI's investigation found that the members of the Coalition spent $28.5 million to lobby Congress in the first six months of 2008, much of it on transportation issues. The Center has also examined the road-building lobby, noting that it spent "more than $65 million on total lobbying expenses last year including paying more than 300 lobbyists to weigh in on federal transportation policy."[346] Included in this group of lobbyists are construction firms, the sand, gravel and cement industries, environmental consultants, the trucking industry and more.

Following President Obama's call for an infrastructure initiative in October 2010, a bipartisan panel of transportation experts recommended major investment in infrastructure projects.[347] The panel included representatives from industry such as the American Association of Railroads, CSX Transportation, Inc., Norfolk Southern Railroad, American Trucking Association, and the American Road and Transportation Builders Association. Public health, environmental, and labor constituencies were excluded from this process. Consequently, the 92-page report, called "Well Within Reach - America's New Transportation Agenda," mentions "health" only once and completely ignores how such a plan would affect noise and air pollution from increased ports, rail yard and highway activity.

At the local level, community residents are faced with an increasing array of goods movement related highway expansion projects, intermodal rail facility growth, the construction of massive distribution centers and never-ending retail stores. They find themselves at odds with development proponents like the Freight Stakeholders Coalition who argue that their projects will be "green" and that they will provide much-needed jobs. Local politicians commonly adopt this "green jobs" frame even when it results in land use policies that put communities at risk and expose workers to economic insecurity.

Nevertheless, successful campaigns and organizing initiatives have resulted in models for injecting health, community, labor, and environmental consideration into goods movement decision-making and simultaneously serving to bring a range of stakeholders together and fuel coalition and movement building. In the section that follows, we present examples of policies that address health, labor, community and environmental considerations in goods movement and that have served as rallying points around which coalitions and local organizations have come together. These examples illustrate the challenges of building capacity, in terms of both knowledge and awareness of issues but also the need to build political power sufficient to influence freight transportation policy and decision-making. We begin with examples in three areas: air quality policies, clean trucks programs and community benefits agreements as successful campaigns that involve coalitions of local and regional groups across the country. We conclude the section with areas where community-based policy campaigns have begun to emerge. Both successful and emerging campaigns recognize the need for strong public policy to ensure that goods movement, decision-making, and projects address the negative impacts on health, community, environment, and labor and that public investment in ports and freight transport systems leads to healthy communities and good jobs.

Air Quality Policies

Across the country, diesel emissions policies have been adopted by regional and state governments, important steps in using air quality standards to address the negative health impacts on communities. Diesel policy campaigns at the local, state, and national levels, once passed and implemented, are important levers for cleaning the air generally, and for focusing attention on emissions from ports. For example, local and regional groups such as the New Jersey Environmental Justice Alliance and the East Michigan Environmental Action Coalition work closely with the national Clean Air Task Force and its Diesel Cleanup Campaign to push for a 70% reduction of diesel emissions by 2020 through engine retrofits, replacements, and use of diesel fuel alternatives. In 2003, California environmental justice and health groups pushed for and won an anti-idling port truck bill which limits port truck idling to 30 minutes when outside port property. Working together, groups in Southern California pushed for anti-idling programs such as rules approved in 2006 by the South Coast Air Quality Management District (the regional authority for air pollution in Southern California). In Oakland and Richmond, the Ditching Dirty Diesel Campaign has also been engaged in similar policy campaigns related to truck idling.

Groups have worked successfully to influence federal level air policy. Members of the Clean Air Task Force are organized to secure Congressional action to fully fund the Diesel Emission Reduction Act (DERA) which provides the opportunity for fast-acting diesel reduction measures, such as requiring the retrofit of public fleets, but which has lacked adequate funding since its original passage in 2005. These coalition efforts reflect and build on federal agency programs such the "Credit for Clunkers" Clean Truck Initiative by U.S. EPA to reduce pollution from large trucks with financial incentives to assist with compliance. A provision in the federal Transportation Reauthorization Bill will require and fund the installation of modern pollution controls on diesel construction equipment used on federally funded transportation projects.

At the international policy level, health and environmental advocates have begun to focus on the inadequate and often negative rulemaking involving regulations of ships by the International Maritime Organization (IMO), the United Nations agency responsible for safety and security of shipping and the prevention of marine pollution by ships. Organizing and advocacy efforts focus on how local governments and state regulatory agencies can play a role (albeit voluntary) to reduce vessel speed in port waters. This provides a strategy to reduce pollution for ocean-going ships that come into port that also bypasses the industry-friendly IMO.

Similarly, the focus on regional and state action has sought to address rail issues that often escape federal intervention. This included the 2005 rules adopted by California's South Coast Air Quality Management District (SCAQMD) that restricted rail idling. As anticipated, the rail industry fought back and challenged the ruling. In September 2010, a federal judge ruled in favor of the rail industry, arguing that SCAQMD could not impose restrictions that interfere with interstate commerce. Environmental justice and health activists are now pushing to develop statewide regulations. In California, community and environmental justice groups have fought to win regulations on port and goods movement operations. See California Air Resources Board for rules such as Cargo handling equipment and vessel fuel rules (www.arb.ca.gov/parts).

The link between energy source and use and emissions has also become a part of the policy arena for activists. As discussed above, this organizing has begun to result in policy advances to protect health. The China Shipping lawsuit in Los Angeles, for example, resulted in electrification of the 176-acre China Shipping Terminal. A new China Shipping Terminal there will also feature electrification. The Port has signed memoranda of understanding with five other shipping lines to implement alternative marine power on vessels and/or their Los Angeles terminals. The Port of San Diego has also recently approved $7.6 million to install electrical plug-ins at its cruise-ship and 10th Avenue terminals so visiting vessels can shut down their diesel engines while in port. On the East Coast, the Port of Miami has begun to electrify its cruise ship terminal although no plans are in place to do the same at its cargo port. The Port of Oakland also received a boost for its shorepower plan with a $5 million grant from the Bay Area Air Quality Management District. The Port of Seattle now requires that cruise ships plug into electricity when docked in the harbor and the Port of Tacoma recent plugged in its first ocean going cargo ship. Environmental and community coalitions in Los Angeles have pushed the San Pedro Bay Ports to implement low sulfur fuel programs targeting shipping lines and terminal operators. The Los Angeles port as well as the New York/New Jersey ports has adopted low-sulfur fuel programs although both are only voluntary.

Clean Trucks Programs

The adoption of clean truck programs across the country point to important policy innovations that can achieve improved health outcomes simultaneously with labor, environmental and community benefits as well. The Clean Trucks Program adopted in Los Angeles, for example, represents an innovative approach to ensure health outcomes through an employment strategy. The approval of the policy reflected the breadth and power of a multi-constituency-based coalition and campaign. The campaign and the policy has become an important national test case for the degree to which such policies will face opposition, and for assessing the level of skill, capacity, resources, and power it takes to win - and implement - the program.

The Clean Trucks Program was approved as a key piece of an earlier adopted policy the Clean Air Action Plan (CAAP). In 2006, the Ports of Los Angeles and Long Beach, adopted the CAAP as the first clean air emissions policy by port authorities in the United States; it was also one of the first times that the two competitive ports in Southern California have partnered on any endeavor). In addition to requiring the development and implementation of a clean trucks program, the policy requires that all new projects meet a residential cancer risk threshold of 10 in 1 million, a goal the ports can only achieve through the use of cleaner equipment and vehicles. Some of the freight industry players, especially the railroads, have strongly opposed key CAAP goals.

The adoption of the Clean Trucks Program in Los Angeles in 2008 as a key program of the CAAP marked a historic and pathbreaking win for port truck drivers and members of the Coalition for Clean and Safe Ports. For the port drivers, the successful campaign,

led by the Teamsters and joined by other labor unions and immigrant labor groups, directly addressed the trucking system put in place with the deregulation of the industry in the 1980s that required independent truck drivers to bear the burden of all maintenance and upkeep for trucks that cost over $100K, along with Port fees, licensure, fuel and other costs of doing business at the Port. In Los Angeles, truck drivers - who owned and operated their own trucks and must compete individually for hauling jobs - net less than $30,000 annually.[348] [349] By adopting a Clean Trucks Program that includes an employee concession model as a key aspect of its implementation, the Los Angeles Harbor Department saw the need to improve air quality by replacing port trucks and recognized the importance of putting the cost of doing business at the Port on port trucking firms rather than individual truckers. As employers, trucking companies must now shoulder the costs of the clean air program instead of the individual truck drivers. Moreover, as employees, truckers now have the right to organize.

The policy was immediately met with opposition from the American Trucking Association (ATA) that filed an injunction blocking the implementation of the employee concession element of the Program from moving forward. The Coalition stepped up its support for the Mayor and the Harbor Department's decision and organized to generate support for the Program.

In August 2010, a federal court judge cleared the way for full implementation of the Program but an appeal filed by the ATA put yet another stop to the implementation of the policy. The judge ruled that the program could be implemented during the appeals process except for the employee concessionaire component that continues to be held up in the legal process. The legal tug of war and the inability of the City of Los Angeles to implement the full program has put additional burdens on the truck drivers who are now required to retrofit their trucks but without any means to do so. As a result, port drivers are foregoing necessary maintenance on their trucks because they cannot afford the costs.

In New York/New Jersey, the Coalition for Healthy Ports built a campaign around the Los Angeles model and participated in the Port Authority's Truck Working Group to develop the clean trucks program. With the legal challenges to the Los Angeles program however, officials balked. Instead a new version, the Regional Truck Replacement Program, contained only a ban of 1994 and older trucks (Los Angeles' plan bans 2004 and older) without the attendant concession system that would require trucking firms to hire drivers as employees. With $7 million of ARRA funds (a grant from the U.S. EPA to the Port Authority) and an additional $23 million of its own funds, the Port Authority set up a loan program for drivers to make the required retrofits to get the estimated 640 old trucks off the road. According to Amy Goldsmith, an organizer with the Coalition for Healthy Ports, most drivers are not eligible for the loans due to restrictive loan criteria and only ten have received one. The ban went into place on January 1, 2011 and many drivers are expected to be out of work as a result. One advocate argued: "There will be no diesel relief, no driver relief, and no community relief. The Plan is a low-road loan model that will have an outcome far worse than what existed before."[350]

Clean trucks program advocates argue that the use of public funds to give loans to drivers will push drivers into financial crisis adding expensive loan payments when they are barely making ends meet and which does nothing to clean the air.[351] This has been the case in Oakland where a similar loan program has been put in place. In a recent survey of drivers who participated in a loan based program in Oakland, 25% of port truckers that participated in the Port of Oakland's financing plan have either filed bankruptcy, lost their home to foreclosure or been evicted.[352] Further, the Port Authority of New York and New Jersey expects its similar loan program to have the same kind of negative impact on drivers with a predicted 1 in 5 default rate. As part of a national legislative strategy, the Coalition for Clean and Safe Ports pushed for the introduction of a Congressional bill, the Clean Ports Act of 2010, to amend the Federal Motor Carrier Act to allow ports to enact and enforce clean truck programs and implement environmental programs above the current federal requirements. The bill was first introduced in July 2010 by Rep. Jerrold Nadler (D-NY) along with 57 co-sponsors; the bill was reintroduced in February 2011 as a way to maintain federal level attention on issue of misclassified drivers and the need for increased authority at the local level.

Adoption of the Clean Trucks Program in Los Angeles reflected the coming together of oft-competing groups - labor, mainstream environmentalists, environmental justice, faith based, and civil rights organizations - all aligned around the fight for good jobs and clean air. The coalition and the policy win also represents an example of what could happen if movements and public officials align and if movements help public officials hold steadfast to key positions and policies. Moreover, the win established a national policy model for other social movements and policymakers in Port and inland communities impacted by global trade and goods movement.

These alliances are critical to counter powerful industry interests who have challenged the health protective policy at every step. Across the country, the campaigns for clean trucks illustrate the challenge and also potential of achieving policy wins at the local as well as national scales. As the policy continues to be

tested in the courts, regional clean trucks campaigns continue to seek to develop the most effective program and policy implementation. Lessons from the Clean Trucks campaign in Los Angeles point to the important role of environmental justice organizations as able to provide the important link between labor and environmental goals with community and health goals in labor-community campaigns. Additionally, the presence of committed labor organizations in coalition with community, environmental justice and health groups, created a community-based yet regionally and nationally powerful coalition able to withstand divide-conquer strategies from opponents of the trucks program.[353] Yet, political pressure by industry along with legal challenges waged by the American Truck Association has resulted in policymakers defaulting to clean trucks programs without the important but contested employee concession element.

The Los Angeles program, therefore, has had mixed outcomes More than 9,000 2007 compliant trucks are now registered to move in and out of the ports and an emissions inventory conducted in 2009 showed a 50% reduction in truck-related emissions due to the initial implementation of the program as well as the 2008-2009 recession.[354] At the same time however, without the employee requirement, the burden to retrofit and maintain these trucks falls to low-wage earning truck drivers, leaving the program in an unsustainable place.

Project Mitigations and Community Benefit Agreements

Combining community organizing and coalition strategies to secure legally binding, project related community benefits agreements has resulted in progressive policies and precedents for securing health, environmental, community and labor protections in goods movement projects. An example from the late 1990s in Southern California was the successful efforts of the Alameda Corridor Job Coalition formed to secure benefits from the $2.4 billion construction of the 20-mile long Alameda Corridor Rail project that built a below-grade rail line connecting the Port of Los Angeles to the rail intermodal train traffic network near downtown Los Angeles. The Coalition negotiated with the Alameda Corridor Transportation Authority to secure a local hiring agreement for construction of the rail project and also funds for community-based organizations to recruit and train residents for entry into pre-apprenticeship and apprentice training in the construction and building trades.

In 2002, NRDC on behalf of community and environmental groups in Los Angeles filed a lawsuit against the City of Los Angeles and Harbor Department for failing to comply with California's Environmental Quality Act (CEQA) when it reviewed the proposed expansion of the China Shipping terminal at the Los Angeles port. The final settlement a year later required the Port to establish a $50 million fund to mitigate the impacts of Port operations in the community and commit to specific steps to address pollution at the new terminal and among other alternative fuel and technological goals, provide electric power for ships berthed at the China Shipping terminal.[355]

Five years later, despite strong community, environmental and health criticism of an inadequate CEQA review in measuring the environmental and community impacts of the proposed expansion, the Port of Los Angeles Harbor Commission approved the TraPac project. The proposal sought to build out the terminal and expand volume to a level equal to the current operations of the Port of Oakland, the fourth busiest container port in the United States. At build out, the terminal expansion would increase ship calls by 30% and add 1,800 daily truck trips.[356] With NRDC's assistance environmental justice, resident, environmental, labor, public health and community groups submitted a letter to the City Council seeking to appeal the Harbor Commission's decision.

The coalition that came together around the appeal worked together to develop a community-based settlement that proposed establishment of an independent nonprofit that would administer an off-port mitigation fund. This would be financed through fees related to future expansion projects and provide over $50 million in off-port property community mitigation projects such as: the installation of air purification and sound proofing systems in public elementary schools and residents' homes, public respiratory health care services at local community clinics and health services providers, studies of off-port property impacts on health and land use, off-port property impacts on public safety, traffic, aesthetics, light glare, recreation and cultural resources and potential wetlands restoration projects in Wilmington and San Pedro. After much negotiation, the City of Los Angeles and the Port of Los Angeles agreed to the settlement in 2008. In his blog about the Port Community Mitigation Trust Fund, NRDC's Adrian Martinez writes, "...people would have thought pigs would fly before community, environmental and port leadership would work together to mitigate harmful impacts from port operations."[357]

In Detroit, the Southwest Detroit Community Benefits Coalition formed to secure community benefits related to building new freight transportation infrastructure. The Coalition was formalized in 2007, four years after community organizations had begun discussions about how to ensure community needs were met by development in Detroit and focuses in three areas: transportation (e.g.tunnel and rail projects), weatherization (e.g., community-benefits

in construction and building retrofits), and food delivery (e.g., creating greater neighborhood food options).[358] A primary target is the New International Trade Crossing (NITC), the international bridge crossing between Detroit and Windsor, Canada. Much of the organizing focuses on state level decision-making since U.S.-based administration of the bridge lie with the Michigan Department of Transportation and the U.S. Federal Highway Administration.

Through project-specific mitigation settlements and community benefit agreements, local communities and workers have been able to leverage benefits out of port expansions and freight transportation projects. However, benefits agreements and project mitigations often do not address the root cause of the conditions communities and workers seek to mitigate. As an example, the TraPac mitigation fund provides an impressive $50 million toward much needed health protective retrofit projects in communities. Continued funding, however, relies upon increased growth of freight at the port. Nevertheless, organizing around the community benefits agreements and project settlements have brought together important groups to raise health, community and labor considerations in decision-making, an important movement building approach that has the potential to shift power and policy in the long-term.

Emerging Policy Campaigns

In addition to successful integration of health into air policies, clean trucks programs, and project mitigation agreements, there are a number of areas where health, labor, community and environmental organizations and coalitions have begun to make connections between goods movement and broader issues.

Military base re-use and redevelopment

At local levels, local organizing and advocacy groups have begun to address planning related to ports and military facilities. Similarly, community groups and local government have begun organizing to influence the redevelopment planning of the former Oakland Army Base. In the case of Oakland, the redevelopment process has provided an opportunity for residents to engage in port development policies and planning. In some cases port planning overlaps with redevelopment planning as in the case of the March, Norton, and Edwards air bases (located in Southern California). However, newly elected California Governor Jerry Brown has proposed the elimination of the state's redevelopment agencies, making it unclear about the future of redevelopment in California cities. In Joliet, Illinois, organizers are looking at redevelopment opportunities as an opening to secure legal employment standing for warehouse workers.

Alternative energy efforts

Campaigns to electrify rail and ships have yet to emerge at the national level, although local groups have built such demands into their campaigns. In Los Angeles, the campaign to halt the rail expansion projects includes an alternative to electrify and utilize the publicly owned Alameda Corridor rail corridor to its full capacity before building additional rail projects. Groups such as the Coalition for a Safe Environment (Wilmington CA) and the Interfaith Community Organization in Long Beach have advocated for Magnetic Levitation (MAGLEV) trains as viable options for consideration, and the Port of Los Angeles has committed to a test project with this technology.

Natural resources

Emerging campaigns and focus on the effect the shipping industry has on coastal and inland waterways. In early March, 2011, due to pressure by environmental and conservation organizations, the U.S. EPA announced that it will issue a new permit to U.S. shippers regulating ballast water discharges from commercial vessels. The discharge of ballast water is the prime source of invasive species that have negatively affected local and regional ecosystems. For example, ballast water from ocean-going ships has contributed to the 185 invasive species now living in the Great Lakes.[359] The agreement settled lawsuits brought by NRDC, Center for Biological Diversity, the National Wildlife Federation, and many regional environmental organizations that challenged the legality of EPA's existing permit.[360] Although the legal and advocacy efforts to address the impacts of freight transport on water and natural resources are not new, there is little engagement between these environmental campaigns and public health and social justice efforts.

Climate policy

In California, AB 32 set in motion a process of land use planning requiring regional planning organizations to implement plans that reduce the vehicle miles travelled by cars and light trucks as a way to reduce greenhouse emission. Although the focus on land use planning to reduce greenhouse gas emissions does not include emissions from the transport of freight in heavy-duty trucks, AB 32's implementation has major implications for communities that are already disproportionately impacted by freight transport. There have also been efforts to consider funding for diesel black carbon reductions as part of federal climate legislation. Although climate change legislation at the national level is now in hiatus, this topic is an important arena for advocacy, education, and future campaign initiatives.

Land use and health

The connection between land use, the environmental and public health is also becoming part of an emerging policy landscape. One of the country's first known buffer zones to separate sensitive uses (homes, schools, hospitals, etc.) and goods movement facilities was adopted in 2002 in Mira Loma, CA, then an unincorporated section of Riverside County where distribution centers were under development. The community-based group Center for Community Action and Environmental Justice (CCAEJ) worked with a regional air quality task force to encourage adoption of a 1000-foot buffer zone, which was later included in "good neighbor" guidelines adopted by the regional council of governments. In 2005, CCAEJ was successful in urging Riverside County to pass truck routing and parking regulations to address the large volume of trucks in residential areas. Using the buffer zone policy as a guideline, the planning commissioners voted to unanimously deny any new warehouse project proposal near sensitive receptors, resulting in 700 acres of land removed from industrial development.[361]

Organizing by the Environmental Health Coalition in San Diego led to National City becoming the first city in California to adopt an environmental justice element into its general plan. The plan includes for example, important language to create buffer zones: "... Create adequate distance separation ... between sensitive receptor land use designations and potential identified sources of hazardous substances such as freeways, industrial operations or areas such as warehouses, train depots, port facilities, etc."

The Clean Up Green Up campaign in Los Angeles seeks to establish "green zones" in environmental justice communities to require stepped-up enforcement and investment priorities to address the existing toxic burden in certain neighborhoods and facilitates new green business development. The campaing includes groups working on port and freight transportation who recognize the role of land use to address mobile and stationary sources of pollution. In Richmond, CA activists successfully organized to get the City to adopt a Health Element in the City's General Plan, as a guiding framework for all project and planning decision-making throughout the city.

The Shifting Landscape of Federal Policy

The Clean Ports Act of 2011 (see also a discussion of Clean Trucks programs above) illustrates an important coalition-led strategy to win federal level policies to ensure health, labor, and community consideration in goods movement. In the economic and political climate facing the 2011-2012 Congress, advancing health, labor, environmental and community protective policies and programs will be difficult but there are openings and possibilities including the following:

Federal Transportation Reauthorization Bill

Ports and goods movement campaigns have begun to focus on legislation, federal programs, and regulatory requirements and will have impact on policies and funding streams at the state, regional and local levels. The Federal Transportation Reauthorization Bill as well as the "Focusing Resources, Economic Investment, and Guidance to Help Transportation Act of 2010," (S.3629) also known as the FREIGHT Act, was introduced in the Senate last year. A similar FREIGHT Act was also introduced in the House (H.R.5976). In addition, the Freight Rail Infrastructure Capacity Expansion Act (FRICEA) (H.R.1806) proposes tax incentives for improvements to freight rail infrastructure. This includes projects such as intermodal facilities and terminals at ports. The Senate Environment and Public Works Committee have begun hearings on federal Transportation Reauthorization and bills are expected to move through Congress in 2011, either separately or part of the omnibus Federal Transportation Reauthorization Bill. This new timeline has pushed health, community, and labor advocates to develop campaigns at the federal and state level in order to influence decision-making about funding criteria and priorities.[362]

The Interagency Task Force on Environmental Justice and Plan EJ 2014

The White House's Council on Environmental Quality recently re-established the Interagency Task Force on Environmental Justice that includes U.S. EPA as well as the Department of Transportation, Department of Housing and Urban Development, Labor, Health and Human Services, Commerce, and others. This renewed focus on environmental justice at the federal level creates openings for integrated and comprehensive policy approaches that could support local and governments to address the health and environmental risks of goods movement.

The U.S. EPA also recently adopted its "Plan EJ 2014" as the overarching way to "expand the conversation on environmentalism and work for environmental justice." The goals of the plan are to: 1) protect health in communities over-burdened by pollution; 2) empower communities to take action to improve their health and environment; and 3) establish partnerships with local, state, tribal and federal organizations to achieve healthy and sustainable communities. The Plan EJ 2014 plan supports the recommendations made by the National Environmental Justice Advisory Committee (NEJAC) Goods Movement Work Group (GMWG) that address local and regional health, community, labor and

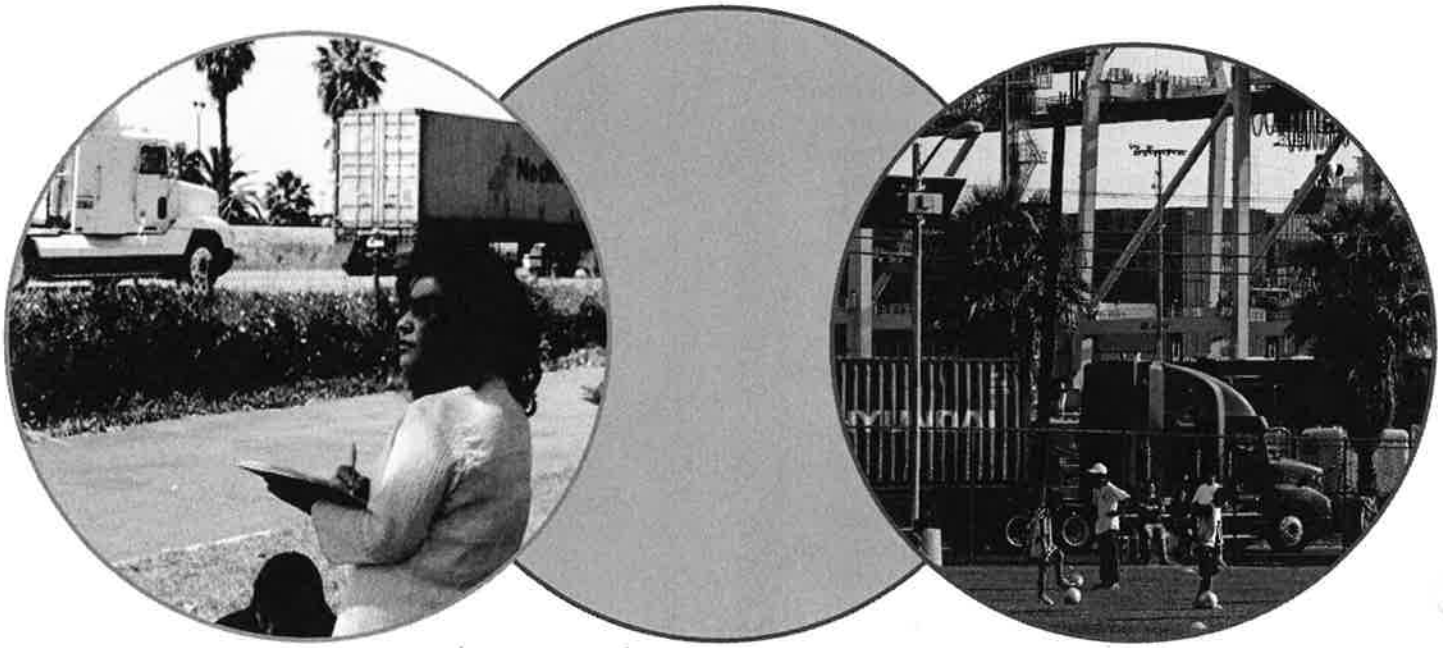
industry concerns. The group's 2009 report[363] directly addresses the environmental, public health and quality-of-life impacts of goods movement and includes recommendations on land use (buffer zones), funding priorities, alternative fuels and decision-making about siting, in particular looking at what U.S. EPA has authority to do.[364] The GMWG recommendations were adopted by NEJAC, and the report has been forwarded to the head of U.S. EPA, Lisa Jackson. Some of the recommendations have begun to be implemented, but much more work needs to be done.

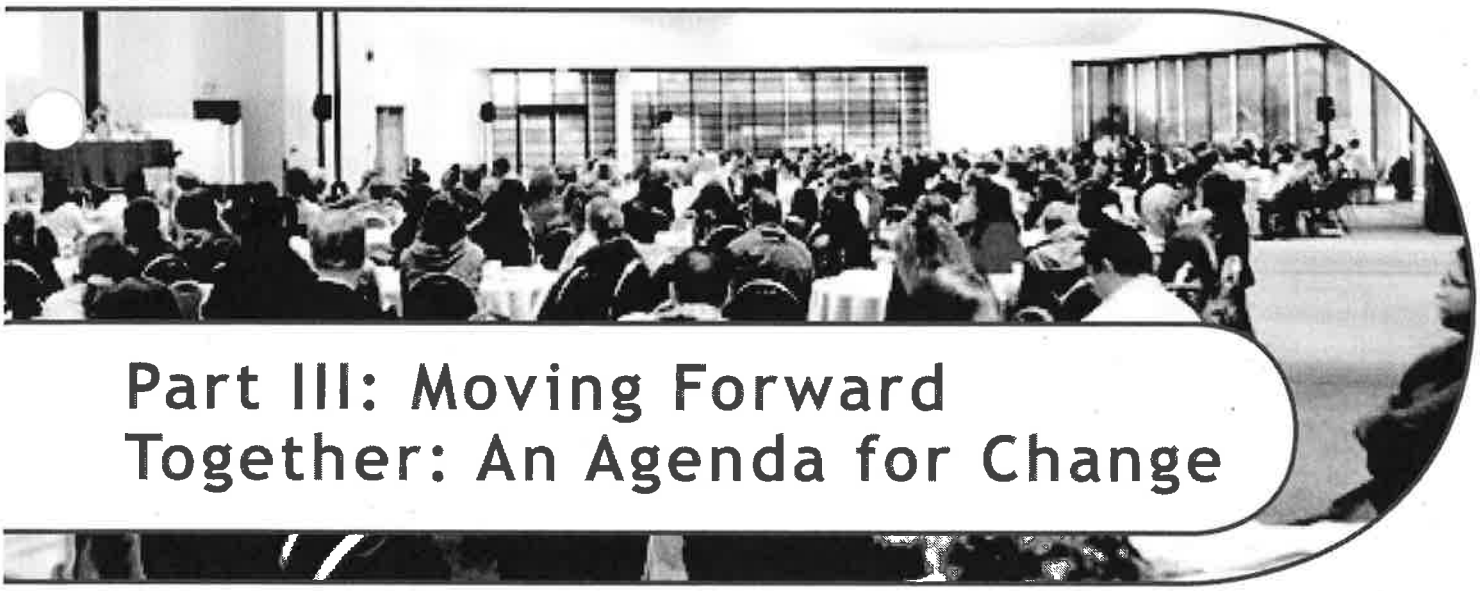
Policy Capacity for Change

Because the goods movement policy debates are framed primarily as economic development and job creation approaches, negative impacts on health, labor, community, and environment continue to be seen only as externalities. However, the groups profiled above have made significant advances to insert these issues into policy debates and decision-making at the local, regional and state levels. Achieving health, labor community, and environmental protections in goods movement decision-making however, also requires federal level policy. The powerful industry challenges to local and state policy advances described earlier in the report illustrate this point.

Organizing and advocacy groups as well as researchers and local and state legislators will need to engage nationally with others like them working to incorporate heath considerations into decision-making and develop effective and influential strategies to engage in national level debates and decision-making. As our cases illustrate, there will be considerable challenges in the face of powerful industry interests. For example, federal funds for port dredging and expansion projects at the Port of Miami, the Charleston Harbor, and the Port of Savannah were not included in the President's proposed budget for 2012-13.[365] Although negotiations over the budget will likely yield changes in proposed funding, heath, labor, community, and environmental coalitions face considerable challenges influencing these decisions given the unequal playing field that tilts toward industry. As our examples illustrate, influencing these national policies along with state, regional and local level planning, policy and project decision-making will require continued and expanded integration of research on the health impacts of freight transportation, widespread community organizing, deeper and more extensive coalitions, and a political will to push for progressive rules and policies.

While much attention focuses on injecting health and environmental agendas into freight transportation and ports policy, organizers also see ports and goods movement policy as a means to achieve clean air and healthy communities in a context where other health and environmental policies may not exist. In regions where there are large port complexes and where air pollution substantially results from ports and goods movement activities, changing ports and goods movement policy may be the most efficient way to achieve environmental goals such as improving air quality. At the same time, groups that primarily focus on freight transportation issues find themselves seeking to develop policies limited to a specific issue related to separate environmental, health, community, or labor agendas. The key - and challenge - is integrating the efforts together into common policy campaign and longer-term movement. When that happens, strong organizing and coalition efforts result in notable policy interventions and innovations that forge health, community, environmental and labor considerations into goods movement policies and decision-making.





Part III: Moving Forward Together: An Agenda for Change

In order to build the capacity of the groups, organizing, and policy innovations that have been described and effectively change how the global trade and freight transportation issues are being framed, several key areas for action emerge. Organizers, researchers, advocates, funders, and policymakers each have a role to play in advancing action in these areas in order to ensure that health, labor, environmental and community considerations are addressed in freight transportation.

Ensure public notice and participation policies

Across the regions, public policies related to public notification and participation have varied widely. In places like Savannah, Charleston, and Miami, port authorities do not make use of the internet to make their agendas available online or post meeting notices and minutes. Without easily accessible information, communities may be unaware of proposed projects and the impact on their health and communities. Some ports, such as New York and Los Angeles (through its Port Community Advisory Committee), have institutionalized a formal community advisory committee, comprised of residents, businesses and other stakeholders, that meets regularly and provides input to the governing body of the Port. The Ports of Los Angeles and Long Beach both videotape their Harbor Commission meetings and have agendas, searchable transcripts and videos online for the public to view.

Connect local organizing to regional organizing

The system of freight transportation directly impacts adjacent neighborhoods and port communities but also impacts communities along freeways that serve as freight corridors, near rail yards and rail lines, and near distribution centers and warehouses. Organizing, research and funding at the regional level captures the relationship between Port communities with the broader impacts of goods movement and the relationship with freeway, rail yard, and distribution center development. For example, organizers in San Diego utilize a regional framework that enables them to link their neighborhood organizing work in Barrio Logan to trucks that cross the Mexican border. In Southern California, environmental justice and warehouse worker organizers in Riverside and San Bernardino counties are forging links with Los Angeles-based organizations as a way to confront a regionally defined system of goods movement that sets a broader common agenda. For those organizing in small Kansas towns like Gardner and Edgerton, where a new rail yard is proposed to be built soon, organizing regionally will enable local efforts to coalesce with other resources and organizations

in Kansas City, who may have experience working on issues related to the nearby Argentine rail yard. Similar regional issues exist for areas like Chicago where organizers face the challenge of bridging bridges between newer suburban warehouse workers with inner city rail yard workers in Chicago's urban core.

Strengthen, expand, and link national networks and international ties

Freight transportation and the logistics industry rely on distribution and consumption that is both national and global in scale. Organizing and influencing this system requires not only an integrated regional approach, but also a parallel national advocacy network of public health, environmental and social justice, and labor advocates. A national network has emerged from the "Moving Forward Together" conferences (2007 and 2010), sponsored by USC and THE Impact Project. The "Moving Forward Together" conferences also helped identify important international connections that can provide linkages to share information and establish shared approaches around specific campaigns and larger change agendas. For example, community groups in Australia are fighting a large intermodal facility in Liverpool and the heavy volumes of truck traffic through residential communities near Melbourne. See Appendix for contact information and organizational websites.

Strengthening this network will provide an important national scale vehicle for continued information sharing, strategizing, research, and resource sharing across a wide range of constituencies interested in and concerned about freight transportation in their communities. Other national network organizations such as the Clean Air Task Force serve as important vehicles for local and regional organizers and advocates to strategize about issues such as diesel pollution and climate change. The labor-anchored Coalition for Clean and Safe Ports represents a unique national coalition focused on implementing the successful clean trucks program in port communities across the United States. These national organizations provide important technical and policy support and serve as critical organizational infrastructure for local and regional organizing and advocacy to achieve scale in terms of influencing national policy. The Natural Resources Defense Council's air office in Santa Monica, CA provides significant legal and technical support to Los Angeles groups and selected ports/rail communities around the country. Building a national network focused on port and freight transportation will complement efforts by these national organizations.

Strengthen and expand research on health and environmental impacts

An increasing number of studies link adverse health and environmental impacts with freight transportation activities. More research in this area needs to be conducted, particularly around rail yard emissions, (where there have been few studies to date), on the health effects of noise, on the potential reproductive effects of exposure to diesel exhaust, on air toxics in diesel exhaust, on ultrafine particles, and on the range of chronic diseases linked to air pollution exposure. In addition, community-university partnerships serve as important vehicles to integrate public health research with advocacy, organizing, and policy campaigns. These partnerships provide important research, organizing, and advocacy infrastructure. Examples of engaged academic institutions include:

- The **Detroit Community-Academic Urban Research Center** (URC), a collaboration of the University of Michigan Schools of Public Health, Nursing, and Social Work, the Detroit Department of Health and Wellness Promotion, and eight community-based organizations in Detroit which focuses on asthma as well as freight transportation and international trade between United States and Canada.
- The **Center for Urban Economic Development (CUED) at the University of Illinois at Chicago** (UIC) provides technical and research support to Warehouse Workers for Justice to examine the trends of logistics industry jobs and the impacts of the expanding warehouse industry has on workers.
- The **Center for the Urban Environment at Thomas Edison State College, New Jersey,** works with the New Jersey Environmental Justice Alliance and other groups in Newark and New York on issues involving the ports.
- **Loma Linda School of Public Health** recently began a study of the health of several thousand residents living near a large freight rail yard in San Bernardino, CA, in conjunction with a community-based organization.
- The **Labor Education Center, Rutgers University, New Jersey** conducts research on the impacts of deregulation on truck drivers and on other labor issues.
- The **Southern California Environmental Health Sciences Center & Children's Environmental Health Center, based at the Keck School of Medicine of USC** conducts studies on the health effects of air pollution, including in goods movement communities and in close

proximity to highways. The Centers include a community outreach and engagement program that focuses on ports and goods movement issues and coordinates THE Impact Project.

- The **Southern California Particle Center based at UCLA**, in conjunction with several community-based environmental justice groups in Southern California, studies the toxicity of particles emitted from equipment at rail yards in Commerce, West Long Beach, and San Bernardino.
- The **University of Medicine and Dentistry of New Jersey's Environmental and Occupational Health Sciences Institute** works with a community-based organization in Newark, NJ on a study of triggers for asthma in children, including diesel soot.
- The **University of South Carolina's Department of Epidemiology and Biostatistics and Institute of Families in Society** works closely on environmental justice issues with community-based organizations in Charleston, SC.
- The **Urban & Environmental Policy Institute at Occidental College** in Los Angeles conducts community-based research and facilitates linkages between labor, community, environmental and social justice organizations, funders and policymakers, and is also a partner in THE Impact Project.
- The **University of Washington's School of Public Health** is engaged with community and labor organizations on a study of diesel trucks and exposures to air emissions.

These forms of collaborations will be critical for deepening the levels of research, organizing, and advocacy necessary to move forward health protective public policies related to goods movement.

Forge and promote public policies that integrate community and worker health protective measures into freight transportation planning and project approval

Comprehensive methods of environmental assessment need to become part of all freight transportation projects and planning. Environmental Impact Reviews/Statements (EIRs and EISs) and Environmental Assessments (EAs) should be required to include comprehensive assessments of all health-related impacts as well as environmental impacts. Assessment tools such as Health Impact Assessments (HIAs) are promising approaches for assessing comprehensive environmental and health impacts. Environmental justice organizations and public health advocates in Southern California have been successful in getting the California State Department of Transportation to agree to include an HIA to assess impacts of the widening of the I-710 Freeway, a major goods movement corridor in Southern California. Meanwhile, the U.S. EPA (Region 9) is proposing the use of an HIA at the Port of Los Angeles. A group called Human Impact Partners is engaged in both of these efforts, conducting the I-710 expansion project's HIA and conducting scoping for a possible Port HIA.[366]

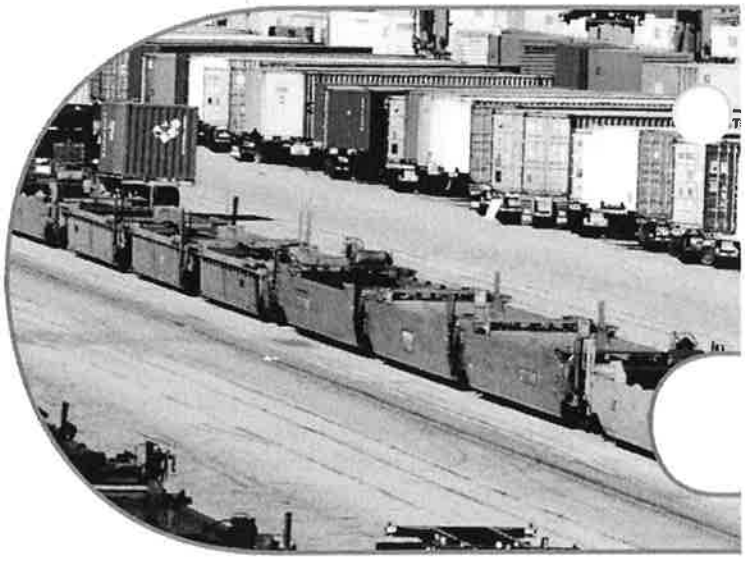
HIAs are a step toward "Health in All Policies" (HiAP) - an approach to policymaking being advocated in many European countries and by many progressive health departments in California. HiAP urges finding opportunities to add a health lens in public policy and program development and to increase collaboration across agencies and with communities. HiAP is an all-inclusive approach to policymaking that would promote healthy communities and ensure that "health" is considered in all transportation policies.[367] [368]

Identifying all health impacts is the critical first step in evaluating risks and the negative impacts of freight transportation. Addressing them will require innovations in technologies and policy requirements and incentives to "clean-up and green-up" the facilities and operations that have operated for decades along with proposed and future projects. Government policies and programs must certify, pilot and promote these technologies to address existing pollution as well reduce future risk.

Increase local government capacity

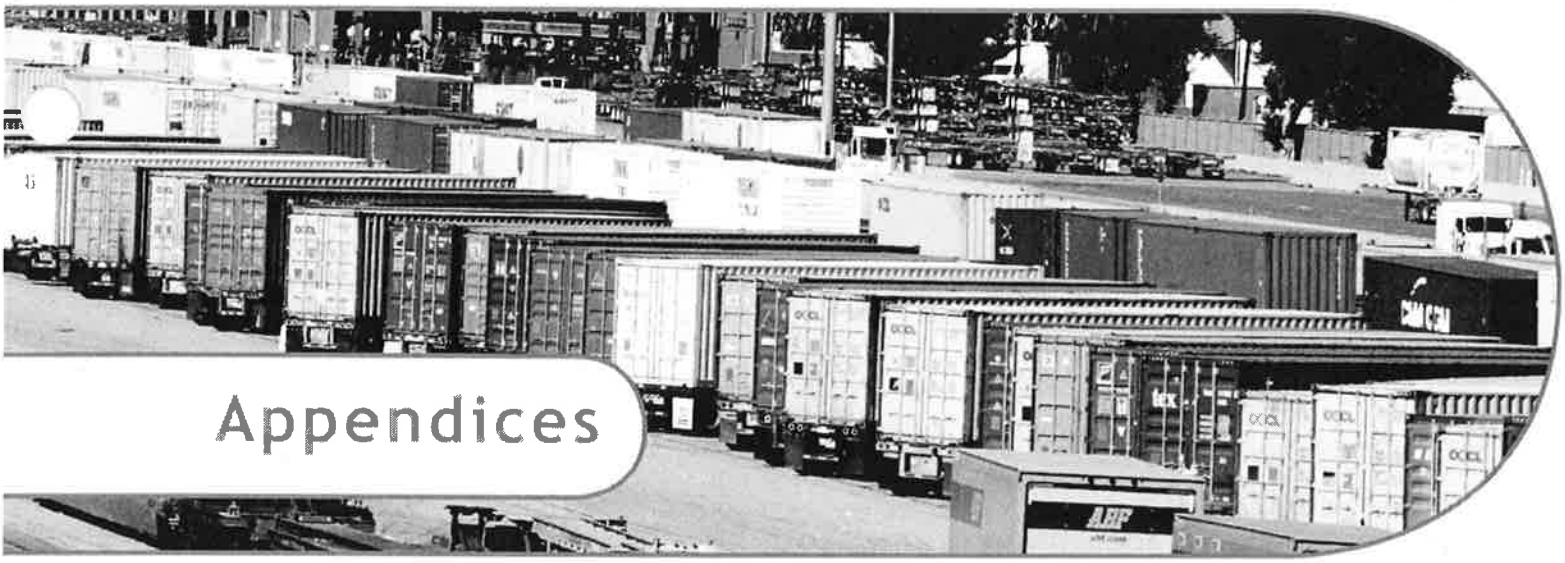
Cities across the country face enormous economic and budgetary challenges. The result has been layoffs, furloughs, and elimination of environmental and regulatory enforcement programs. New partnerships with the nonprofit sector, when deeply rooted in the public sector, can provide local authorities more tools and resources to address health and environmental impacts of goods movement. In the City of Commerce, CA, for example, members of East Yard Communities for Environmental Justice successfully urged the City Council to adopt an environmental justice resolution and an Environmental Justice Task Force. Further north, in the City of Richmond CA, organizing and policy advocacy led by the Richmond Equitable Development Initiative (REDI), pushed that City to include a specific Health Element in its General Plan. In December 2006, The California Endowment - through Policy Link, a national research and policy organization - awarded the City of Richmond an initial $255,000 grant to develop a Health Policy Element for the General Plan. By including a specific Health Element in the General Plan, city officials and the residents of Richmond have the opportunity to assess the health impacts of all of the major development projects.

Broaden and strengthen the movement



In order to challenge the way goods are moved throughout the country, organizers and advocates must be able to connect and deepen the relationships between a wide range of constituent groups in order to build a broad-based movement that links health, labor, and environmental aspects of goods movement. As a system, goods movement represents a "maypole" around which many other issues can be linked and organized: public health, environmental justice, consumer rights, consumerism, community and economic development, regional planning, climate justice, land use, housing and transportation, and food security. At the national scale, "connecting the dots" across movements can integrate health, environment, community, and labor, and bring discussions of climate change, smart growth, sustainable food systems, regional transportation planning, and health to the goods movement discussions and decision-making.

In sum, this report has been designed to demonstrate how efforts to incorporate community, environmental, health, and labor issues into the global trade and goods movement discussions about policies and impacts are crucial. There is a growing movement of people and organizations across the country that are concerned about the health of their communities that has taken steps to reframe the debates and change the ways that global trade moves throughout the United States. These groups have forged and pushed for important policy solutions. However, without continued growth and power of engaged community, labor, health, and environmental interests across the country, these new rules and policies for ports and freight may fail to address the critical health needs facing communities along the entire system of trade. These efforts are now at a crucial juncture, requiring greater awareness about the importance of the agenda for change that extends at each point along the global trade and goods movement system's pathways.



Appendices

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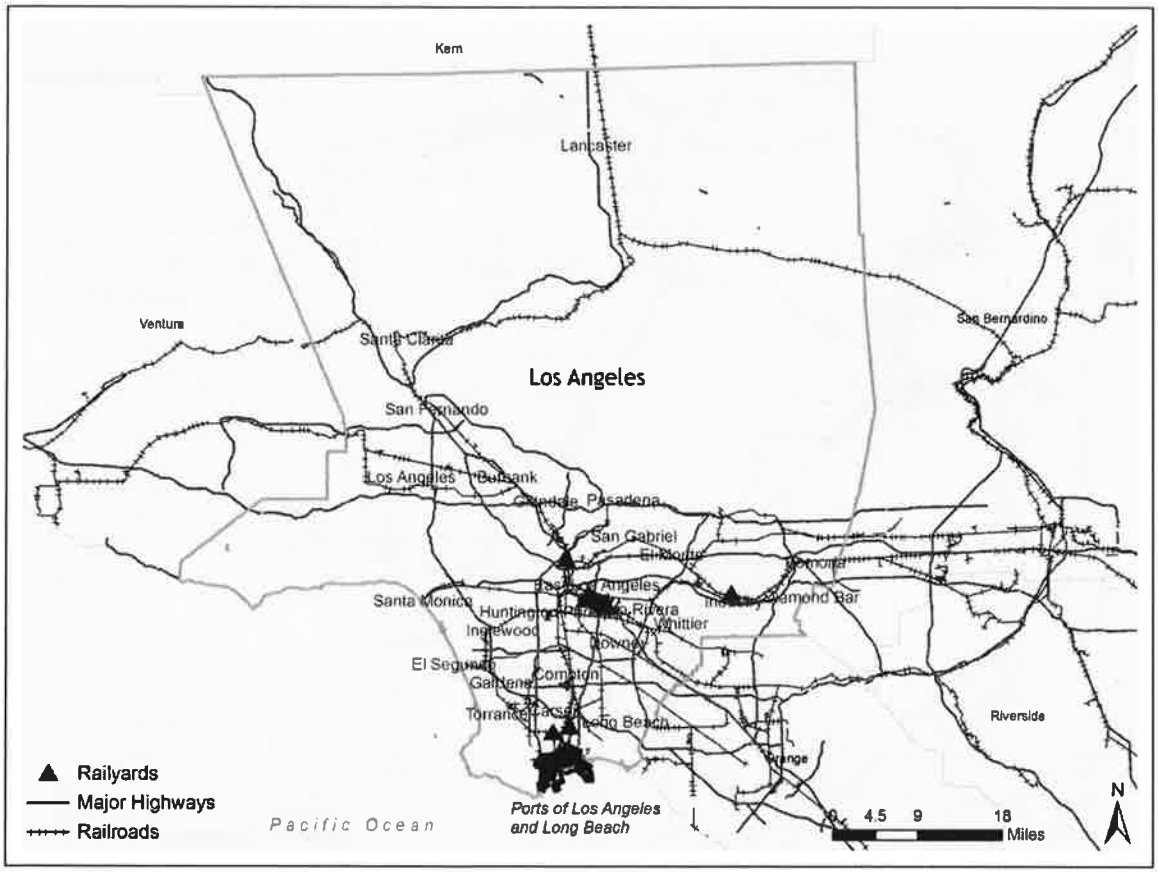
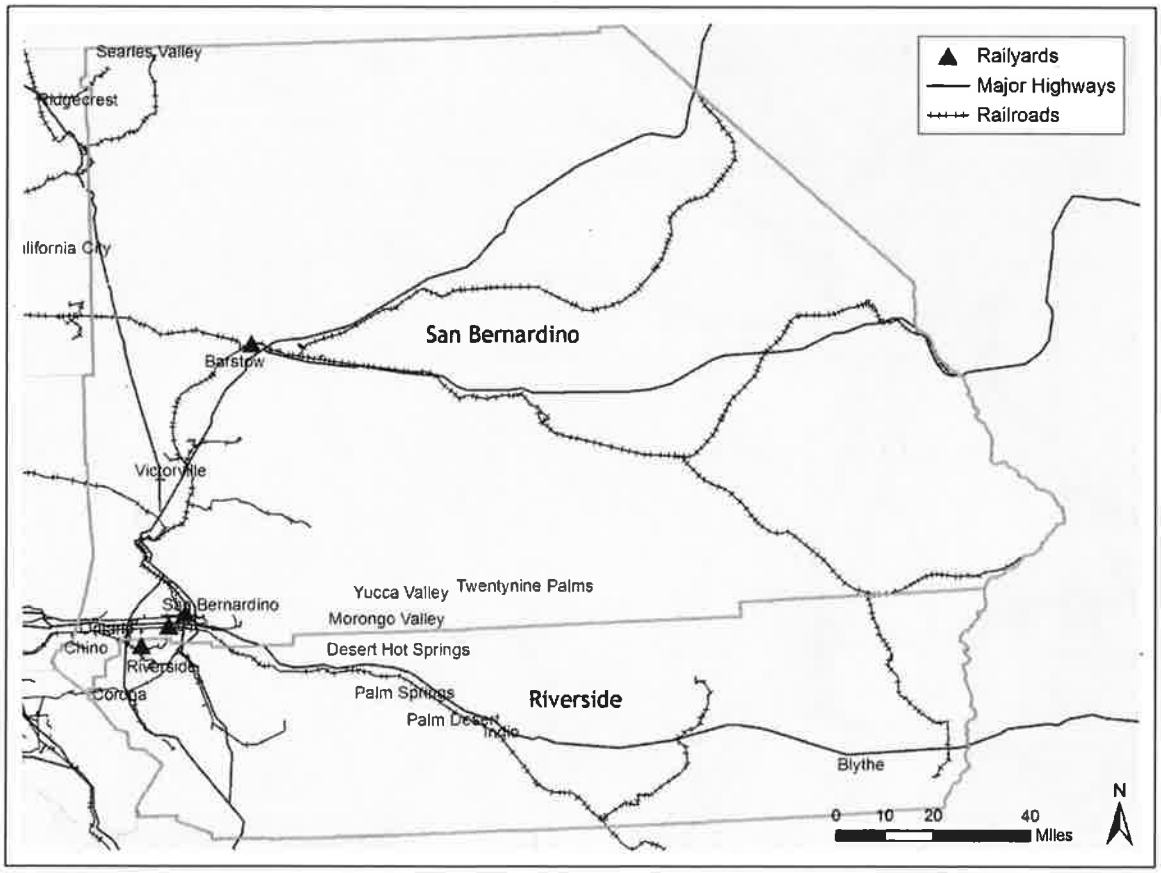
Christopher DeScherer
Charlottesville, Virginia
http://www.southernenvironment.org

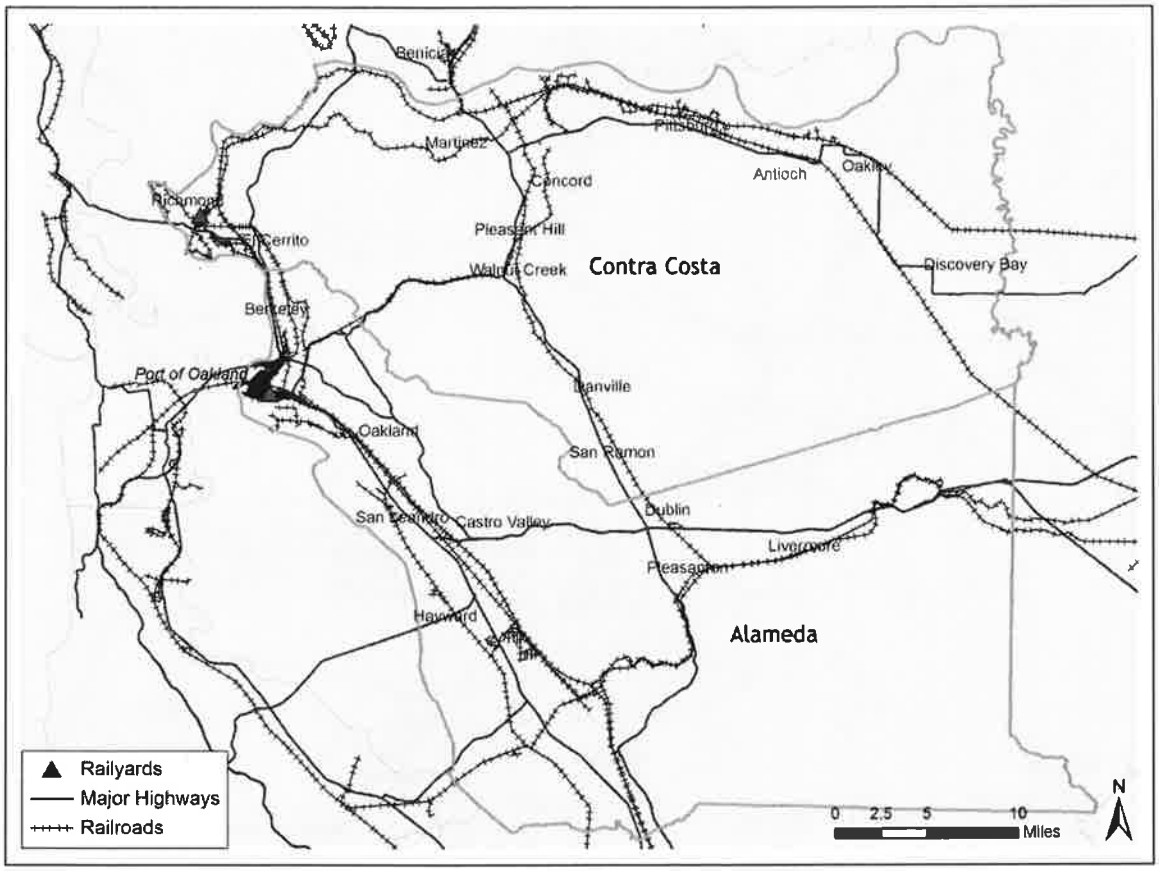
Los Angeles/Long Beach, California Map



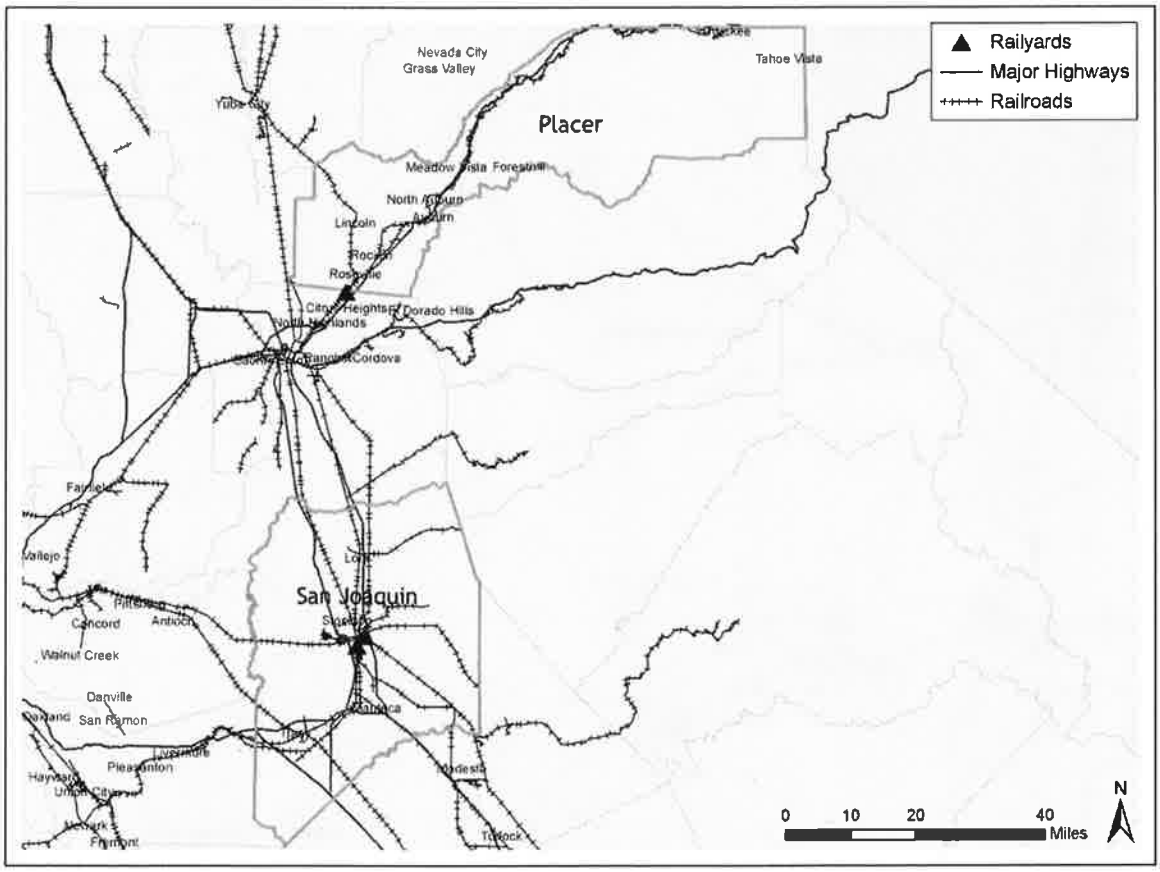
The Inland Valleys: San Bernardino and Riverside Counties, Californa Map



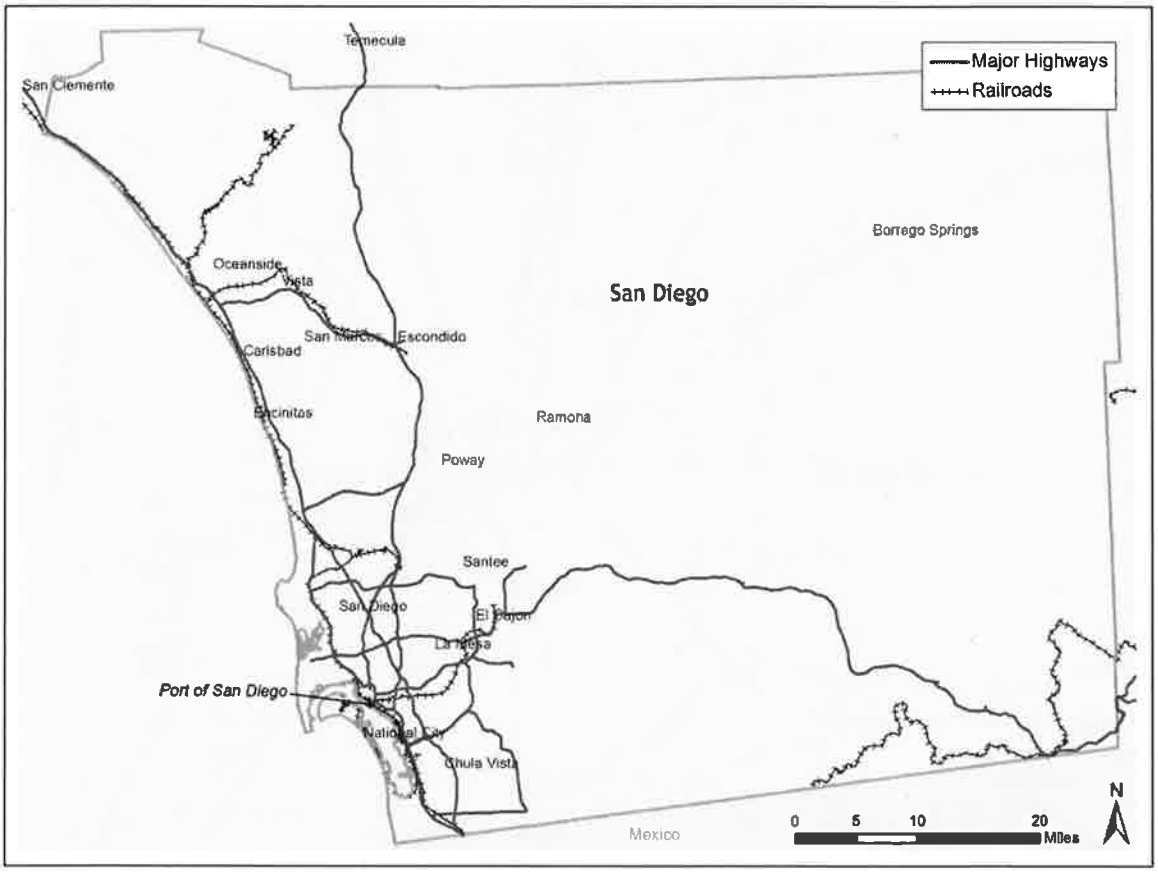
Oakland/East Bay, California Map



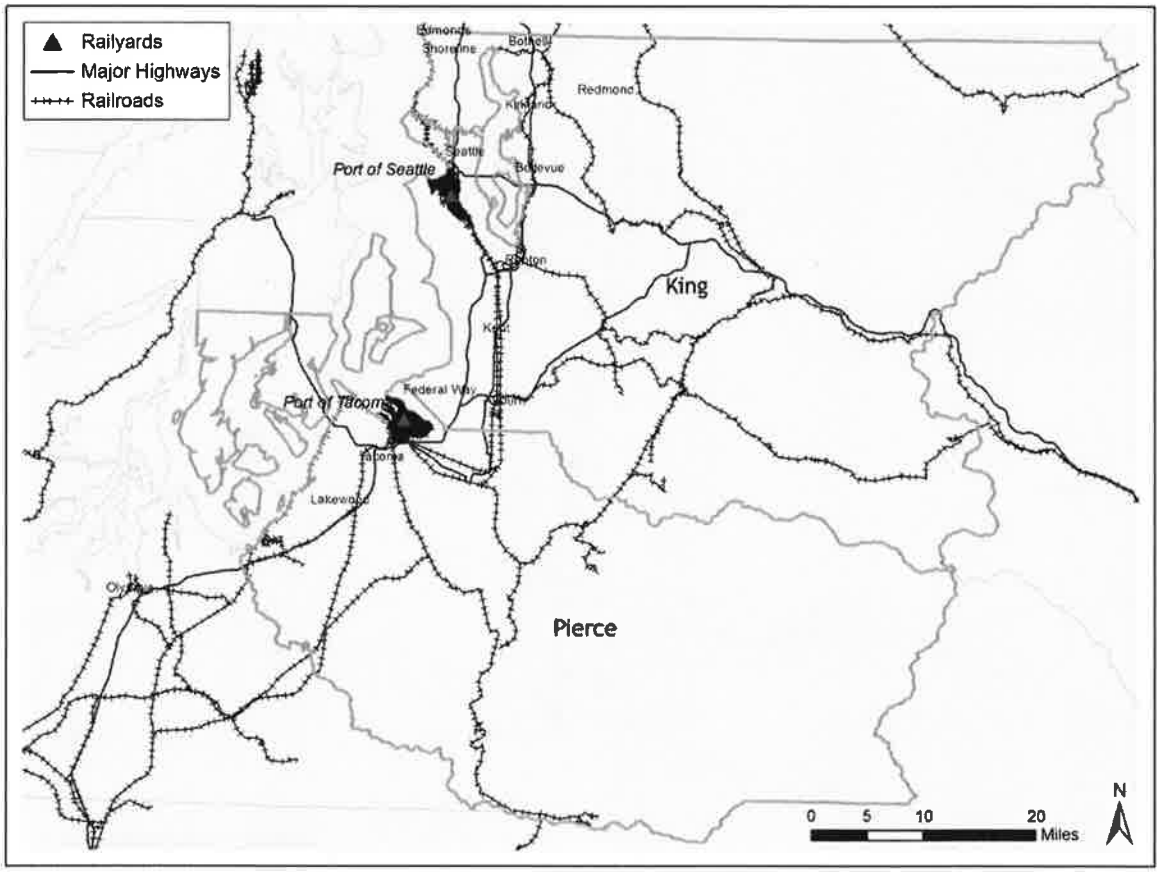
Central Valley: Stockton, California Map



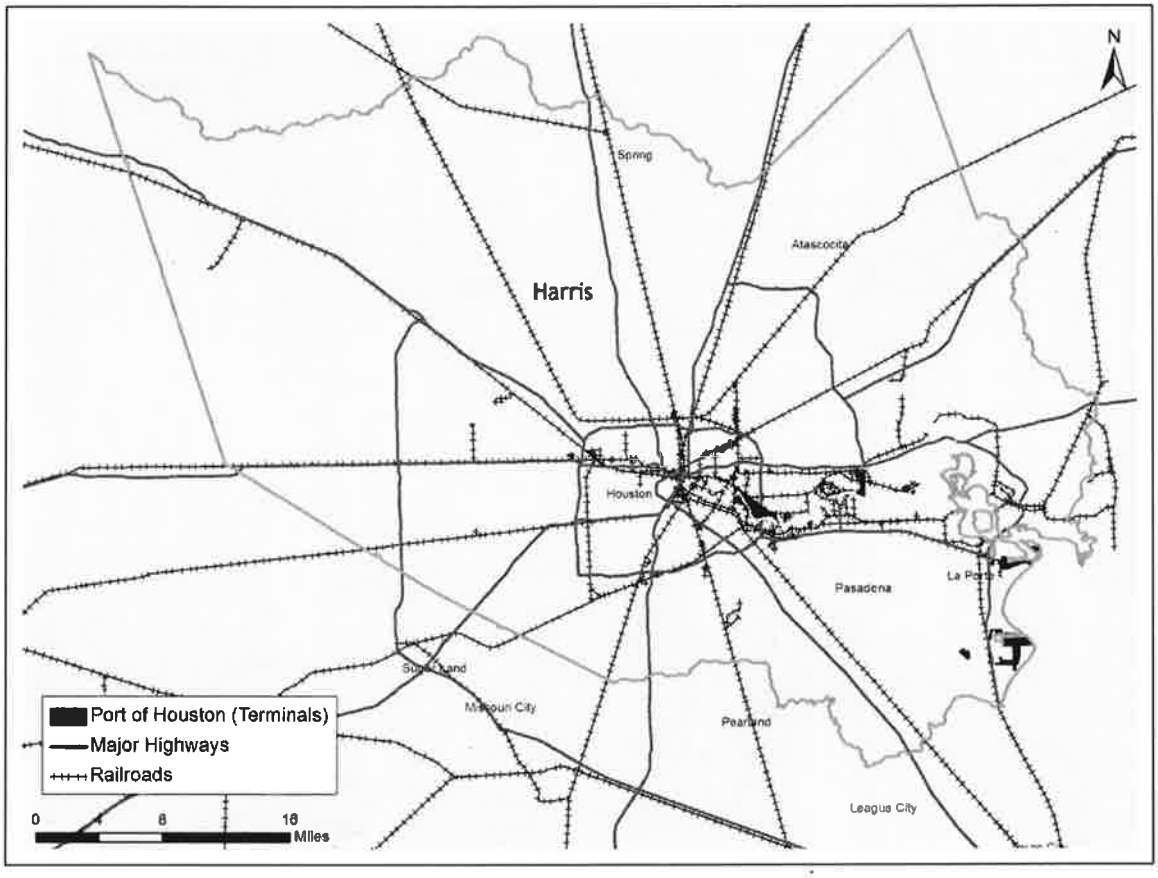
San Diego, California Map



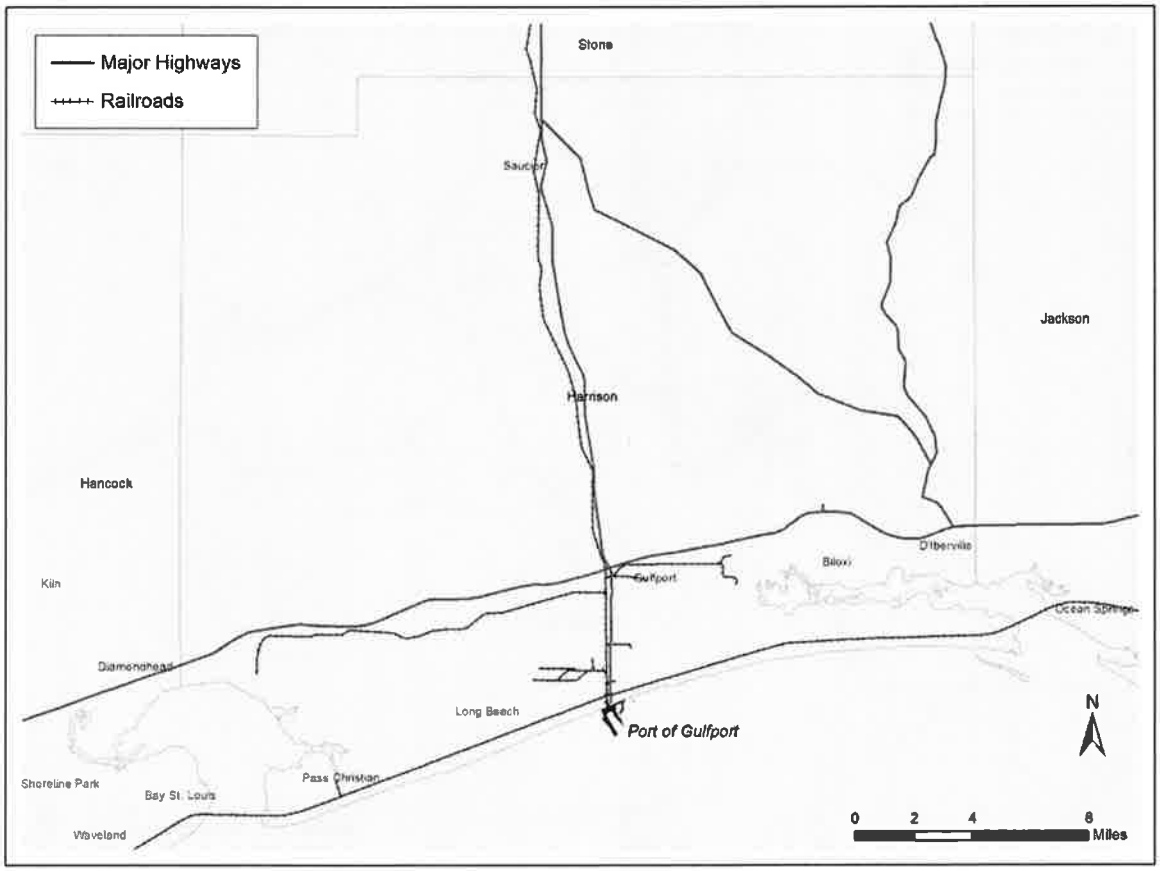
Seattle and Tacoma, Washington Map



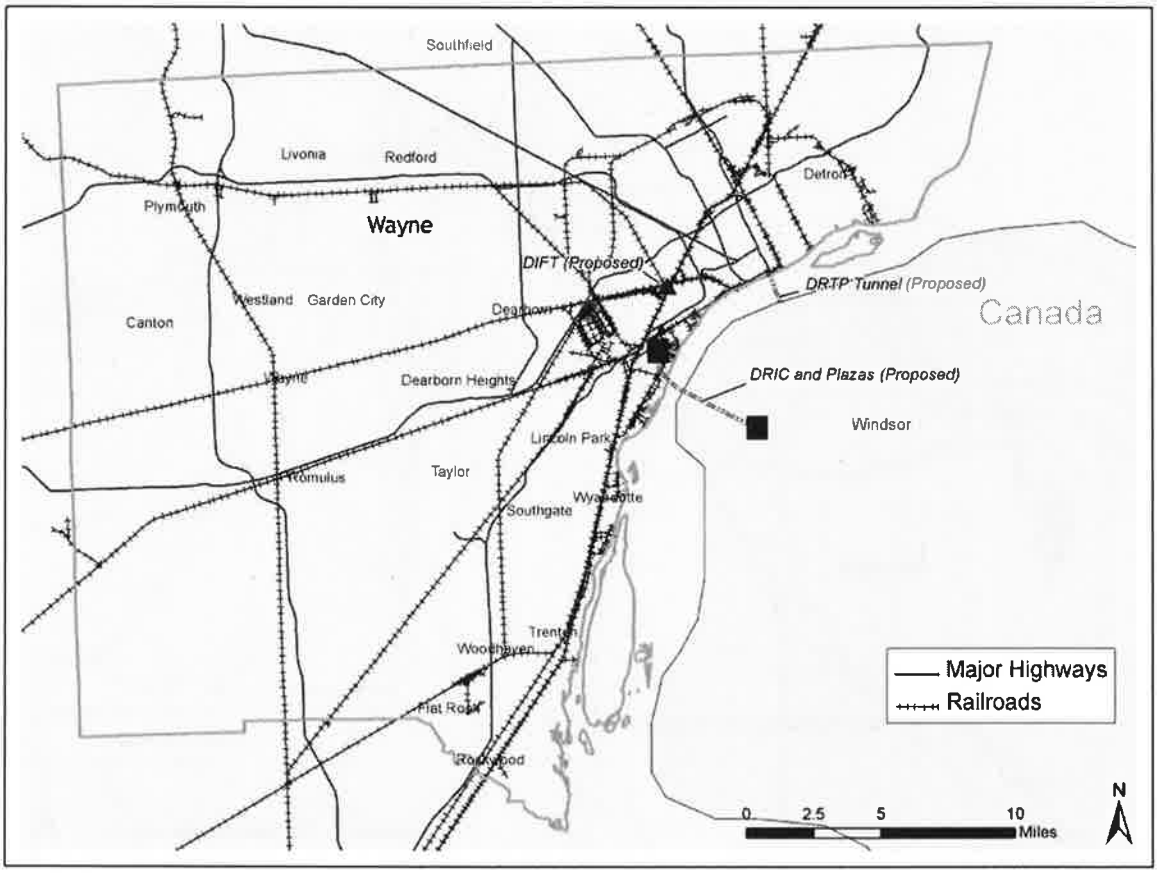
Houston, Texas Map



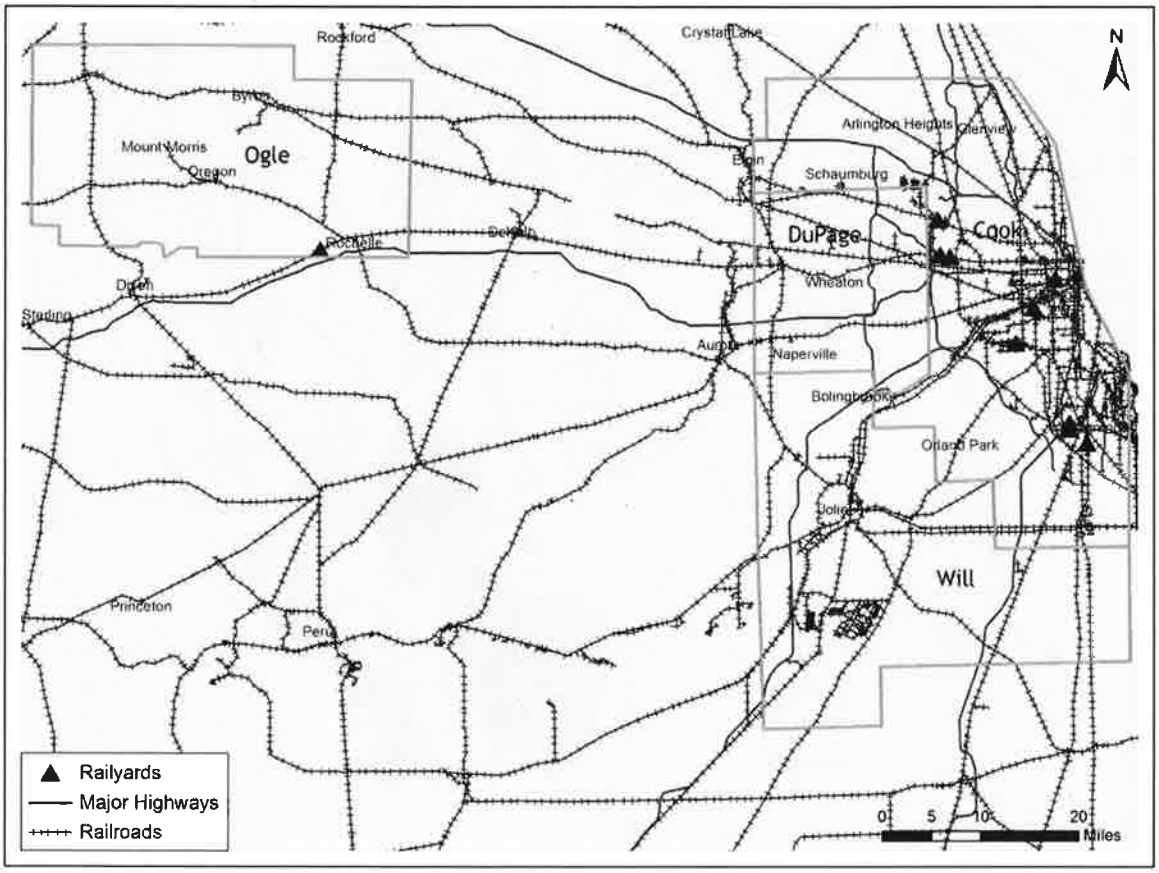
Gulfport, Mississippi Map



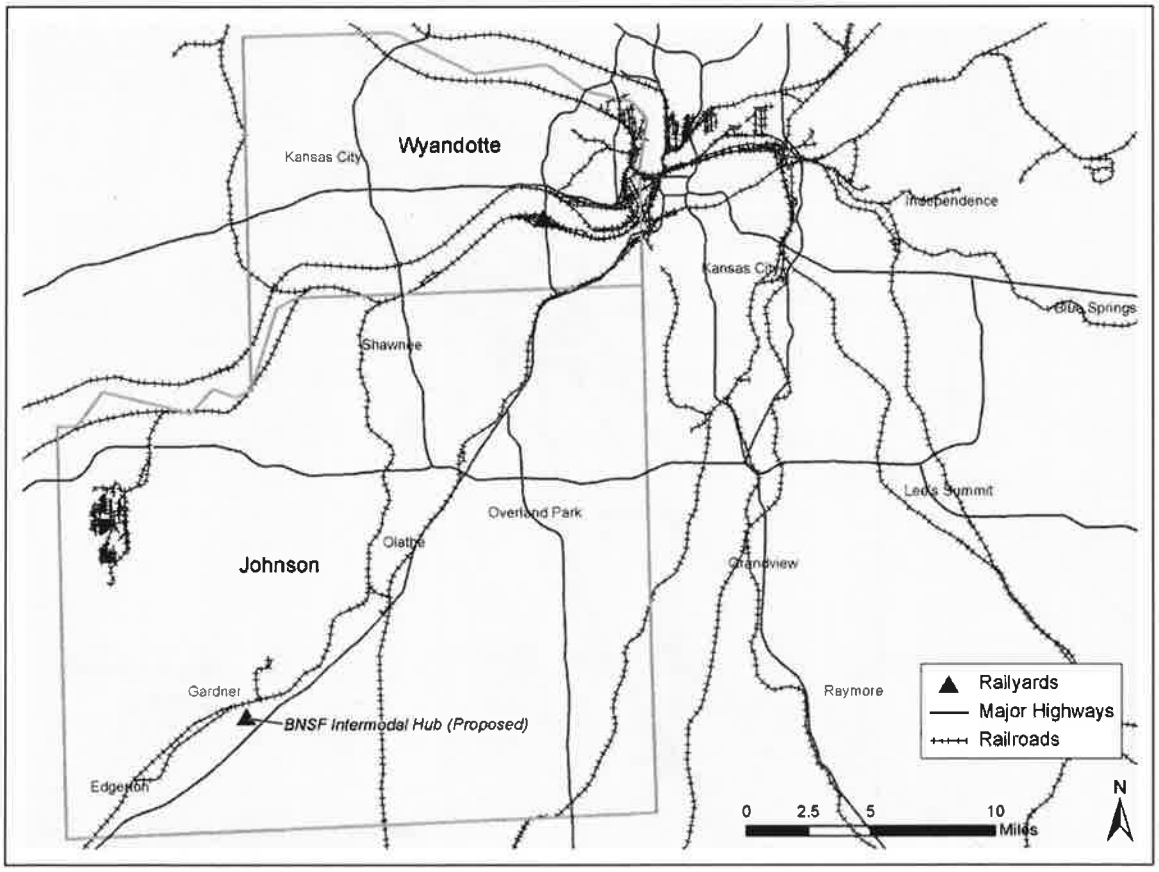
Detroit, Michigan Map



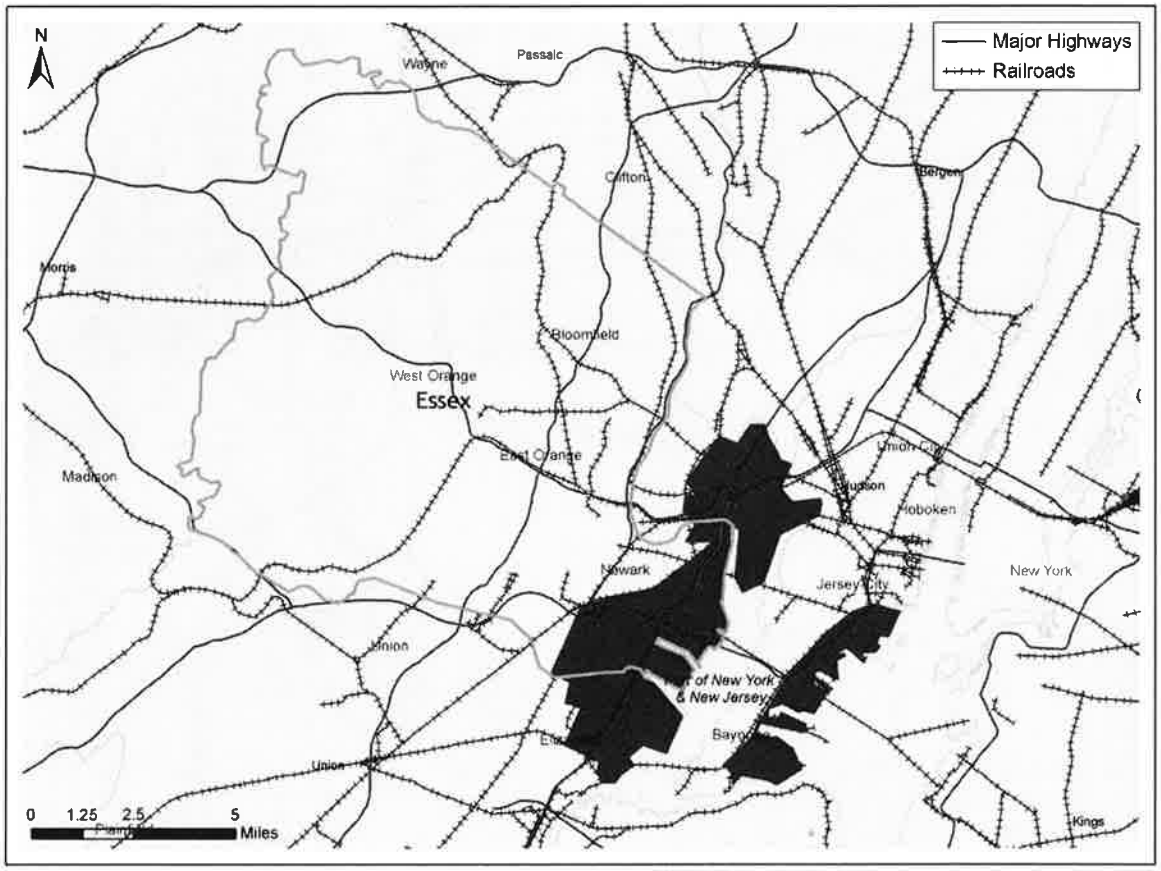
Chicago, Illinois Map



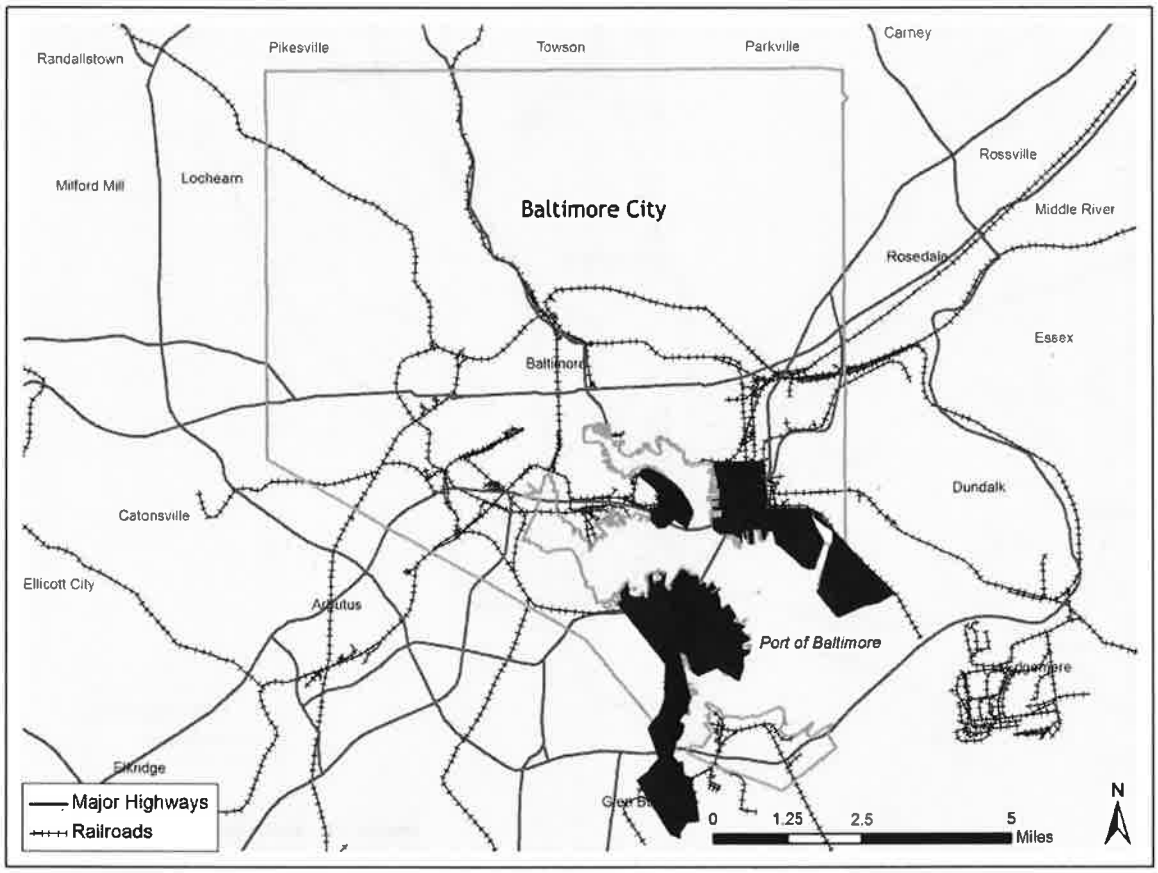
Kansas City Map



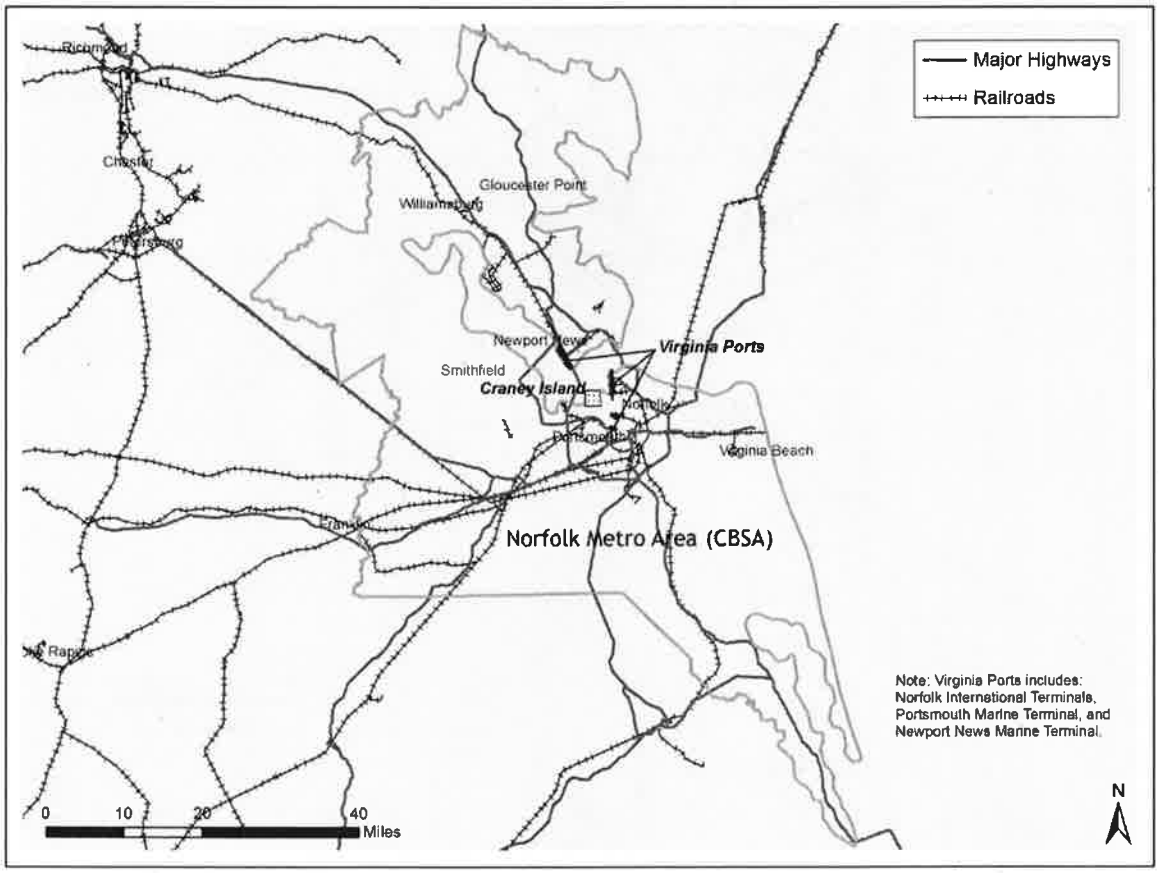
New York/New Jersey Map



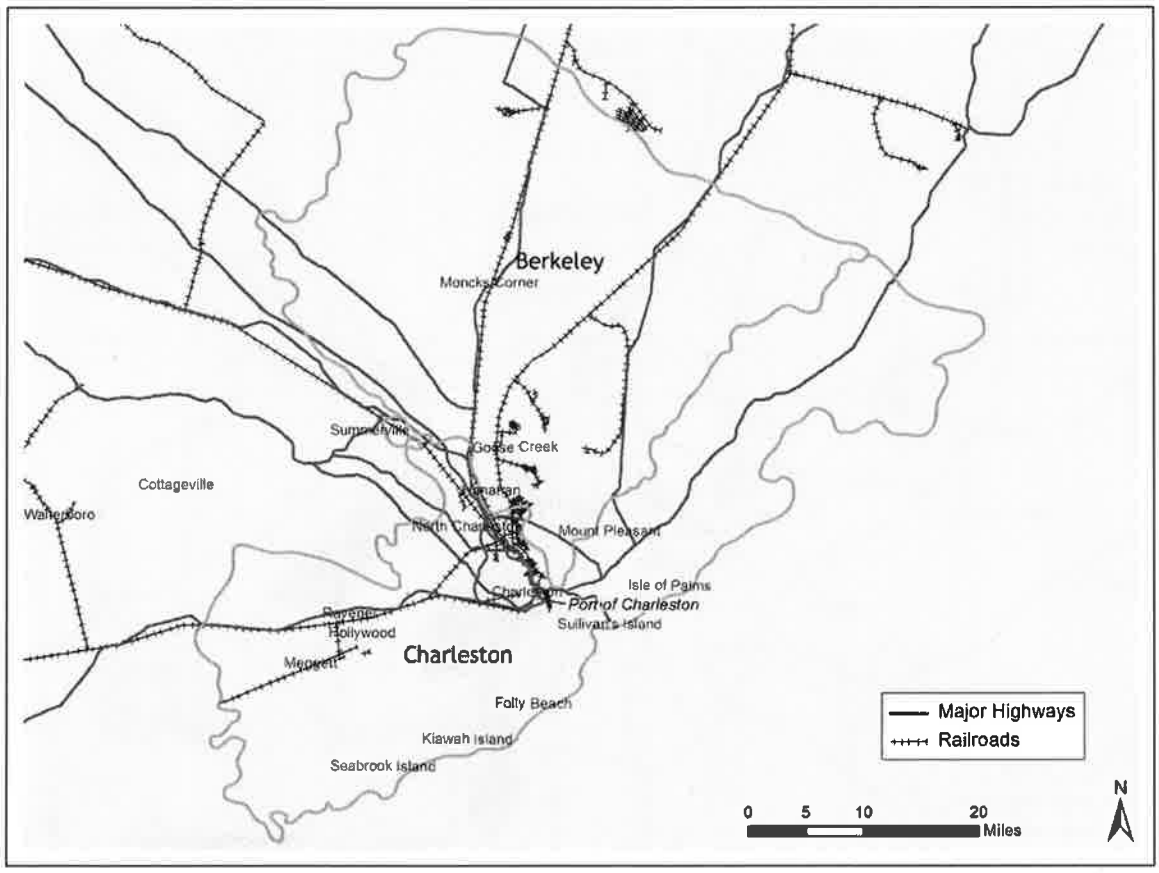
Baltimore, Maryland Map



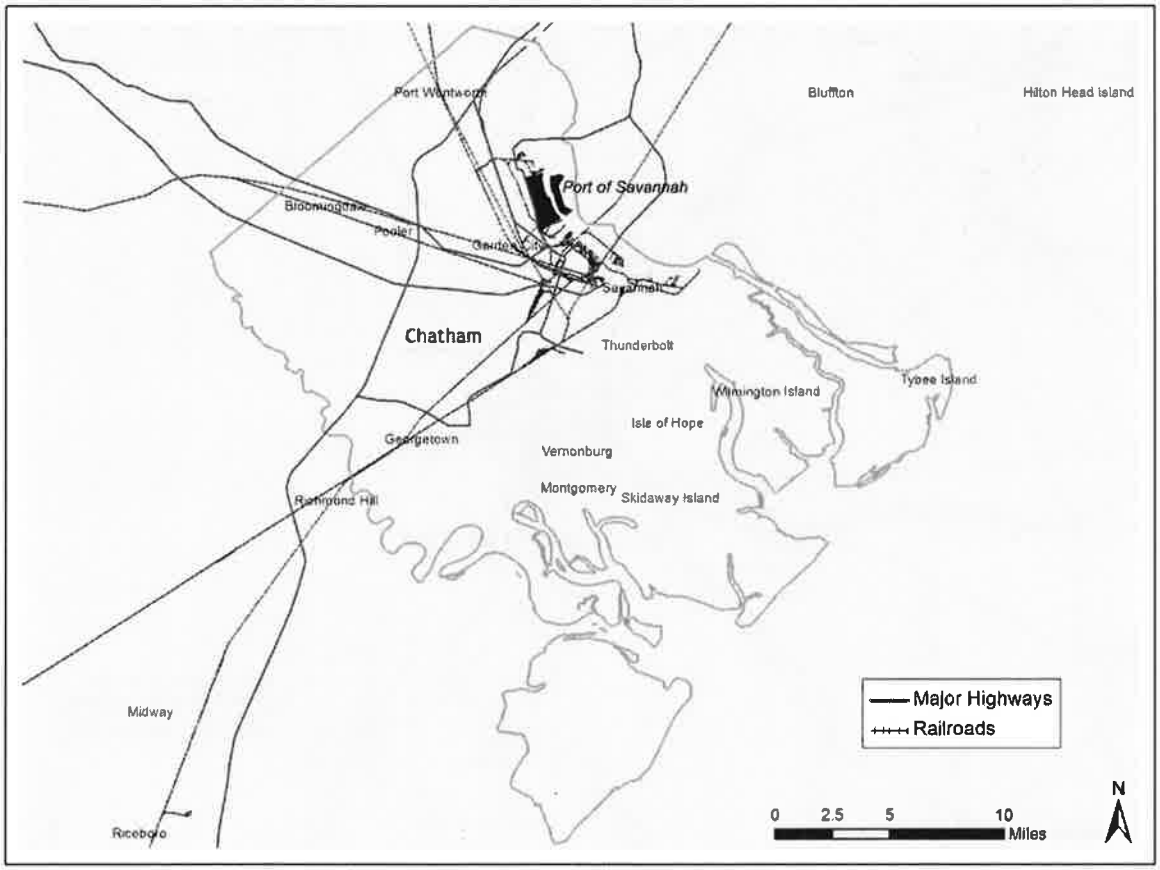
Norfolk/Hampton Roads and Inland Virginia Map



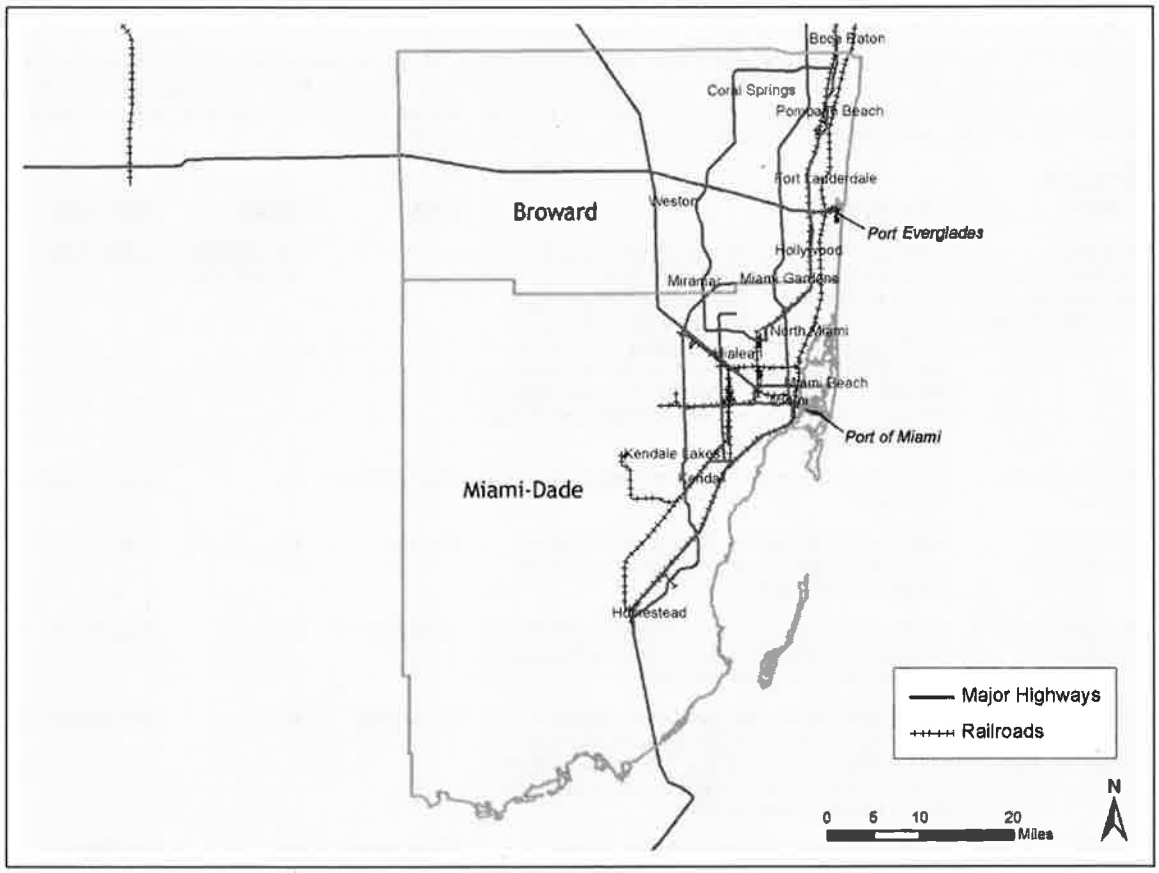
Charleston, South Carolina Map



Savannah, Georgia Map



Miami/Port Everglades, Florida Map



Jacksonville, Florida Map

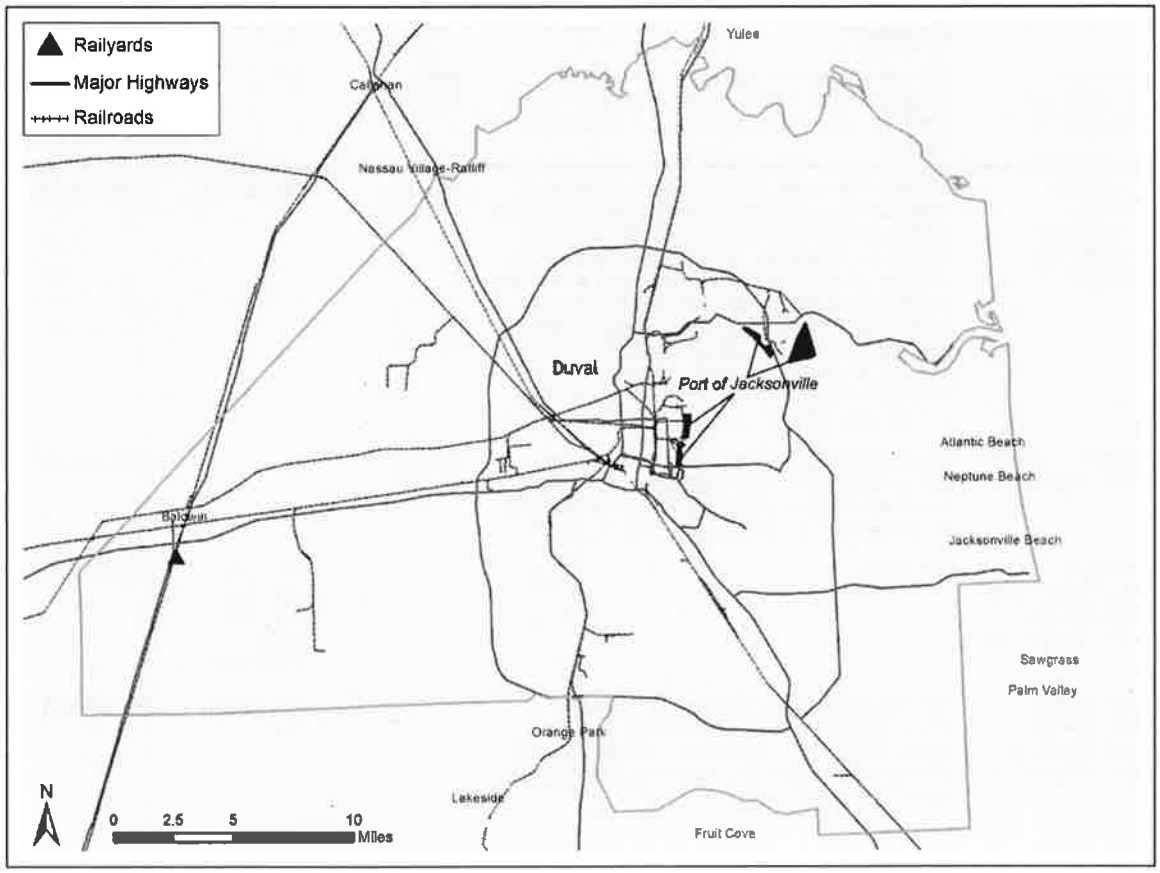


Table 1. Ports and Freight Transportation Related Transportation Investment Generating Economic Recovery (TIGER) Grants

Chart adapted from U.S. Department of Transportation/Transportation for America Summary of Grants. The chart includes the categories for: 1) Freight Rail; 2) Ports; 3) Ports Freight Rail. Select projects from "Road Projects" are also included because of their primary focus on freight transport. Projects in other categories such as "Bridge Repair/Replacement" are not listed here but may also facilitate freight transportation. See www.t4america.org for a list of all projects.

	Project Type	Project Title	Description	City	State	Amount	Round
Northeast	Freight Rail	National Gateway Freight Rail Corridor (OH, PA, WV, MD)	A package of rail infrastructure and intermodal terminal projects will enhance freight transportation service options along three major freight rail corridors owned and operated by CSX through the Midwest and along the Atlantic coast, allowing trains to carry double- stacked containers, increasing freight capacity and making the corridor more marketable to major East Coast ports and shippers.		MD (also OH, PA, and WV)	$98,000,000	TIGER I
	Freight Rail	Fast Track New Bedford	Reconstruct four inadequate and dangerous freight rail bridges.	New Bedford	MA	$20,000,000	TIGER I
	Ports	Revitalizing Maine's Ports	Help the Port of Portland to upgrade the wharf and upland storage facility at the International Marine Terminal Facility.	Portland	ME	$14,000,000	TIGER I
	Freight	Aroostook Rail Preservation	Restore the rail routes serving Northern Maine and re-establish a vital rail link to meet regional and national goods movement needs.	Aroostook	ME	$10,546,436	TIGER II
	Ports	Port of Providence: Electric Cranes	Expand and upgrade the Port of Providence in Rhode Island, replacing two aged diesel cranes, one of which is currently non-functional, with new electric, barge-based cranes that will enable the Port to handle container traffic.	Providence	RI	$10,500,000	TIGER II
	Roads, Freight Rail	Central Pennsylvania Rail and Road Expansion	Improve the safety and efficiency of freight movement for Pennsylvania's publicly owned short line railway system, making system-wide improvement for the 200 miles of track owned by the SEDA-Council of Governments Joint Rail Authority.	Pennsylvania	PA	$10,000,000	TIGER II
	Rail/Roads	Access to Quonset Wind Energy Project	Improve freight transportation at the port, achieve a state of good repair, extend the useful life of former military assets and increase port capacity.	North Kingston	RI	$22,300,000	TIGER I
Central	Freight Rail	Crescent Corridor Intermodal Freight Rail Project	A major intermodal freight program to support development of Norfolk Southern's rail intermodal route from the Gulf Coast to the Mid-Atlantic. Once fully-developed, the Crescent Corridor will improve domestic rail intermodal service between the Northeast and Southeast and connect this 2,500-mile network of existing rail lines with regional intermodal freight distribution centers and will strengthen domestic and international freight distribution in the Southeast, Gulf Coast and Mid-Atlantic markets.	Memphis	TN, also AL	$105,000,000	TIGER I
	Freight Rail	CREATE Program Rail Projects	A package of 78 projects that address freight rail congestion in the Chicago area, a nationally significant freight bottleneck adversely affecting the delivery of goods throughout the country.	Chicago	IL	$100,000,000	TIGER I
	Freight Rail	National Gateway Freight Rail Corridor (OH, PA, WV, MD)	See above		OH; also (PA, WV, MD). SEE ALSO NORTHEAST	$98,000,000	TIGER I
	Freight Rail	Appalachian Regional Short Line Rail Project (KY, WV and TN)	This will rehabilitate hundreds of miles on five unconnected short-line railroads in three states, all operated by a single holding company. Investments include rail, crossties, grade crossing, bridge and tunnel work.		TN, KY and WV	$17,551,028	TIGER I

Project Type	Project Title	Description	City	State	Amount	Round
Freight Rail	Reconstruct Mitchell-Rapid City Railroad	The reconstructed rail line will increase the capacity and efficiency of the line principally used for transportation of agricultural commodities.	Mitchell	SD	$16,000,000	TIGER II
Freight Rail	Minot Grade Separation	This will construct a grade separated pass over Burlington Northern Santa Fe's mainline and service tracks and approach roadways on 55th Street NE.	Minot	ND	$14,130,000	TIGER II
Freight	Great Plains Freight Rail (Kansas and Oklahoma)	This project will construct yard, shop, and rail line improvements and relocate the South Kansas and Oklahoma Railroad hub from an urban to rural area.	Coffeyville	KS	$10,230,597	TIGER II
Freight Rail, Bridge Repair/ Replace-ment	Staples North/ South Corridor	This will construct a new crossing over the Burlington Northern Santa Fe (BNSF) Railroad and U.S. Highway 10 in Staples, Minnesota, where a pair of grade crossings receive an average of 52 trains per day, meaning Staples residents sit in congestion for hours each day waiting for trains.	Staples	MN	$7,650,000	TIGER II
Freight	The Southwestern Illinois Intermodal Freight Transpor-tation Hub	Funds construction of a public harbor on the Mississippi River which will be used for barge loading and unloading. The project will allow the Tri-City Regional Port District to expand barge, rail and truck transportation systems in the region and allow shippers, including Midwest agricultural shippers, to move goods down the Mississippi River from Illinois to the Gulf of Mexico without the use of a lock.	Granite City	IL	$6,000,000	TIGER I
Freight Rail	Freight Rail Reactivation & Rehab	Rehabilitates the 7.5 mile rail line from the Chadron East Yards to the west end of Dakota Junction.	Chadron	NE	$4,923,509	TIGER II
Freight Rail	Crescent Corridor Intermodal Freight Rail Project	See above.	Birmingham	AL (also IN)	$105,000,000	TIGER I
Freight	Tower 55 Freight Rail Improvements	Adds an additional north-south track and install new signals and a new interlocking system to improve the flow of train traffic through Tower 55, a major rail and traffic bottleneck in downtown Fort Worth, TX, where Union Pacific and Burlington Northern Santa Fe railroad lines cross.	Fort Worth	TX	$34,000,000	TIGER II
Ports, Freight Rail	Port of Miami Rail Access	Helps establish intermodal container rail service to the Port of Miami by building an intermodal yard and making necessary rail and bridge improvements.	Miami	FL	$22,767,000	TIGER II
Ports, Freight Rail	Port of Gulfport Rail Improvements	Upgrades 76.5 miles of rail so that double-stack trains will be able to run at 49 mph instead of the current 10 mph speed for just single-stacked trains, massively increasing capacity and speed for freight. This connects the Port of Gulfport to Chicago and Canada as well as to New Orleans and the East Coast	Gulfport	MS	$20,000,000	TIGER I
Ports	Northwest Tennessee Port (Lake County)	Tiger II dollars will be used to build a port and harbor facility on the Mississippi River, at Cates Landing in Tennessee. Dock facilities will be constructed and additional, necessary, on-site improvements will be made to create a connection between barge traffic at the port and truck freight movement.	Dyersburg	TN	$13,000,000	TIGER II

Project Type	Project Title	Description	City	State	Amount	Round
Roads	Bella Vista Bypass (AR and MO)	The project will create a 19-mile, access-controlled, 4-lane, partially tolled road around the City of Bella Vista in Northwest Arkansas and Southwest Missouri. The bypass will complete a link for I-49, connecting the Port of New Orleans with a number of interstates and improving the flow of goods to the Great Lakes and Canada. Major corporations and universities are located along this fast growing corridor. (TIFIA Loan Grant)	Bella Vista	AR	$10,000,000	TIGER I
Roads	Port Manatee Marine Highway	A 32 acre container terminal will be constructed adjacent to the existing 1,000 foot berth, expanding the Port's cargo storage capacity both for the Marine Highway operation and for other tenants.	Bradenton	FL	$9,000,000	TIGER II
Freight Rail	Alameda Corridor East: Colton Crossing	Eliminates the mainline at-grade rail crossing of the Union Pacific Railroad and the BNSF Railway at Colton in San Bernardino County. This crossing is on the major east-west corridor for each of the two carriers, and at its peak in 2006 the crossing handled 129 trains a day. The trains that wait and queue behind the crossing create a major choke point for traffic moving to and from Southern California.	Colton	CA	$33,800,000	TIGER I
Roads	US-491 Safety Improvements	A collaborative effort of three regional ports in California to develop and use a marine highway system as an alternative to existing truck and rail infrastructure. The Port of Oakland along with the inland Ports of Stockton and West Sacramento have formed a partnership to provide freight service via barge, primarily for consumer goods moving by ocean vessel and agricultural products grown in Central California.	McKinley County	NM	$31,000,000	TIGER I
Ports	California Green Trade Corridor/ Marine Highway Project	This is a collaborative effort of three regional ports in California to develop and use a marine highway system as an alternative to existing truck and rail infrastructure. The Port of Oakland along with the inland Ports of Stockton and West Sacramento have formed a partnership to provide freight service via barge, primarily for consumer goods moving by ocean vessel and agricultural products grown in Central California. This will improve the quality of life for Northern Californians by reducing greenhouse gas emissions and air pollutants and relieving congestion and wear-and-tear on Northern and Central California's highways, helping to reduce round-trip and overall truck miles traveled to and from distribution centers and port facilities in the area, with corresponding savings in fuel costs achieved by shipping goods by barge rather than exclusively by truck.	Stockton	CA	$30,000,000	TIGER I
Ports	Reconstruction of Pier 29 in Honolulu Harbor	In 2008, the Pier 29 container yard at the Honolulu Harbor suffered structural failures, displacing the international carrier that used it. These funds will reconstruct Pier 29, adding approximately 12 acres of upgraded cargo yard while also increasing efficiency and safety in Honolulu Harbor. Reconstructing Pier 29 will reduce truck traffic on busy and congested roadways in downtown Honolulu near Piers 1 and 2 by moving much of the traffic west towards the reconstructed Pier 29. Since Pier 29 is closer to Nimitz Highway and the primary inter-modal highway routes, reconstructing Pier 29 helps reduce fuel consumption and greenhouse emissions from cargo movements at Piers 1 and 2 in the downtown Honolulu area.	Honolulu	HI	$24,500,000	TIGER I

Project Type	Project Title	Description	City	State	Amount	Round
Roads	Otay Mesa Port-of-Entry I-805/SR-905 Interchange	This is a critical interchange linking I-805 in San Diego to the new SR-905 highway now under construction. Once complete, the project will provide a direct 6-lane highway link to the Otay Mesa Port of Entry at the Mexican border, with reduced grades and improved shoulders. Otay Mesa is the largest freight border crossing between California and Mexico. International freight will use the new highway instead of using heavily congested Otay Mesa Road. Completing this Interstate connection is a high priority for reducing congestion at the border on a major international freight route. The project will improve efficiency and reliability in the movement of goods and services and will reduce border wait times.	San Diego	CA	$20,200,000	TIGER I
Port/ Freight	Port of Los Angeles: West Basin Railyard	This project will construct an intermodal railyard, which includes staging and storage tracks connecting on-dock railyards with the Alameda Corridor, and includes a railyard for a short-line railroad serving major carriers and both major ports. The project will remove two at-grade rail-highway crossings, relieving congestion. This project mitigates disruptions to commercial activity that cost an estimated $9.1 billion per year, improves safety by reducing truck trips on I-710, which has the highest accident rate in California, and by removing two at-grade rail-roadway crossings between a residential community and waterfront area, and creates nearly 2,000 construction jobs in an economically distressed area.	Los Angeles	CA	$16,000,000	TIGER II
Freight Rail	Coos Bay Rail Line (Coos, Douglas, Lane Counties)	This will rehabilitate the track structure of the 133-mile Coos Bay Rail Link, which closed in 2007 as a result of deferred maintenance, including replacement of worn-out rails, fasteners, and wood ties; re-establishment of proper surface, line, and dress of the track; drainage improvement of the track bed; and enhancing the ballast bed. It will reduce truck shipments for former customers of the line, customers who currently face an average 330 mile truck dray, and puts the freight back on rail, decreasing costs by 20 percent.	Coos Bay	OR	$13,573,133	TIGER II
Freight	West Vancouver Freight Access	The West Vancouver Freight Access project will construct a new rail access route to alleviate rail traffic congestion at the Port of Vancouver. The project will relocate facilities within the port to provide a new entryway into port terminals. This will create an estimated 400 permanent new jobs in two to five years, and up to 1,836 construction jobs per year, in a county with the highest unemployment rate (13.3 percent as of July 2010) in the state of Washington and support the port's commitment to expand its use of rail from 72 percent to 85 percent.	West Vancouver	WA	$10,000,000	TIGER II
Ports	Auke Bay Loading Facility	This will fund Phase II of the Auke Bay Loading Facility in Juneau, which includes an additional half-acre of storage, lighting, security gate and fences; a freighter loading facility and ramp; and a fisheries dock, improving freight transfer activities for Alaska communities that use barge and landing craft as primary marine services. This will help deliver government programs to remote communities and contribute to lower costs of living and improved living standards. The facility is important to serving the needs of the fishing community by reducing the need for long and dangerous voyages around Douglas Island to reach Juneau, and provides almost direct access to Juneau airport for fresh seafood exports.	Juneau	AK	$3,640,000	TIGER I

Endnotes

1 Throughout this report, we use the terms "goods movement" and "freight transportation" interchangeably to mean the movement of goods (usually in cargo containers) across oceans into ports and along supply chains to rail facilities, warehouses, and retail stores. "Goods movement" is the term most often used in California; "freight transportation" is often used by U.S. transportation agencies; and "freight transport" is a phrase commonly used in Europe. "The logistics industry" refers to managing the flow of goods, e.g., from Asia to Chicago. The "supply chain" is the network of those involved in moving a product from a supplier to a customer. A recent report by the U.S. EPA provides the following definition: "*Goods movement refers to the distribution of freight (including raw materials, parts, and finished consumer products) by all modes of transportation including marine, air, rail, and truck. Goods movement facilities, also called freight facilities, include seaports, airports, and land ports of entry (border crossings), rail yard and rail lines, highways and high truck traffic roads and warehouse and distribution centers.*" Definition from: National Environmental Justice Advisory Council. Reducing Air Emissions Associated With Goods Movement: Working Towards Environmental Justice. U.S. EPA, Nov. 2009. (Accessed February 25, 2011, at http://www.epa.gov/compliance/ej/resources/publications/nejac/2009-goods-movement.pdf)

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To download the report, please see any of the following web sites:

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Acute Health Effects of the Enbridge Oil Spill

November 2010
(Minor revisions 12/20/2010)

Michigan Department of Community Health

MDCH

CALHOUN COUNTY MICHIGAN
ORGANIZED 1833

Acute Health Effects of the Enbridge Oil Spill

November 2010
(Minor revisions December 20, 2010)

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Acute health effects of the Enbridge Oil Spill

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Executive Summary

On Monday July 26, 2010, Enbridge Energy Partners, L.P., reported the burst of a 30-inch pipeline near Marshall, Michigan, in Calhoun County. In response to concerns about acute health effects from exposure to spilled oil in this major disaster, state and local public health in Michigan quickly set up a multi-faceted public health surveillance system that included health care provider reporting, community surveys, calls from the public to the Poison Control Center, and analysis of data submitted to the state's syndromic surveillance system. The surveillance system received 147 health care provider reports on 145 patients, identified 320 (58%) of 550 individuals with adverse health effects from four community surveys along the impacted waterways, identified one small worksite symptomatic employees, and tracked 41 calls that were placed to the poison center by the public. Headache, nausea, and respiratory symptoms were the predominant symptoms reported by exposed individuals in all reporting systems. These symptoms are consistent with the published literature regarding potential health effects associated with acute exposure to crude oil.

I. Background

On Monday July 26, 2010, Enbridge Energy Partners, L.P., reported the burst of a 30-inch pipeline near Marshall, Michigan, in Calhoun County. The spill started at least a day earlier based on 911 calls and other reports of strong odors starting July 25. Approximately 800,000 gallons of crude oil spilled into the Talmadge Creek, a waterway that feeds the Kalamazoo River. The contamination ultimately affected 25 miles of the creek and river. While the greatest impact was in Calhoun County, the spill also affected an area of Kalamazoo County encompassing five miles of the river downstream from the border of Calhoun County to a dam just upstream from the city of Kalamazoo (See map in the appendix). The U.S. Environmental Protection Agency (EPA), Calhoun County Public Health Department (CCPHD), Calhoun County Emergency Management, the Michigan Department of Community Health (MDCH) and many other agencies and organizations quickly became involved with public health and environmental response to this massive spill.

Beginning July 26, when the spill was reported to authorities, individuals near Talmadge Creek and the Kalamazoo River began complaining of strong, noxious odors and associated health symptoms in calls to CCPHD and the Michigan PCC. Subsequently, once it had been established, citizen concerns and complaints were routed to a phone hotline developed by Enbridge. Callers reported respiratory, gastrointestinal, and neurological symptoms, predominantly headache and nausea.

To adequately characterize the impact of the oil spill on the public's health, CCPHD, MDCH, and the Kalamazoo County Health and Community Services Department (KCHCS) developed and implemented a public health surveillance system to collect data on individuals with adverse health outcomes secondary to exposure to spilled oil and its vapors. The goal of this surveillance was to describe the magnitude and distribution of human health impacts due to exposure to the spilled oil, so that decision-makers could make informed decisions about actions needed to protect the public.

The surveillance system included four components: (1) active solicitation of health care provider reports, under legal authority of the Public Health Code, and (2) door-to-door health surveys in selected communities self-identified as particularly impacted by the spill, (3) monitoring daily counts of self-reported illnesses based on calls to the PCC, and (4) utilization of MDCH's syndromic surveillance system.

In order to protect personal confidential medical information, MDCH obtained a "Medical Research Designation".[1] This designation legally protected individual identifying information from disclosure by the participating public health authorities to other parties, including those situations in which the information

could be requested under the Michigan Freedom of Information Act or by subpoena.

This report describes the methods and results of the public health surveillance system established to measure and monitor health impacts from the Enbridge oil spill. Information about environmental sampling, clean-up efforts and other aspects of the spill response are available elsewhere.[2]

II. Methods and Results

A. Health care provider reporting

Methods

Initially, contacts were made at the two hospitals in the area, and they were asked to provide a daily count of the number of patients seen in the Emergency Department (ED) or admitted with oil exposure-related complaints. Then, on August 5, the CCPHD and the KCHHS sent out "blast faxes" to all health care providers in their respective counties requesting that clinicians and healthcare facilities formally report any patients seen due to illness or symptoms associated with oil spill exposure. Providers were advised that this reporting is required under the Michigan Public Health Code (R 325.71-75), and they were provided reporting information and forms.[3] To gather data on patients who were seen at the local ED prior to establishment of this healthcare reporting system, medical records of patients identified as exposed to the oil or its vapors were abstracted by the MDCH medical epidemiologist.

The Michigan PCC was authorized as a legal agent of the state to receive the reports from health care providers for the purposes of this investigation. This allowed for 24/7 reporting, and allowed for PCC medical toxicologists to provide consultation to health care providers regarding oil spill-related patient diagnosis or treatment. Patient information collected included name, contact information and demographics, medical encounter date, clinical effects, laboratory test results, diagnosis, treatment, and contact information for the reporting provider.

Reported information was entered into Toxicall®, the electronic case management system used by the Michigan PCC. Each case was given a "medical outcome" classification based on information about reported clinical effects as follows: no effect (no symptoms due to exposure); minor effect (some minimally troublesome symptoms); moderate effect (more pronounced, prolonged symptoms); major effect (symptoms that are life-threatening or cause significant disability or disfigurement); death; not followed, judged as nontoxic exposure (clinical effects not expected); not followed, minimal clinical effects possible (no more than minor effect possible); unable to follow, judged as a potentially toxic exposure; unrelated effect, the exposure was probably not responsible for the effect(s); or, confirmed non-exposure.

Daily summary reports were provided by the PCC to MDCH, CCPHD, and KCHHS on numbers of reports and severity of illness (i.e. "medical outcome"). A spreadsheet of all case information was provided to MDCH for data analysis. Analysis was performed using SAS® version 9.2 (SAS Institute, Cary, NC).[4]

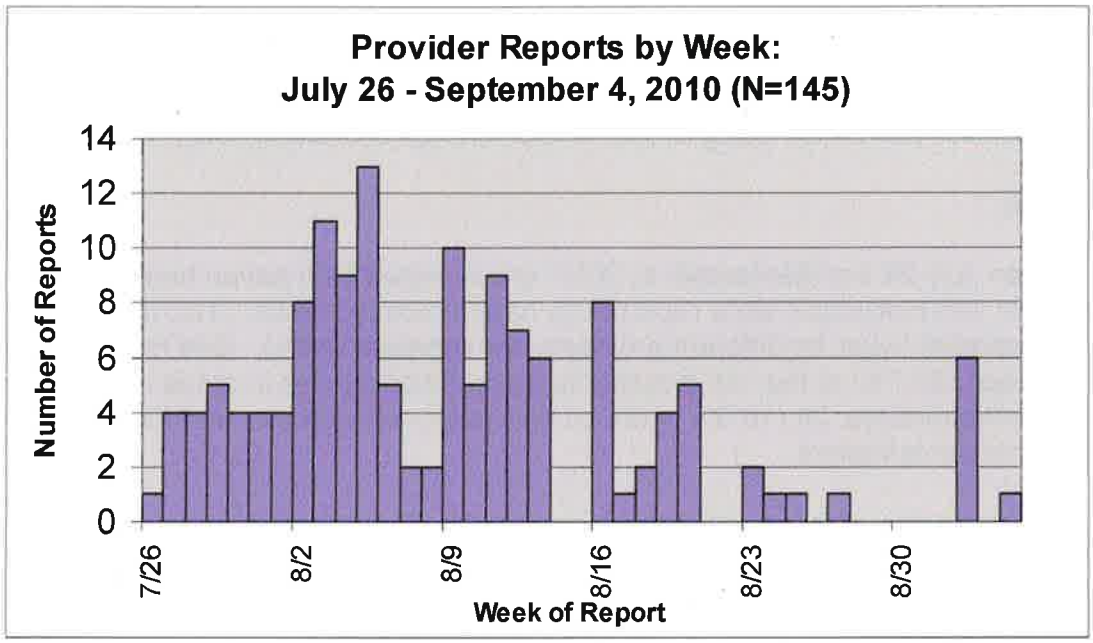
Results

Between July 26 and September 4, 2010, one hundred forty-seven health care visits for 145 individuals were reported by health care providers. (Two individuals were reported twice, by different providers, for separate visits.) One hundred seventeen (80.7%) of the 145 individuals lived and/or worked in areas near the affected waterways, 24 (16.5%) were oil-spill response workers, and four (2.7%) were transients/visitors.

The average age of these 145 individuals was 38. There were slightly more females (77/53.1%) than males (68/46.9%) reported. Adults age 18 to 64 predominated (100/69%), with the remainder being children under age 18 (36/24.8%), and a small number of adults over age 65 (9/6.2%).

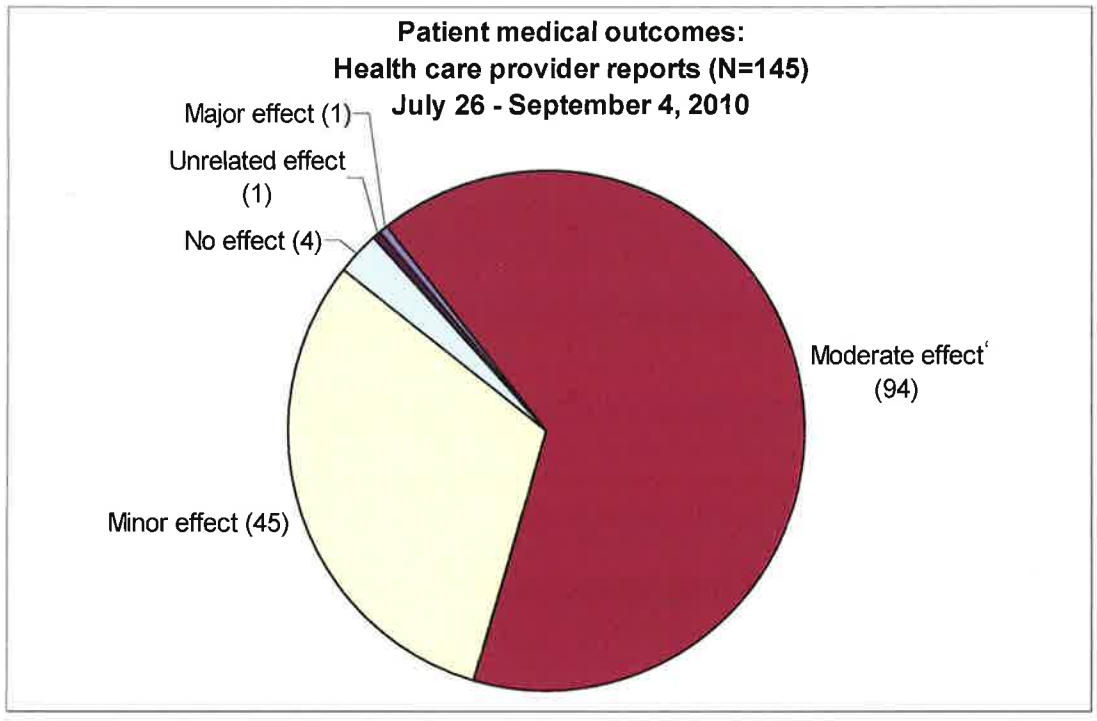
The date of the reported visit to the health care provider is shown in **Figure 1**. (The two individuals reported twice are counted for date of their first visit.) The frequency of reported health care provider visits peaked in the second week after the spill, coinciding with the week providers were notified of the new reporting requirements. These visits included outpatient (N=77; 53%), hospital emergency department (N=64; 44%), hospital inpatient (N=1; 0.6%), and 3 (2%) where type of facility was unknown.

Figure 1



Approximately one-third (31%) of the medical outcomes of these individuals were classified as minor and two-thirds (64.8%) as moderate. There were no deaths. **(Figure 2**) The one individual with medical outcome classified as "major" had significant exposure and had 8 reported clinical effects. Those with a medical outcome of "moderate" had on average 3.7 clinical effects whereas those classified as "minor" had 2.4 clinical effects.

Figure 2



Four (2.8%) of the reported individuals had no clinical effects. The remainder had from one to more than six clinical effects each (**Table 1**).

Table 1

Number of Clinical Effects in Health Care Provider Visits		
	Total	
	N	%
Number of Clinical Effects		
0 symptoms	4	2.8%
1 symptom	21	14.5%
2 symptoms	38	26.2%
3 symptoms	26	17.9%
4-5 symptoms	37	25.5%
6+ symptoms	19	13.1%

Neurological effects were reported most frequently (94/ 64.8%), with headache being the predominant of all neurological effects reported 83 (57.2%). Eighty-six individuals (59.3%) had at least one gastrointestinal clinical effect, with nausea predominating, and 68 (46.9%) had with at least one respiratory clinical effect with cough and choke predominating (**Table 2**).

Table 2

Frequency of Clinical Effect Categories in Health Care Provider Visits		
	Total	
	N	%
Clinical Effect Category		
Cardiovascular	11	7.6
Dermal	9	6.2
Gastrointestinal	86	59.3
Nausea	*57*	*39.3*
Neurological	94	64.8
Headache	*83*	*57.2*
Ocular	23	15.9
Renal	1	0.7
Respiratory	68	46.9
Cough/Choke	*47*	*32.4*
Other	41	28.3

B. Community and Workplace Surveys

Methods: Four communities along the Talmadge Creek and Kalamazoo River and one small workplace were identified (from calls to the toll-free number and the CCPHD) as having multiple reports of adverse health effects and concerns about oil spill impacts.

A door-to-door health survey was conducted by MDCH and the CCPHD in each community. The community survey obtained information on whether the household had, or were planning to, relocate because of the spill; observations about the intensity and duration of the odor since July 25; and, for all members of the household, information about chronic/pre-existing health conditions that made them sensitive to fumes or odors. They were also asked about new or exacerbated health symptoms after the spill event. After the first survey, a question was added to assess whether those who had symptoms had seen a physician for their symptoms. For the most part, answers were provided by the person answering the door for all household members. Answers were provided in an open-ended format. Where no one was home, information was left at the door; in the second, third, and fourth communities, including a fact sheet from EPA on the oil spill and a cover letter that invited someone in the household to call a toll-free number at MDCH to answer the survey questions by telephone. In order to have an approximate measure of socio-economic status for each of these communities, a local realtor was asked to provide his estimate of the range of home prices that could be expected in each community.

The first health survey was conducted on August 6, 11 days after the spill was reported, in a neighborhood approximately 14 miles downstream from the spill origin and immediately adjacent to an area of wetland fed by the Kalamazoo River. Previously, on August 2, the CCPHD had visited the neighborhood to assess the need for temporary relocation of individuals concerned about the odors and their health, and to give information about how Enbridge would cover the costs of that relocation. However, information about health symptoms was not requested at that initial visit. Home prices in the neighborhood, which is referred to as "Neighborhood" in the tables, are estimated to range from $500 to $15,000.

The second community survey was conducted 16 days after the spill in a subdivision approximately two miles downstream. Home prices in the "Subdivision" are estimated to range from $120,000 to $325,000.

The third community, referred to as "Spill Site" in the tables, was surveyed in two parts, 22 and 24 days after the spill. This community included the homes surrounding the immediate area on the Creek where the pipeline burst. It was the only community where a voluntary evacuation notice had been issued, due to air sampling indicating elevated levels of benzene– a potential concern for long-term health. Each of these two surveys took place within 24 hours after the

evacuation notice was lifted for that area. A environmental contractor accompanied the survey team and offered air monitoring outside and inside homes to each of the interviewees, using a real-time monitoring instrument. Home values in this community are estimated to range from $75,000 to $350,000.

The fourth survey occurred 23 days after the spill in a small village of approximately 80 homes, situated directly on the river about five miles downstream from the spill's origin. Home values in the "Village" are estimated to range from $10,000 to $125,000.

For comparison purposes, a door-to-door survey was conducted 25 days after the spill in a community approximately fifteen miles stream upstream of the spill, in order to obtain information on the occurrence of health symptoms in the previous four weeks. The six neighborhoods surveyed in this community were on the Kalamazoo River; they were similar to the exposed communities in demographics and the range of home prices, encompassed homes valued from $5,000 to $225,000.

All 12 workers at the small workplace located a little less than one mile northeast of the confluence of Talmadge Creek and the Kalamazoo River were interviewed using the same open-ended format as the community surveys.

Results

Community Surveys

Table 3 shows the survey completion rates by community and in the Comparison community. Overall, 201 (59.6%) of the 337 homes visited provided information for a total of 550 household members in the exposed communities, and 51 (27.9%) of the 183 homes surveyed in the Comparison community provided information on 137 individuals. The average number of household members ranged from 2.5 to 3.1 in the exposed communities and was 2.7 in the Comparison community.

Table 3

Survey Completion by Community						
	Neighborhood	Subdivision	Spill Site	Village	Total	Comparison
Total Number of Homes Visited	78	121	55	83	337	183
Number of Homes that Completed Survey	45	75	37	44	201	51
Number of Homes that Refused Survey	0	0	0	1	1	18
Number of Homes with No One Home	33	46	18	38	135	114
Percentage of Homes Surveyed	57.7%	62.0%	67.3%	53.0%	59.6%	27.9%
Number of Individuals with Survey Information	117	233	92	108	550	137
Average Number of Individuals per Household	2.6	3.1	2.5	2.5	2.7	2.7

In terms of race/ethnicity, all communities were almost entirely white. There were some differences between communities in other demographics. The community at

the spill site was on average older, had fewer children, and was over 50% male, in contrast to the other three exposed communities and the Comparison group. Smoking prevalence, which was asked in all surveys except at the Neighborhood, was notably different, with the two communities with more expensive homes reporting much lower smoking rates in adults (Spill site: 5.1%; Subdivision: 6.0%) than the other one (Village) at 20.7%. Smoking prevalence in the Comparison community was 19.8% (**Table 4**).

Table 4

Demographics and Smoking Profile by Community						
	Neighborhood	Subdivision	Spill Site	Village	Total	Comparison
Gender (%)						
Male	47.8%	44.2%	53.3%	46.3%	46.9%	45.3%
Female	52.2%	55.8%	46.7%	53.7%	53.1%	54.7%
Average Age (yrs)	32.1	35.8	48.9	41.9	38.4	39.1
Age Distribution (%)						
0-7 yrs	13.9%	12.2%	2.2%	5.7%	9.6%	9.0%
8-17 yrs	15.6%	21.8%	13.0%	16.2%	17.9%	14.3%
18-30 yrs	20.0%	6.5%	6.5%	9.5%	9.95%	10.5%
31-50 yrs	28.7%	31.3%	16.3%	29.5%	27.85%	34.6%
51-65 yrs	18.3%	17.8%	43.5%	26.7%	24.0%	19.6%
66+ yrs	3.5%	10.4%	18.5%	12.4%	10.7%	12.0%
Missing (n)	2	3	0	3	8	4
Smoker (age 18 and older)	not asked	6.0%	5.1%	20.7%		19.8%

The percent of residents that reported symptoms according to smoking status is shown in **Table 5**. A higher proportion of non-smokers reported no symptoms (39.6%) compared to smokers (25.0%). Similarly, a higher proportion of smoker reported 1 symptom and 4+ symptoms (39.3%, 10.7%), compared to non-smokers (26.8%, 5.4%). The proportion of residents that report 2-3 symptoms was very similar between smokers and non-smokers.

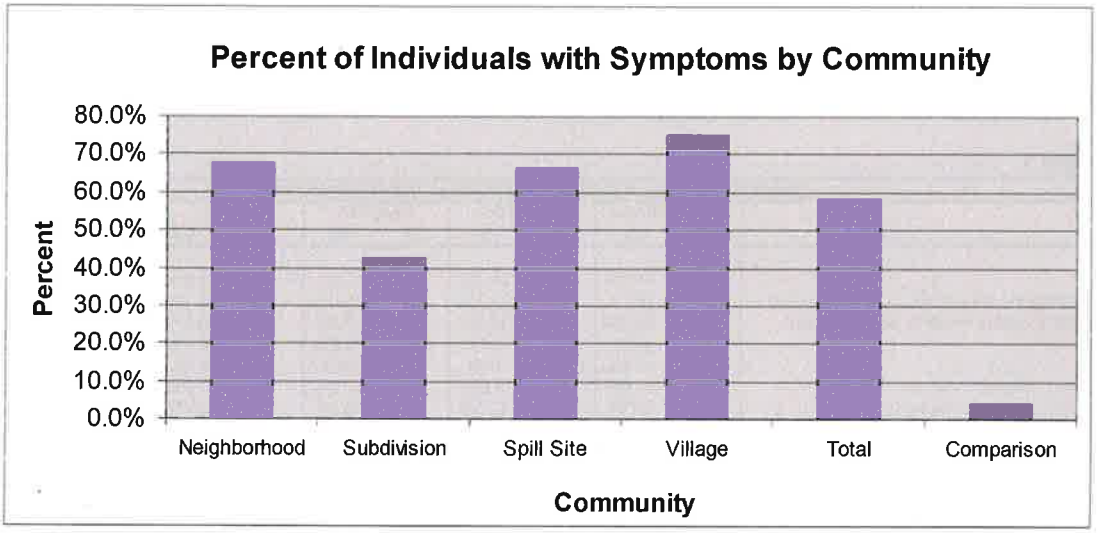
Table 5

Symptoms by Smoking Status among Adults		
	Smoker	
	Yes	No
Percent with Symptom		
0 symptoms	25.0%	39.6%
1 symptom	39.3%	26.8%
2-3 symptoms	25.0%	28.2%
4+ symptoms	10.7%	5.4%

Nearly all of the households in each of the four exposed communities reported noticing an odor since July 25, 2010 (Neighborhood: 100%, Subdivision: 97.3%, Spill Site: 97.2%, Village: 100%). In comparison, only a small minority of households in the Comparison community reported smelling an odor at any time after July 25 (15.7%).

Overall, 320 (58.2%) of the 550 individuals reported at least one new or exacerbated symptom after July 25 in contrast to 4.4% in the Comparison community. The frequency of symptoms varied by community. The Subdivision, which has homes more widely spread out from the river than any of the others, reported the lowest frequency (42.5%), and the Village had the highest (75.7%). By contrast, only 6 (4.4%) of the 131 individuals in the Comparison community reported any new or worsened symptoms in the timeframe following the spill (**Figure 3**).

Figure 3



Of the 320 individuals in the exposed communities who reported symptoms, 42.8% reported only one symptom, 44.7% reported 2-3 symptoms, and 12.5% reported 4 or more symptoms (**Table 6).** The proportion of exposed residents reporting symptoms was significantly greater than the proportion in the comparison community ($p < .0001$).

Table 6

Frequency of Symptoms by Community												
	Neighborhood		Subdivision		Spill Site		Village		Total		Comparison	
	N	%	N	%	N	%	N	%	N	%	N	%
Number/percent without any Symptoms	38	32.5%	134	57.5%	31	33.7%	27	25.0%	230	41.8%	131	95.6%
Number/percent with any Symptom	79	67.5%	99	42.5%	61	66.3%	81	75.0%	320	58.2%	6	4.4%
1 symptom	27	34.2%	49	49.5%	27	44.3%	34	42.0%	137	42.8%	5	83.3%
2-3 symptoms	37	46.8%	44	44.4%	28	45.9%	34	42.0%	143	44.7%	1	16.7%
4+ symptoms	15	19.0%	6	6.1%	6	9.8%	13	16.0%	40	12.5%	0	0.0%

Headache was the most frequently reported symptom (34.5%) in all exposed communities, ranging from 25.3% in the Subdivision to 42.6% in the Village. This was followed by respiratory symptoms (e.g., breathing difficulty, cough) at 29.6% and gastrointestinal complaints (e.g., nausea and vomiting), 21.6% (**Table 7**). In the Comparison community, only 1 resident reported headache symptoms and

respiratory symptoms, respectively. None of the comparison residents reported gastrointestinal or skin/eyes symptoms. New onset or worsened anxiety was reported by 4.9% of all exposed residents. The Subdivision reported the least amount of anxiety (1.3%) and the Neighborhood reported the most (11.1%). There were no reports of anxiety among any of the residents in the Comparison community. Data on other symptoms were also included and compiled into an 'other' category, with 24.7% of residents in the exposed communities reporting other new or worsened symptoms and only 3.6% in the Comparison community.

Overall, 12.2% of exposed residents visited a doctor for new or worsened symptoms, and doctor visits ranged from 9.8% in the Spill Site to 14.8% in the Village. While only 6 individuals in the Comparison community reported new or worsened symptoms, 4 (66.7%) saw a health care provider for these symptoms.

Table 7

Frequency of Types of Symptoms and Doctor Visits by Community												
	Neighborhood		Subdivision		Spill Site		Village		Total		Comparison	
	N	%	N	%	N	%	N	%	N	%	N	%
Symptoms												
Headache	48	41.0%	59	25.3%	37	40.2%	46	42.6%	190	34.5%	1	0.7%
Respiratory (breathing diff., cough, sore throat/nose)	34	29.1%	53	22.7%	23	25.0%	53	49.1%	163	29.6%	1	0.7%
Gastrointestinal (nausea, vomiting, stomach ache)	41	35.0%	31	13.3%	15	16.3%	32	29.6%	119	21.6%	0	0.0%
Skin/Eyes	10	8.5%	11	4.7%	11	12.0%	23	21.3%	55	10.0%	0	0.0%
Anxiety	13	11.1%	3	1.3%	7	7.6%	4	3.7%	27	4.9%	0	0.0%
Other (dizziness, fatigue, chest pain, & other)	51	43.6%	20	8.6%	25	27.2%	40	37.0%	136	24.7%	5	3.6%
Individuals with One or more Symptoms	79	67.5%	99	42.5%	61	66.3%	81	75.0%	320	58.2%	6	4.4%
Individuals who Visited a Doctor for these Symptoms	11	13.9%	10	10.1%	6	9.8%	12	14.8%	39	12.2%	4	66.7%

The prevalence of reported chronic conditions/pregnancy potentially causing increased sensitivity to odors ranged from 23.6% in the Subdivision, to 26.1% (Spill site), 40.7% (Village), and 61% (Neighborhood), including four pregnancies. The prevalence of chronic conditions in the Comparison community was 40.7%. (It should be noted that some individuals reported chronic conditions that were not likely to increase sensitivity to odor, e.g., musculoskeletal disorders.) Individuals with chronic conditions reported proportionally more symptoms than individuals without chronic conditions (**Table 8**).

Table 8

Frequency of Symptoms by Chronic Condition		
	Chronic Condition	
	Yes	No
Number of Symptoms (%)		
0 symptoms	30.9%	47.1%
1 symptom	27.0%	23.9%
2-3 symptoms	29.8%	24.2%
4+ symptoms	12.3%	4.8%

Information was available on 501 of the 550 individuals in the four communities on relocation after the spill and 169 (33.7%) of the 501 relocated. These included 50 households where everyone left and 10 households where only some members left. Thus, relocation impacted 60 (29.9%) of the 201 households surveyed. Symptoms were more prevalent overall in the 169 individuals who

relocated (71.6%) than the 332 individuals who did not (50.9%). A greater percent of those with symptoms who relocated saw a physician (11.8%) than those who did not relocate (5.1%) (**Table 9**).

Table 9

Symptoms by Relocation Status				
	Relocated (n=169)		Didn't Relocate (n=332)	
Number/percent without Symptoms	48	28.4%	163	49.1%
Number/percent with any Symptom	121	71.6%	169	50.9%
1 symptom	44	26.0%	77	23.2%
2-3 symptoms	58	34.3%	72	21.7%
4+ symptoms	19	11.2%	20	6.0%
Number/percent that Visited Doctor/ED	20	11.8%	17	5.1%

Workplace survey

At the small worksite where the symptom survey was conducted, 100%* of the workers noted the odor. Eighty-three percent noted that the worst days for odor were early in the first week following the oil release (the week of July 26). The others did not identify the worst days.

- 92% said they still smelled the odor when they were interviewed, which was three weeks after the spill.
- 33% noted that they had pre-existing chronic health conditions that made them sensitive to fumes or odors.
- 92% noted a variety of new onset or worsened symptoms after the release, including: headache (92%), respiratory symptoms (33%); dizziness (50%); gastrointestinal symptoms (33%); fatigue (33%); eye, nose, throat irritation (75%); and anxiety (42%).
- 17% noted that they were planning to see a physician for these symptoms.

C. Calls to the PCC from the public

Methods

As noted above, within a few days of the spill, individuals began making calls to the PCC with concerns about the oil spill, using the nationwide poison control toll-free number. Although the PCC toll-free number and its services were not publicized to the public in Calhoun and Kalamazoo Counties during the spill event, these calls were consistent with the understanding among the general public that poison centers are available to answer questions about chemicals, poisonings, and toxic exposures. All calls were logged according to PCC standard operating procedures. They were coded so that they could be identified

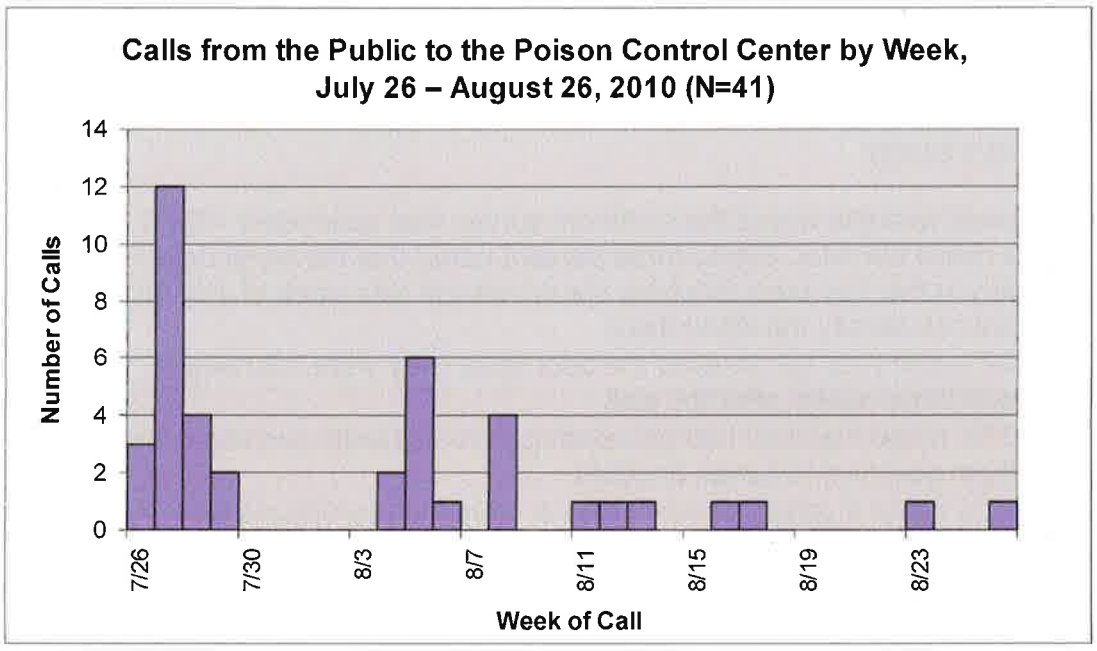
* Because of the small number of employees, numbers are not presented.

as related to the Enbridge spill event. Daily summaries of citizen calls were provided by the Michigan PCC to MDCH, in conjunction with the daily summaries of health care provider reports.

Results

Between July 26 and August 26, 41 calls were received by the PCC from individuals reporting health effects from exposure to the oil spill. No calls were received after August 26. **Figure 4** shows the number of calls by day of call. Over half (51%) of the calls (21 of 41) were received in the first week of the spill; July 27 was the day with the greatest number of calls (N= 12; 29%).

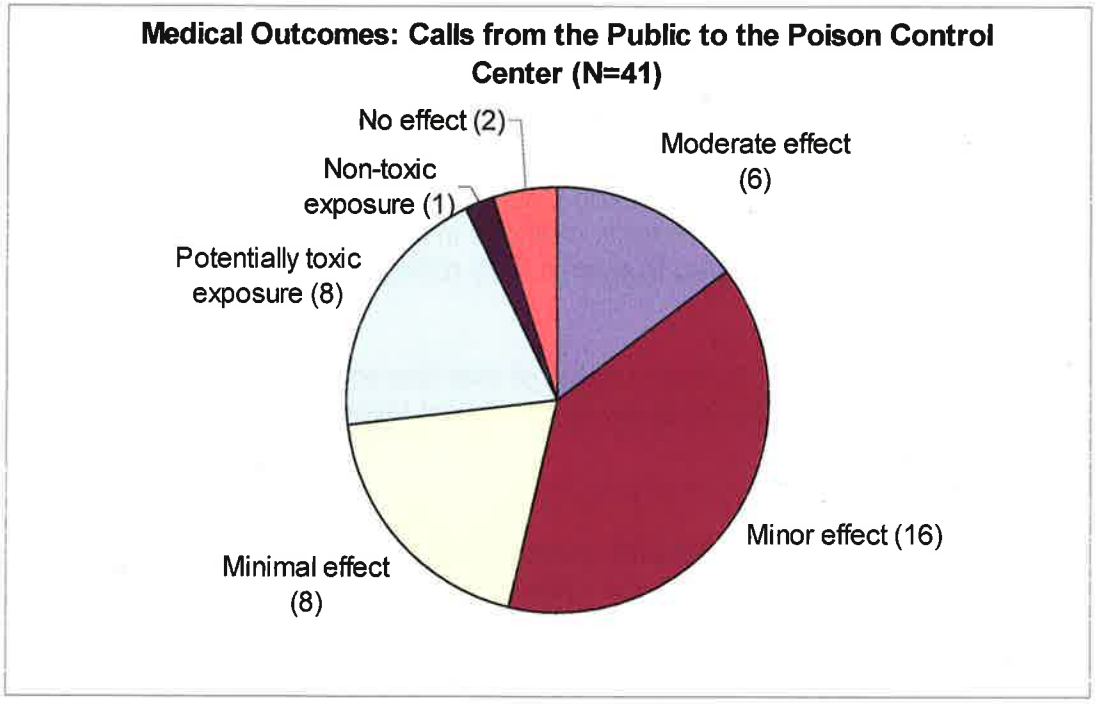
Figure 4



The ages of the individuals for whom a call was made ranged from 1 month to 60 years, with a mean of 26 years. Twenty-three (57.5%) of the 40 reports that documented gender were female.

The medical outcome classification assigned by the PCC for these calls included 39% with minor outcomes; 19.5% had possible minimal effects and 14.6% were classified with moderate effects (**Figure 5**). Nine (22%) individuals noted that they had seen a health care provider for their clinical effects, but no health care provider reports were received about these individuals.

Figure 5



D. Syndromic surveillance

Methods

The MDCH maintains a syndromic surveillance system designed to facilitate early and rapid detection and response to outbreaks that may be the result of bioterrorism, natural and/or emerging infectious disease, or other public health threats and emergencies. Real-time detection of significant increases in patients presenting with similar symptoms at designated Michigan hospital EDs is done through the use of statistical algorithms; these are applied to data obtained from hourly electronic searches through patient "chief complaints" in the electronic medical records. An "alert" is triggered when the proportion of visits for defined syndromes or geographic areas exceeds three standard deviations above predicted values, which are based on historical data. MDCH identified those potentially associated with oil exposure, which included rash, neurological, respiratory, and gastrointestinal syndromes. In addition, MDCH added an ad hoc query in order to detect chief complaints in the ED that contained "oil" and/or "spill". A limitation of this method, however, is that not all hospitals in the impacted communities participate in the MDCH ED syndromic surveillance system.

MDCH also monitors over-the counter pharmaceutical sales from several hundred retail pharmacies throughout the state, using computer algorithms to detect statistically significant increases in daily sales of: anti-diarrheal and anti-fever medications, cough syrup and other respiratory medications; child

electrolytes; and thermometers; and related products. This system was continually monitored with attention to the communities within the area of the Enbridge oil spill.

Results

One "alert" was recorded in Kalamazoo County for rash several days after the spill. Otherwise there were no notable changes in the frequencies of syndromes of interest in the area compared to overall daily rolling averages and yearly comparisons.

There were no notable increases in sales of over-the-counter pharmaceutical products; numbers of sales remained within typical levels for the season.

III. Discussion

MDCH and the impacted local health departments quickly established a multi-component public health surveillance system to assess and measure the health impacts associated with exposure to crude oil, its vapors, and/or its odors resulting from the Enbridge pipeline spill in July 2010. The surveillance system received 147 health care provider reports on 145 patients; identified 320 (58%) of 550 individuals with adverse health effects from four community surveys along the impacted waterways, and tracked forty-one calls that were placed to the poison center by the public.

Headache, nausea, and respiratory symptoms were the predominant symptoms reported by exposed individuals in all reporting systems. These symptoms are consistent with the published literature and the Enbridge Material Safety Data Sheet regarding potential health effects associated with acute exposure to crude oil.[5,6] A number of epidemiologic studies performed in the weeks or months following major oil spills have reported similar types of symptoms to those identified in our community surveys. Studies of acute health effects from an oil spill in Shetland, Scotland and Wales documented significant differences in similar sets of self-reported symptoms between exposed residents and control groups.[7,8] The post-spill prevalence of headache in the exposed for these two studies was similar to our that in our community surveys (Shetland: 32%; Wales: 38%; Enbridge communities: 34.5%) but higher in their control groups than our Comparison group (Shetland: 8%; Wales: 14.1%; Enbridge: 0.7%). This pattern was similar for other symptoms. In a study of a spill near Karachi Pakistan, the frequency of one or more symptoms was markedly higher in both the exposed and the control groups (96% in exposed and 70% in controls) than in our populations.[9] In a comprehensive review of all studies regarding the impact of oil exposure on human health, Aguilera et al. concluded that most studies "...provide evidence on the relationship between exposure and the appearance of acute physical, psychological, genotoxic and endocrine effects in the exposed individuals."[10]

Symptom prevalence as determined by our community surveys was significantly greater overall in the exposed communities than in the comparison community upstream from the spill. At the same time, there were some differences between the four communities regarding symptom prevalence. These differences may be associated with differences in geographical proximity to the river or health risk factors, including prevalence of chronic health conditions and smoking, both of which are inversely associated with socio-economic status. Symptom prevalence was lowest in the community (the Subdivision) with the lowest smoking and chronic disease prevalence, and the highest home values; and it was highest in the "Neighborhood", which had the highest chronic disease prevalence as well as the lowest home values.

There are a number of potential biases and limitations to the data obtained using this surveillance system. Regarding health care provider reporting, it is very likely that there was a significant amount of under-reporting by clinicians, a common problem with public health surveillance systems based on health care provider reporting. Reasons for under-reporting may include: not making a diagnosis that associates the oil exposure (either to the oil itself or to odors from the spill) to the symptoms, lack of understanding of reporting requirements, or lack of compliance because of barriers (e.g., time, office staffing, or concerns about patient confidentiality).

In the community surveys, there may have been response biases in the exposed communities associated with exaggerated reporting of symptoms, due to the considerable publicity surrounding the event and attendant legal issues. At the same time, there could have been underreporting of symptoms given the possibility that most affected individuals and households had relocated and were not at home when the door-to-door surveys were completed. Additionally, underreporting could have occurred because the respondents at the households were not completely familiar with the range of symptoms experienced by other household members about whom they provided information during the survey.

The lower completion rates in the Village and Comparison communities may have been because the survey teams started earlier in the evening than at the other sites, and thus missed people not yet home from work. It is unknown how this might have affected results. However, the very low refusal rate in the exposed communities suggested that these individuals understood why they were being interviewed and that it may have been in their best interest to participate. There was a much higher refusal rate in the Comparison community than the exposed communities (15% vs. 0.5%). We did not determine the reasons for refusing and therefore we do not know how this would have biased results from the comparison community survey. It could have reflected that there was no self-motivation for individuals in the Comparison community to participate other than general concern and good will, and thus some people were not willing

to take the time to talk with the interviewers, but there could have been a variety of reasons.

The survey of the workers in the one small worksite should be interpreted with caution. Results are subject to the instability of small numbers and there are no comparison data by which to judge the significance of the findings. Additionally, like the community surveys, there are a number of factors that could have contributed to recall bias, resulting in over- or under-reporting of symptoms. Because these individuals worked closely together, individual responses could have been influenced by prior discussions and concerns about the release. Further, overstated reporting of symptoms could have resulted from the considerable publicity surrounding the event and attendant legal issues. On the other hand, the open-ended format of the questions, rather than a structured list of possible responses, could have resulted in individuals being less likely to remember and report on specific types of symptoms.

A number of studies of the health effects of previous oil spills have focused on acute and chronic health effects to responders.[11,12,13] Current surveillance of response workers in the Deepwater Horizon spill in the Gulf of Mexico is tracking all injuries and illnesses of response workers, not just illnesses associated with oil exposure.[14] Our surveillance system, which was established to provide rapid detection of and response to acute health effects of oil exposure, was not designed to evaluate all injuries and illnesses, short or long term, in response workers. Other systems were in place within the Unified Command structure of the response to track all illnesses, injuries and "near-misses" among the response workers. Nevertheless, approximately 18% of the health care provider reports were of response workers experiencing health effects apparently associated with exposure to the oil.

Mental health effects of disasters, including anxiety, post-traumatic stress disorder, and depression have been an area of particular concern. Studies following the *Exxon-Valdes* oil spill in Alaska[15] and the *Sea Empress* in Wales[6] found that post-spill prevalence of a number of psychiatric disorders was significantly higher in exposed populations than unexposed individuals. Likewise, there was a greater proportion of individuals with self-reported psychiatric symptoms in our exposed communities than our Comparison community (4.7% vs. 0%), but overall prevalence was much lower than other studies. Unlike some other studies, which used validated mental health survey methodologies, our survey included only an open-ended question about symptoms, thus psychological symptoms were captured only if volunteered. Therefore, our assessment may have not fully captured the mental health effects of the spill.

Use of the PCC as the surveillance data center was an effective and responsive approach to the need for a rapidly functioning data collection and analysis system. Daily reports of numbers and types of reports were thus able to be provided by the PCC to the Command Center from where the spill response was

coordinated. The ED syndromic surveillance system was not notably sensitive, but this was not surprising because the hospital ED closest to the spill site does not participate in the system.

Beyond the significance of the health data itself for documenting the health impacts of the spill, the value of the face-to-face encounters between public health officials and the families coping with feelings of ill health, plummeting home values, and anxieties about their safety should be noted. These personal encounters provided some assurance to families that their needs and concerns were being heard and provided public health with an in-depth understanding of the situation. Combining a rapid community needs assessment and a health assessment is an approach that is being used more and more frequently during disasters.[16] Currently, the Centers for Disease Control and Prevention and the Council of State and Territorial Epidemiologists are organizing a series of trainings and workshops in "disaster epidemiology."[17] Results of these efforts will help inform future responses in Michigan to disasters.

IV. Conclusion

In response to concerns about acute health effects from exposure to spilled oil in this major disaster, state and local public health in Michigan quickly set up a multi-faceted public health surveillance system that included health care provider reporting, community surveys, calls from the public to the poison control center, and analysis of data submitted to the state's syndromic surveillance system. In spite of the limitations noted above, these data appear to provide a reasonable picture of the oil spill's acute health impacts, and these findings are consistent with other studies of oil spills.

A number of aspects to the public health surveillance response are noteworthy for consideration by public health agencies that are refining their non-infectious disease surveillance emergency response plans.

- A multi-component surveillance system was necessary to support the response.
- Chemical poisoning reporting regulations, which Michigan had put in place in 2007, were essential to support mandated health care provider reporting of oil-spill related illnesses.
- Use of the poison center as the data repository for reports by health care providers was an innovation that was effective and efficient. Daily summaries from the poison center provided the responders and public health agencies with sufficient information to understand the magnitude of the actual on-going health impacts of the spill, rather than relying on rumors or anecdotes.
- Epidemiologic competencies necessary for a quick response included survey design, data management, and analytic skills in descriptive epidemiology.
- Having the surveillance response take place in the oil-spill's Command Center, rather than public health offices at the state or county level, was

critical for ensuring that surveillance activities supported the daily needs of the Unified Command.

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Appendix: Map of the oil spill in Michigan (source: EPA[18])

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Tutorial on Upgrading of Oilsands Bitumen

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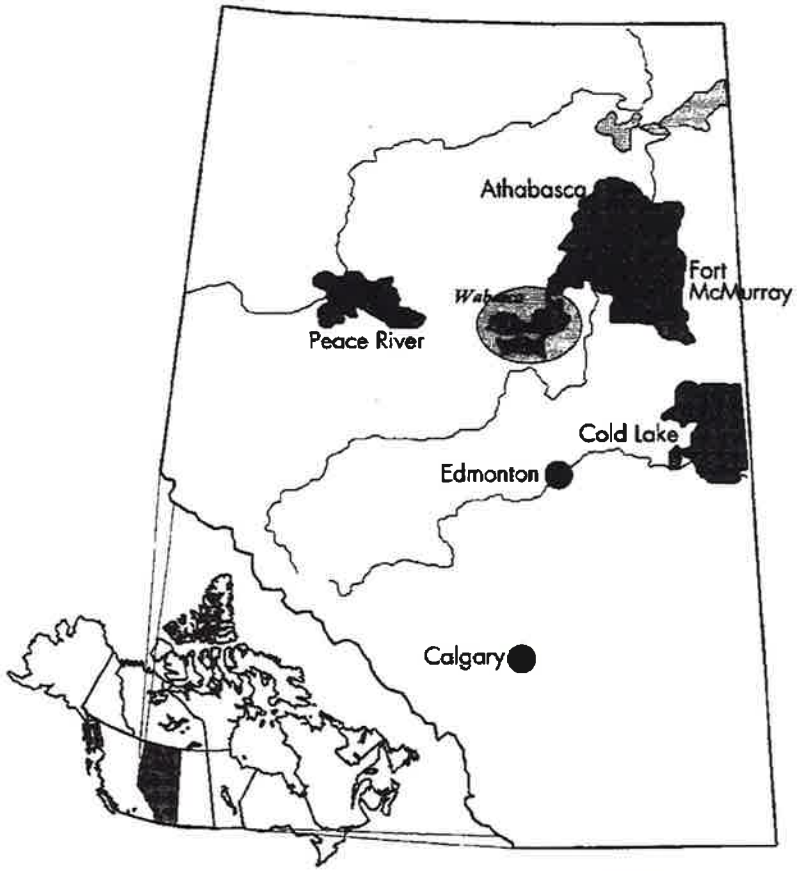
Oil Sands Upgrading

Department of Chemical and Materials Engineering

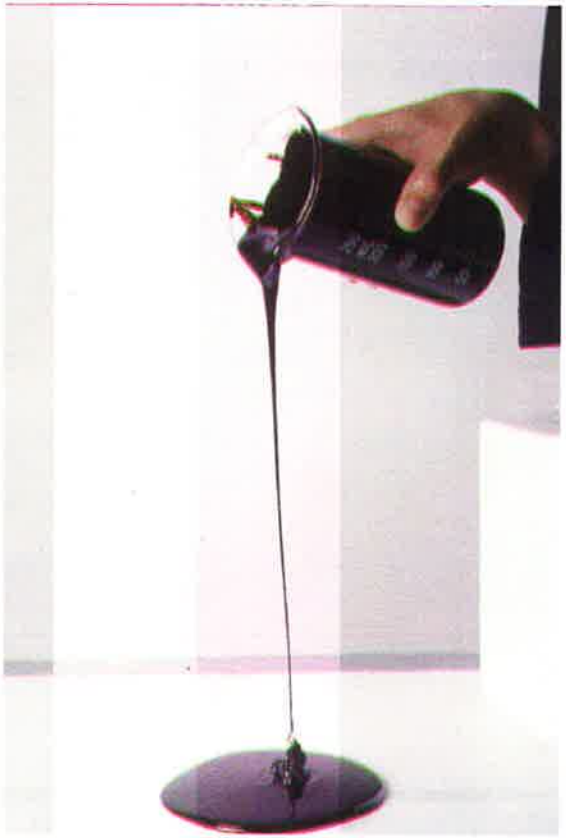
University of Alberta

What is Oilsands Bitumen?

- Heavy crude oil extracted from oilsands
- High viscosity, high sulfur oils
- Major production from mining near Fort McMurray, Alberta
- Also produced at Cold Lake and Peace River



Why Upgrade Bitumen?



- Oil refineries are designed for lighter crudes (density < 950 kg/m^3)
- Bitumen has density over 1000 kg/m^3, over 4% sulfur, viscosity 1000x light crude oil
- Needs upgrading to sell to most refineries

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Bitumen and Heavy Oil Upgrading

Upgrading: any fractionation or chemical treatment of bitumen or heavy oil to increase its value

- Minimum objective: reduce the viscosity to allow shipment by pipeline without adding a solvent
- Maximum objective: process to give a crude oil substitute of high quality (a "synthetic crude oil")

Bitumen and Heavy Oil Upgrading

- Minimum upgrading approach
  - Reduce viscosity and control solids and water
  - None in operation; research underway on simple, small scale processes
- Full upgrading approach
  - Reduce sulfur and maximize distillable oil
  - Large expensive plants much like refineries
- Focus on oil components that are too heavy to distill (boiling point over 524 °C)

Bitumen Properties

- Up to 1/2 of the bitumen can be recovered by distillation under a vacuum
- The residue contains large molecules, with molecular weights over 400
- Complex mixture of chemical species, including aromatics, substituted aromatics, organic sulfur and nitrogen compounds

Sulfur, Nitrogen and Metals in Residue Fraction of Feeds

	Sulfur, wt%	Nitrogen, wt%
Athabasca	5.14	0.56
Cold Lake	5.10	0.45
Lloydminster	4.69	0.53
Peace River	7.02	0.63

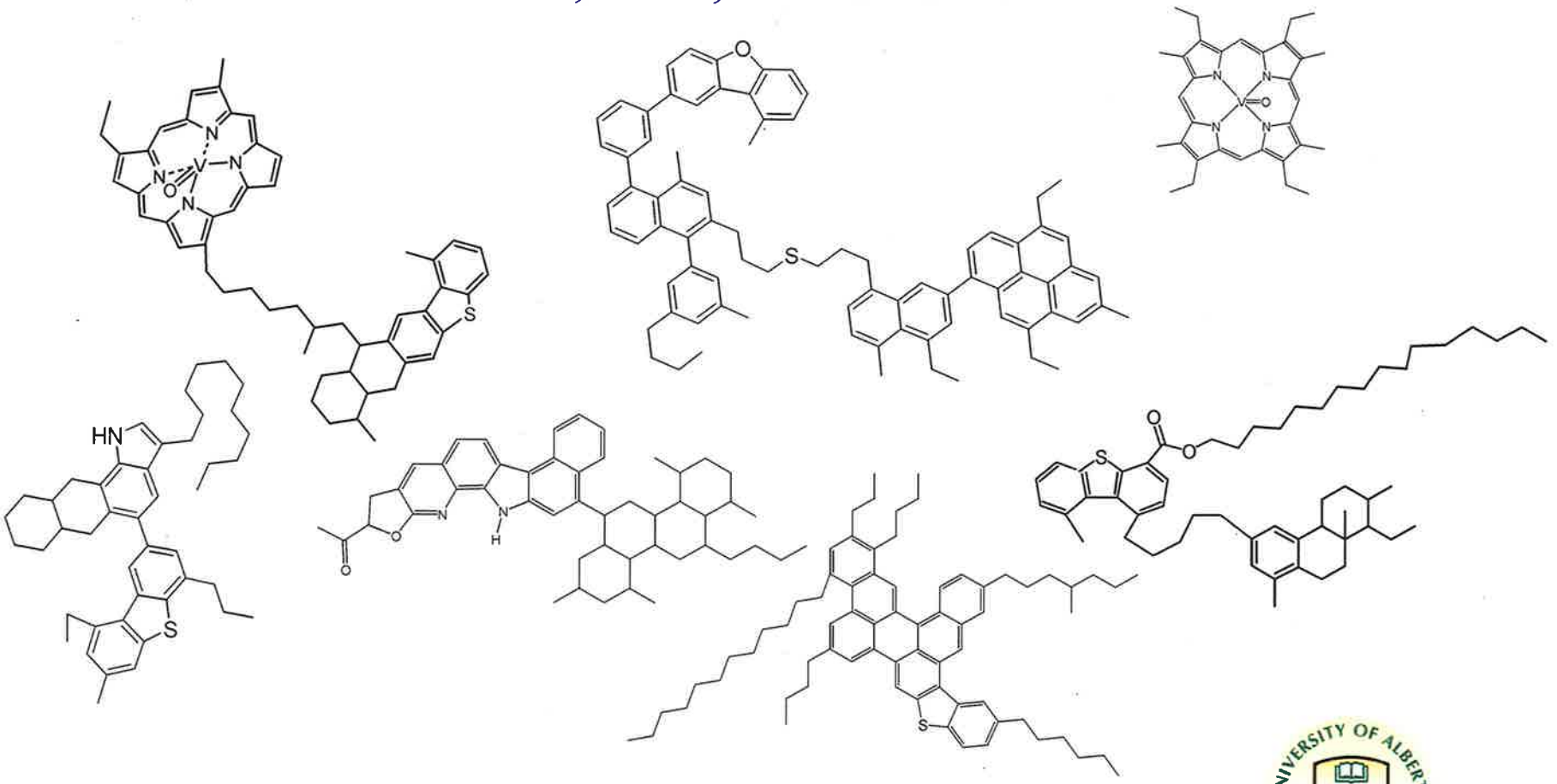
	Metals, ppm	
	Nickel	Vanadium
Athabasca	150	290
Cold Lake	200	490
Lloydminster	140	190
Peace River	130	410

Asphaltenes in Bitumen

- Dilution of Alberta bitumens in n-pentane or n-heptane gives 10-20% of brown/black solid precipitate
- This fraction is called "asphaltene", contains highest molecular weight (up to 5000) and most polar components in the oil
- Asphaltenes are very reactive during processing

Asphaltene molecules

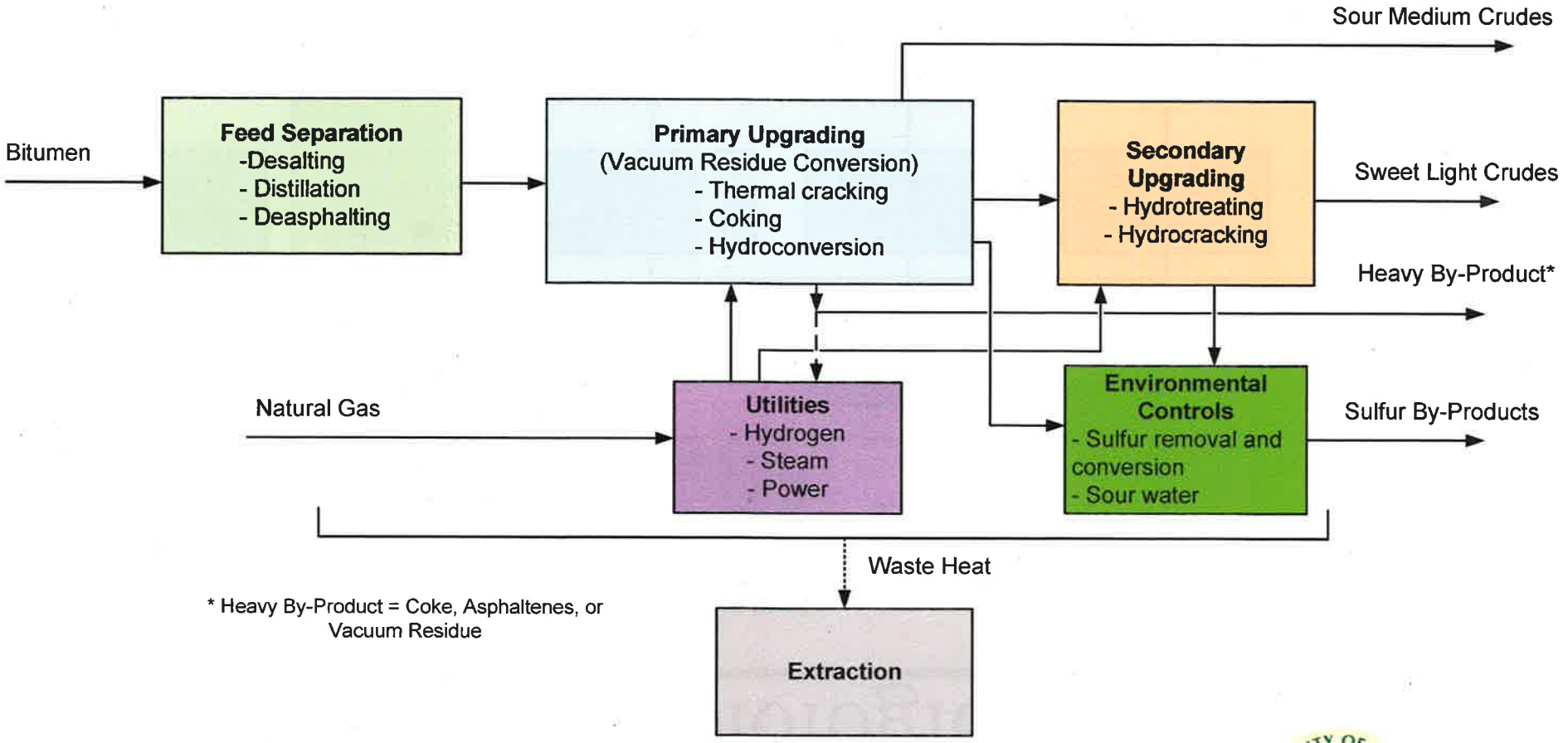
Based on Sheremata et al., 2004; Strausz 1999



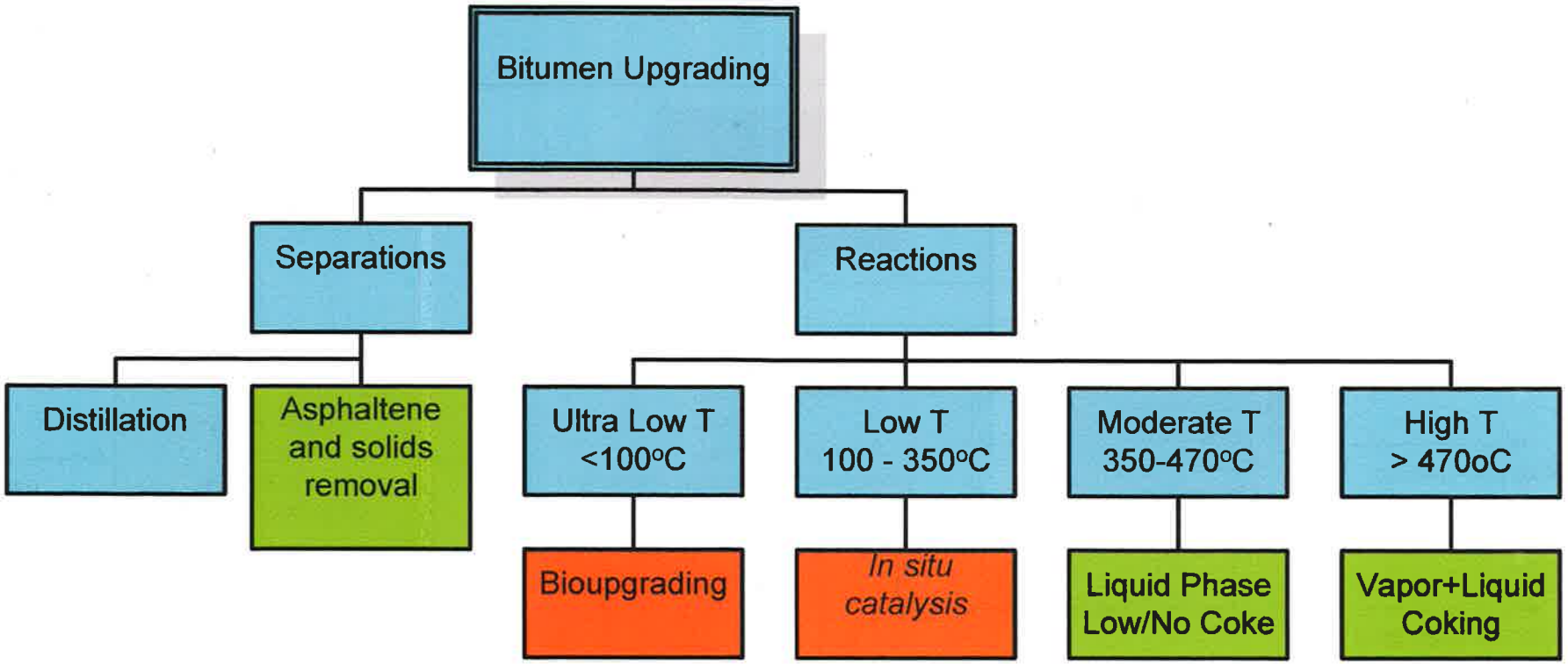
Upgrading Steps

- Large molecules that will not distill (called "Residue") must be *cracked* to give lighter molecules
- Cracking takes place in the *primary upgrading* step at over 400 °C
- Cracking products rich in sulfur and nitrogen, so *secondary upgrading* is required to get sulfur below 0.5%

General Upgrading Scheme



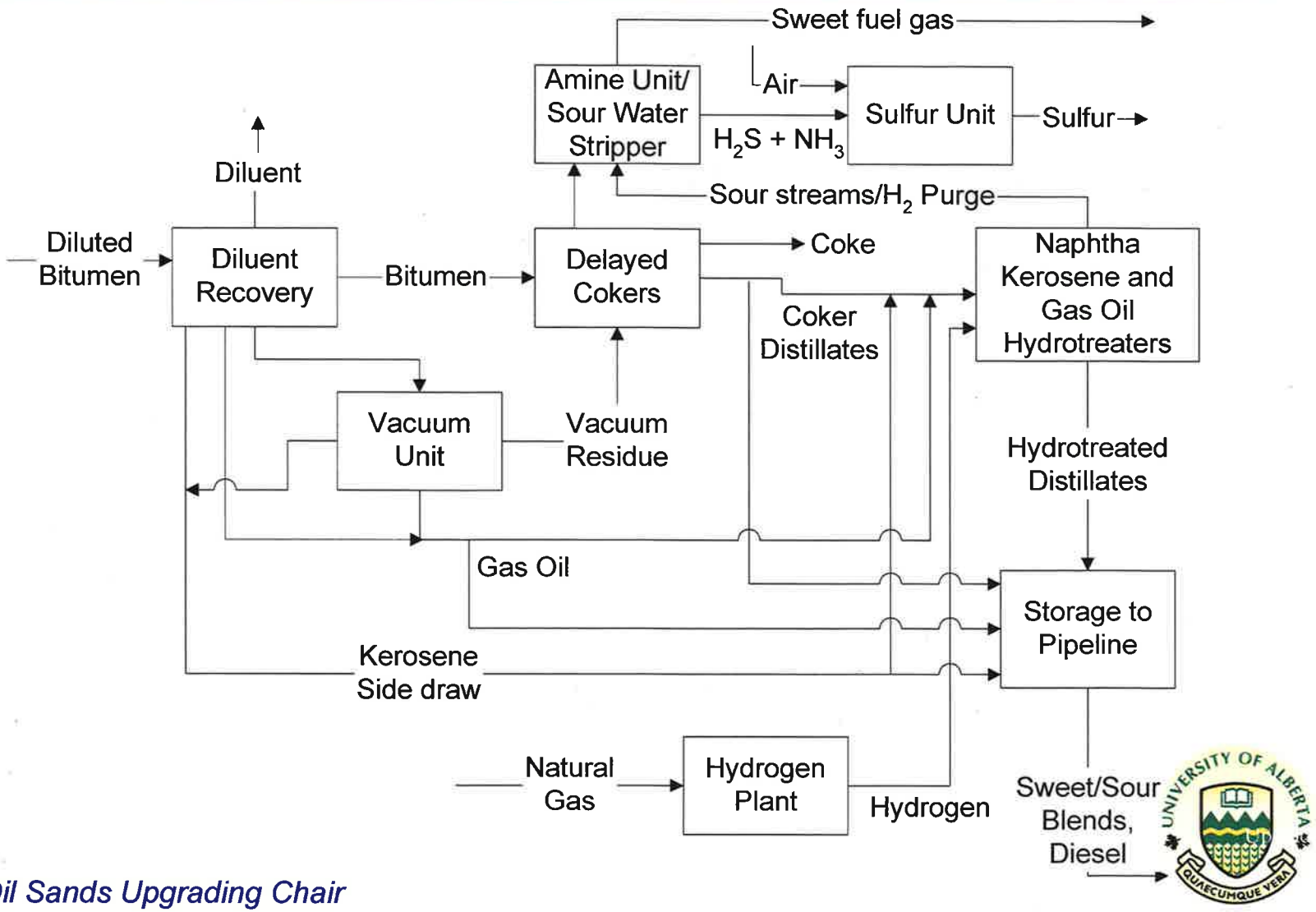
Upgrading Technologies



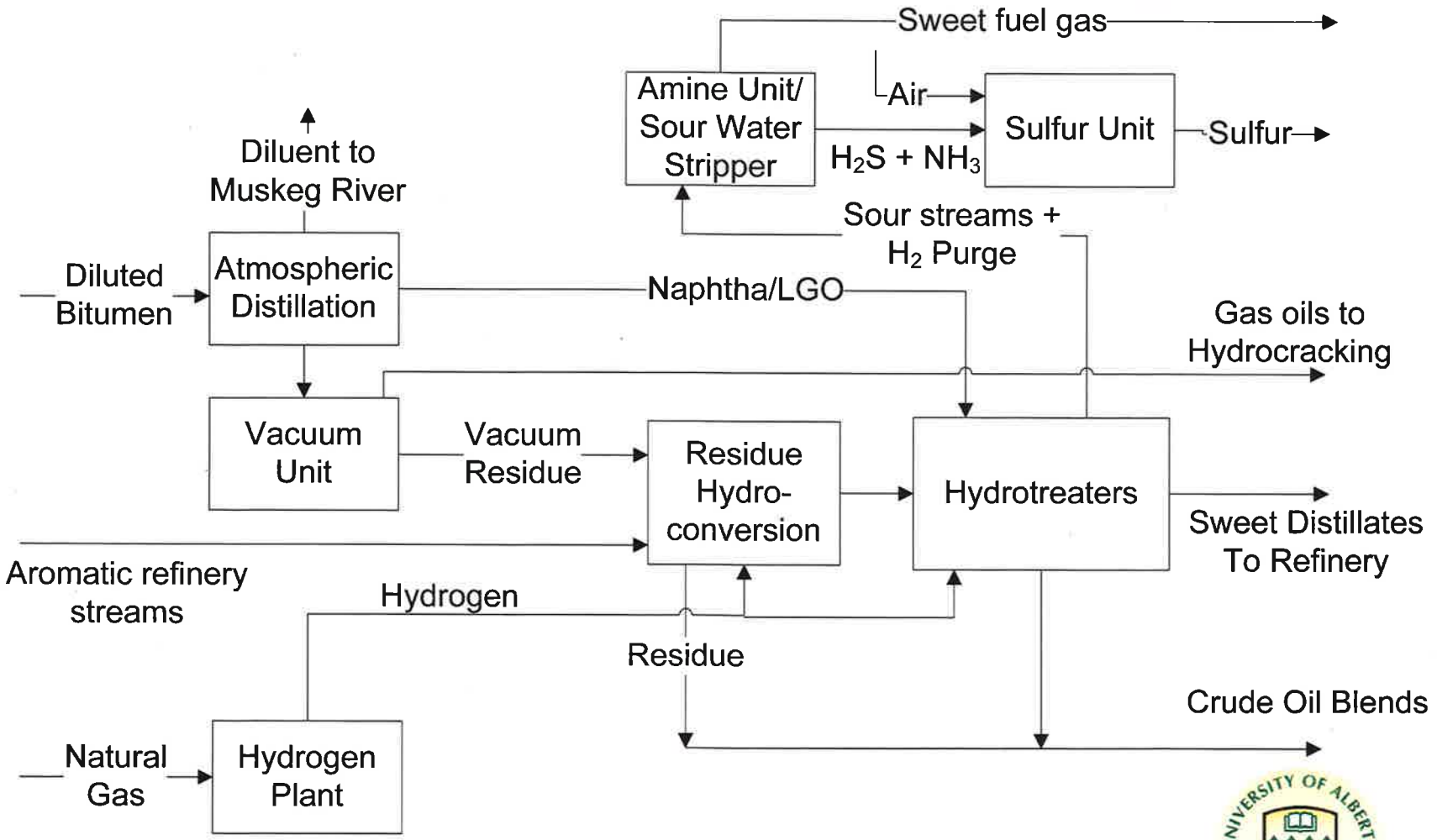
Commercial technology

Under development

Suncor Base Plant and Millennium

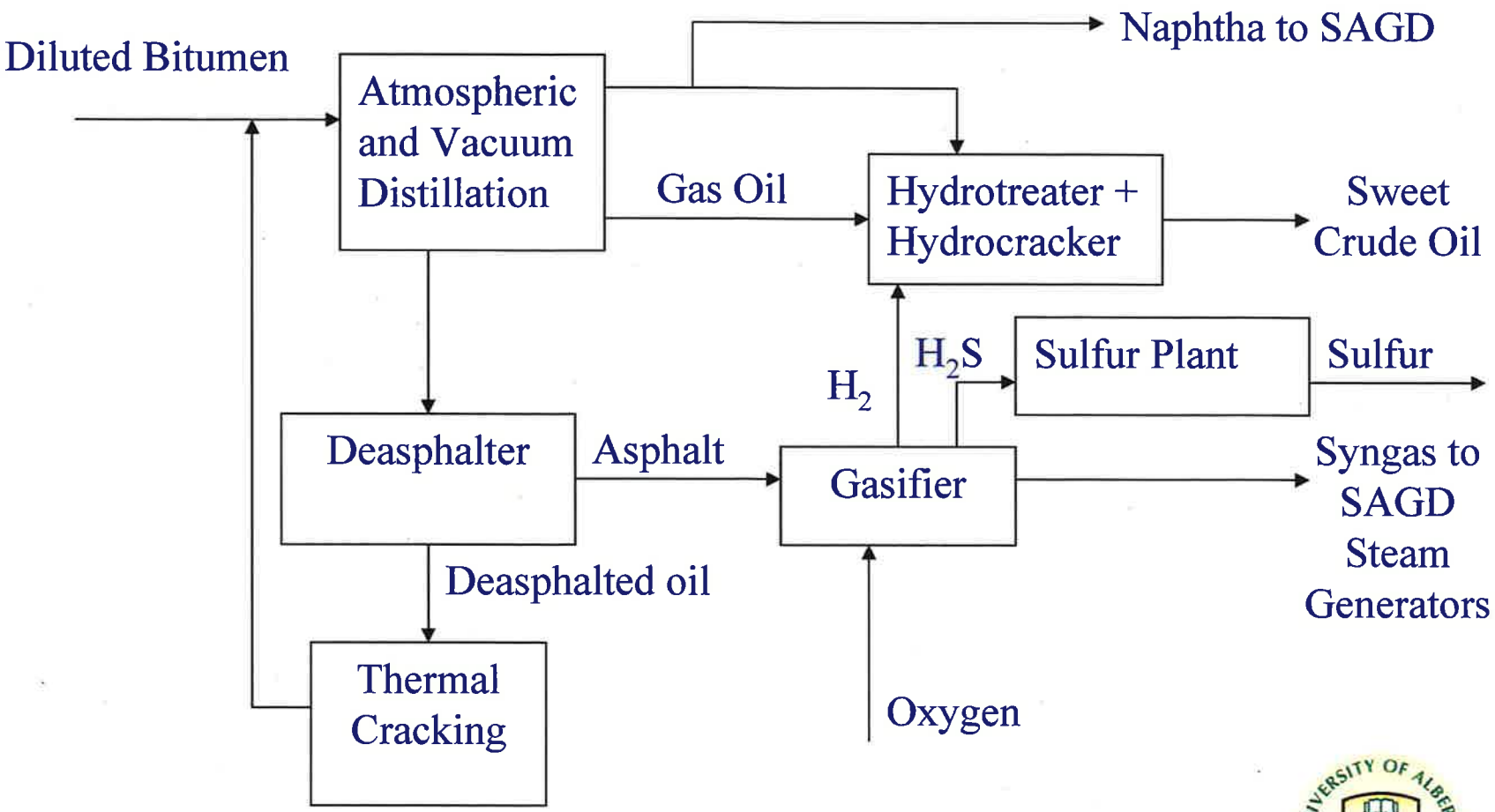


Shell Upgrader

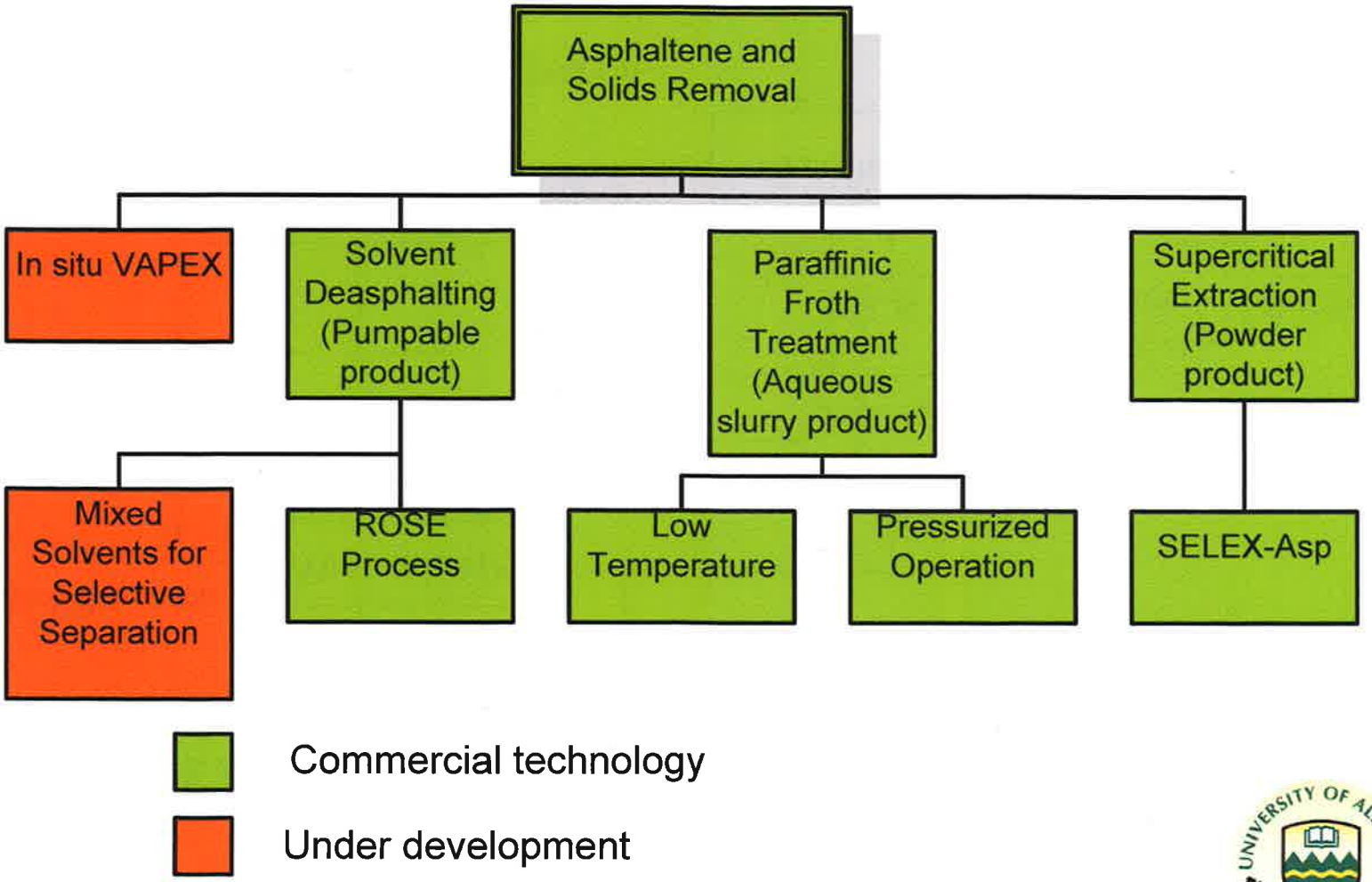


Oil Sands Upgrading Chair

Long Lake Upgrader



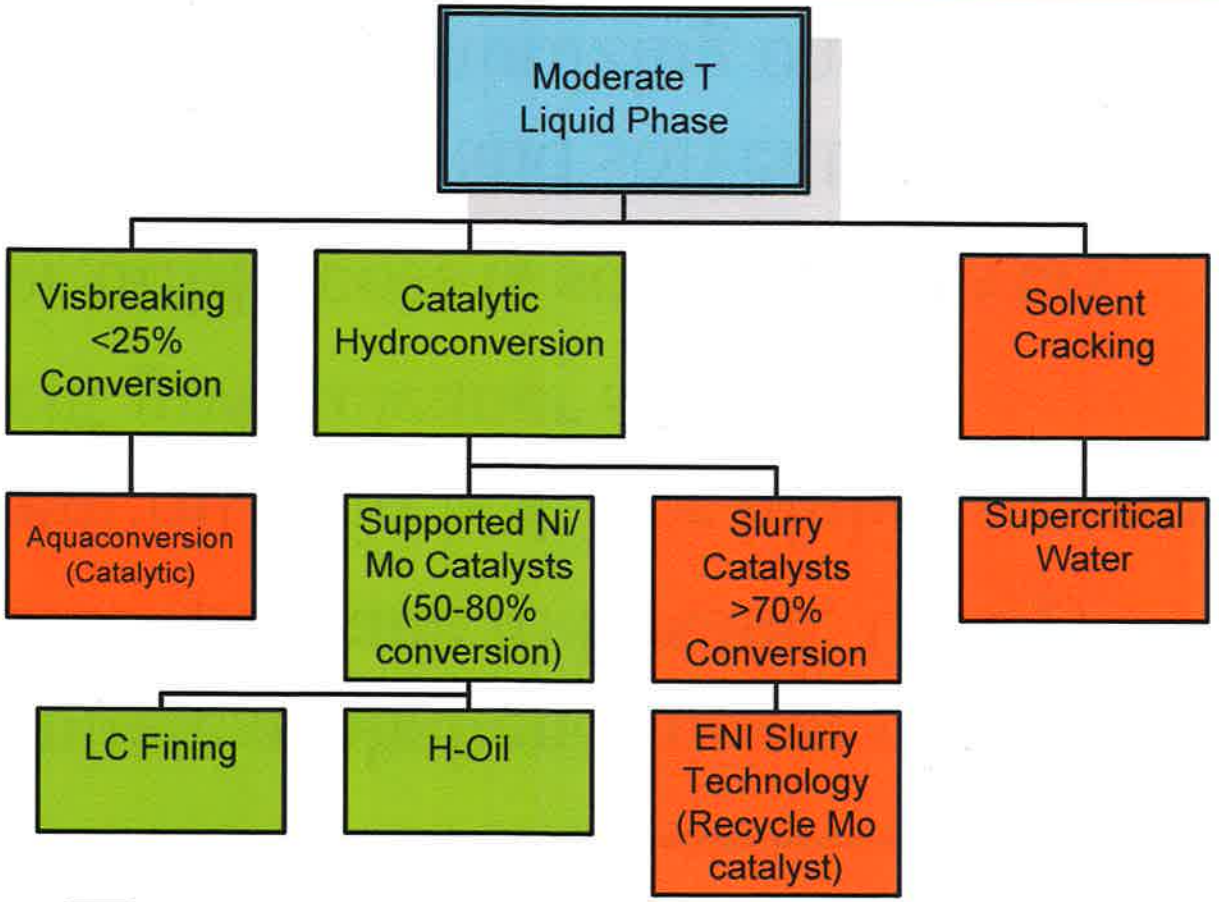
Asphaltene Separations



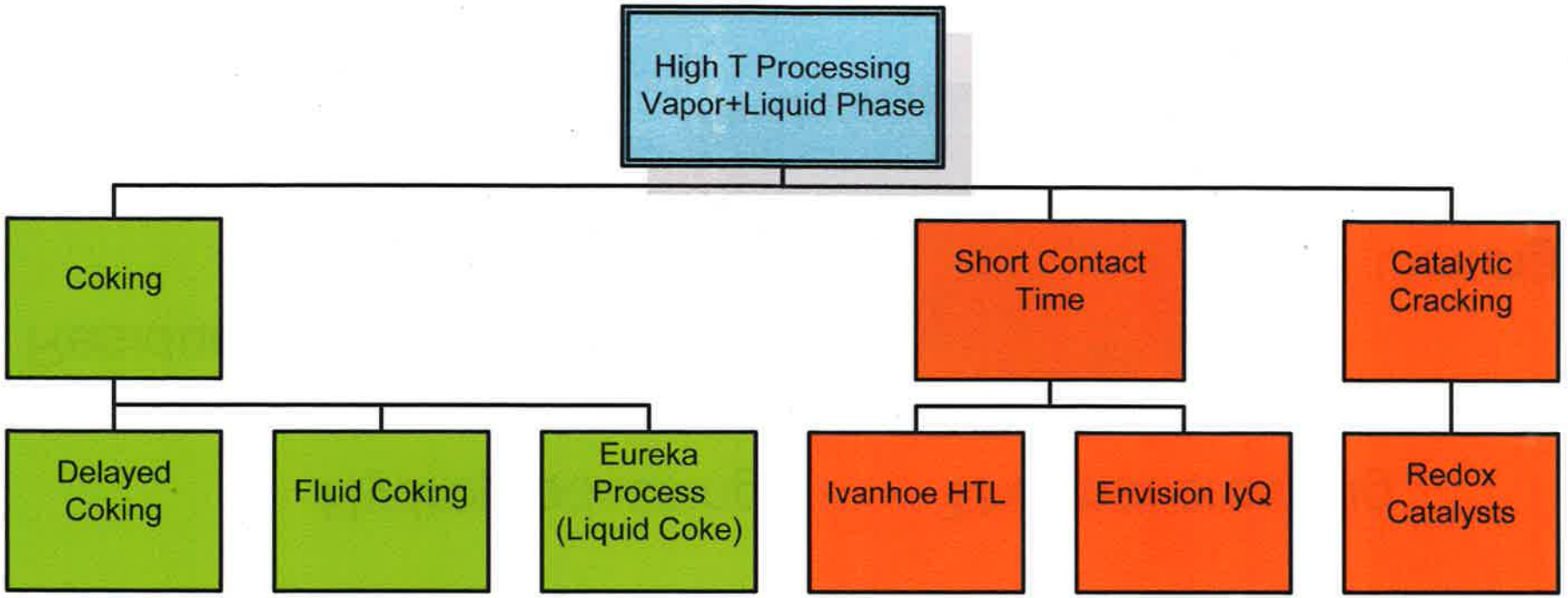
Solvent Deasphalting

- Precipitate asphaltenes using paraffinic solvents (propane to hexane; C_3-C_6)
- Solvent:oil ratio is 4:1 or higher - more solvent gives cleaner separation
- Major utility cost is solvent recovery
- Separation of oil and solvent is easy above T_c of solvent, minimizing energy costs
- Nexen Long Lake only oilsands example

Moderate Temperature Processes



High Temperature Processes

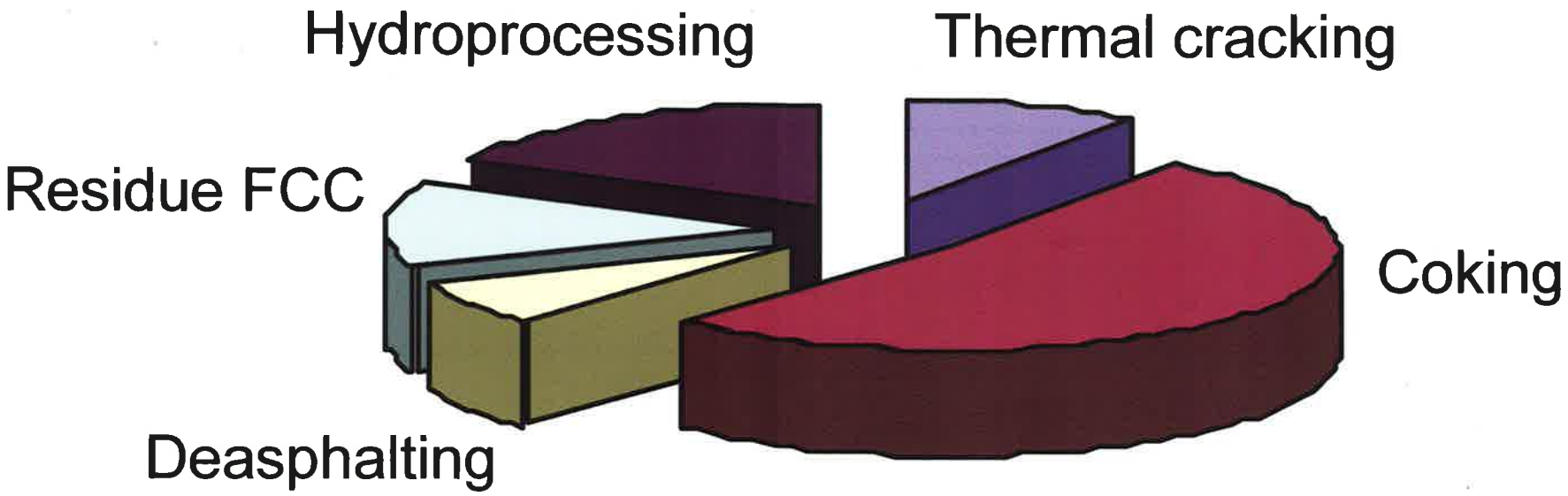


Commercial technology

Under development

Oil Sands Upgrading Chair

Processes for Primary Upgrading



North America + Venezuela, 1998

Thermal Conversion

- Thermal cracking underlies all current commercial reactor technology
- Rely on hydrogen gas to stabilize products

Process Characteristics - Coking

- Low pressure, reliable, safe process
- Low operating costs
- Low liquid product yields
- Coke by product is rich in sulfur, so this material is stockpiled in mines
- Main process for CNRL, Suncor and Syncrude

A Coke Primer

coke (kok) *noun*
A solid residue left after incomplete combustion of petroleum etc.

Probably from Northern English dialect
colk, core, of unknown origin

Thermal Cracking Reactions

- Breaking a C-C bonds required to get significant upgrading
- Two types of products - one stable and one unsaturated and unstable (alkenes)
- Use catalytic hydrotreating to stabilize the unsaturated products

Coking Technologies

- Delayed coking - liquid feed cracks in a large drum, and coke accumulates at the bottom with time (CNRL and Suncor)
- Fluid coking - liquid feed is sprayed into a bed of hot coke particles (Syncrude)

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Hydrogen Addition Processes

- Hydroconversion or Hydroprocessing
  - Combine some catalytic activity with thermal cracking
- Hydrotreating
  - Catalytic sulfur and nitrogen removal
  - Minimal cracking
- Hydrocracking
  - Gasoline production using a bifunctional catalyst

Hydroconversion

- Used by Husky, Shell, and Syncrude for primary upgrading
- Feed cracks in the presence of hydrogen and a supported-metal catalyst (Mo + Ni on aluminum oxide)
- Hydrogen suppresses coke, helps remove sulfur

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Hydrotreating

- Lower temperature process, with a similar catalyst (nickel + molybdenum on a support of aluminum oxide)
- High level (>90%) removal of sulfur, >70% removal of nitrogen
- Negligible cracking

Hydrocracking

- High pressure catalytic process used by refineries (Shell Scotford, Suncor Edmonton) to convert heavy distillates into feeds for gasoline
- Catalyst gives both cracking (e.g. zeolite) and hydrogenation activity
- Higher hydrogen pressure than hydroconversion or hydrotreating

The Chemistry of the Alberta Oil Sand Bitumen

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T6G 2G2

Bitumen chemistry is a complex field and poses a considerable challenge to the chemist. Bitumens are known to be comprised of an exceedingly large number of organic molecules which, in the case of the Alberta oil sand bitumen (AOSB), range from the simplest organic molecule, methane, to large polymeric molecules having molecular weights in excess of 15,000. In the AOSB hundreds of organic molecules representing paraffinic, olefinic, aromatic and heterocyclic structures with various functional groups have been identified. Most of these molecules are neutral but some have acidic or basic functions and are capable of salt formation. Others, by virtue of their skeletal or functional reactivity, may form π-bonded, hydrogen bonded or charge transfer complexes. While most of the constituent molecules are stable, some are relatively unstable and can undergo thermal decomposition even well below room temperature. Indeed, it has been demonstrated in recent studies that slow thermal processes are taking place in some of the major Alberta oil sand formations. These reactions are accelerated by the catalytic effect of the mineral matter present in the bitumen.

What distinguishes bitumen from conventional petroleum is the small concentration of low molecular weight hydrocarbons present and the abundance of high molecular weight polymeric materials. The latter are amorphous solids which are dissolved in colloidal form in the lower molecular weight liquid constituents, endowing the bitumen with a viscous, syrupy consistency. The high molecular weight solids are soluble in liquid aromatics such as benzene or toluene and insoluble in low molecular weight paraffins and therefore can be separated from the bitumen by *n*-pentane precipitation from a benzene solution of the bitumen. The solid precipitated this way is called asphaltene. It is evident that such an operational definition of the asphaltene based entirely on solubility may group together a large number of structurally unrelated molecules. Current studies provide clear evidence that the polymeric framework of the asphaltene molecules derived from various sources may be quite different.

Most asphaltenes are rich in heteroatoms, oxygen, nitrogen and especially sulfur. The asphaltene content of the AOSB is in the 16-25% range and the asphaltene contains ~80%C; 8.0%H; 8-9%S, 2.5%O; and 1.0%N.

The main difficulty associated with underground recovery of the AOSB is the consequence of the extremely high viscosity of the bitumen for which the asphaltene is mainly responsible. Therefore, in order to develop a rational approach to the conceptual understanding and practical resolution of the problem one must start with the study of asphaltene itself. The molecular structure of this material has not yet been fully understood and is still the subject of some conjecture. Apart from its pertinence to *in situ* recovery technology, knowledge of the molecular structure of asphaltene is the key to its possible future commercial utilization, to the elucidation of its origin and diagenetic history, and may conceivably shed some light on the history of the bitumen formations.

The chemical composition of the deasphaltened bitumen, the maltene, is also of interest from both a practical and conceptual point of view. This fraction represents the bulk of the bitumen and is one of the major factors which determine the commercial value of the separated bitumen. Detailed

knowledge of the chemical composition of this fraction in conjunction with geological and microbiological data will ultimately lead to the unravelling of the mystery of the source and origin of the bitumen formations of Alberta, the relation between the various major oil sand deposits and their interrelation with conventional oil accumulations of the region.

The Thermal Maturation Process.

The realization that a slow, thermal maturation is presently occurring in the bitumen formations of Northern Alberta was the ultimate conclusion of a recent kinetic study of the low temperature thermal behaviour of the oil sand [1-4].

The oil sand in its natural state was shown to contain small quantities of volatile hydrocarbons, methane, propane, propylene and, curiously, neopentane, together with some acetaldehyde. The latter molecule is too reactive to survive long periods of time in the oil sand and must have been formed relatively recently. Heating of the oil sand under controlled conditions resulted in the production of additional amounts of the volatiles present originally in the sand and also some new products. The amounts of gases in question are small, in the ppb-ppm range, nonetheless they can be measured with reasonable accuracy. Thus, measurements of gas evolution at moderately high temperatures provided a key to the detection and monitoring of chemical changes which take place in the oil sand. In the course of the measurements it was found that the rate of evolution of gases, hydrocarbons, acetaldehyde, CO_2, CO, H_2S, COS, CS_2, SO_2, etc., obeyed Arrhenius' law which made it possible to determine the activation energy and preexponential factor for the production of each gas. The activation energies were all low, 5-23 kcal/mole, and the preexponential factors high. From these Arrhenius parameters it was then possible to determine the rate constant for the evolution of each gas in the formation at the formation temperature, 5°C. This in turn made it possible to estimate the time interval required for the accumulation of the quantity of gases present. The results indicate time periods from several days to several months and therefore we are forced to conclude that a slow thermal maturation is taking place presently in the Athabasca oil sand formation and the measured concentration of gases represent a steady state distribution governed by the rate of formation and rate of loss by chemical reactions and/or diffusion. The role of microbial degradation as compared to the thermal processes appears to be negligible.

The total amount of gases released is small and even at 210°C comprises only a few tenths of one percent of the bitumen. But because the volatilized fragment represents a relatively small proportion of the molecules affected by the decomposition, the fractional amount of bitumen still capable of undergoing maturation would be much higher, probably in the order of several percent.

Similar studies on the separated bitumen, asphaltene and maltene, have indicated that the mineral matter present in the oil sands acts as a catalyst for decomposition and that maltene and asphaltene behave in a similar manner but the activation energies associated with their decomposition are significantly higher than those of the whole oil sand. For a better understanding of this maturation process it will be necessary to extend these studies to all major oil sand deposits, examine the relation between reactivity and depth, identify reactive precursors such as those of neopentane and acetaldehyde and to improve the reproducibility and accuracy of the experimental techniques employed.

At present virtually no information exists on the magnitude or composition of the gas accumulations in the oil sand although this would be extremely important in order to understand what is transpiring in the formations.

The results of current similar studies on the Cold Lake formation indicate an analogous thermal behaviour for the bitumen. The total amounts of volatile hydrocarbons produced in the formations are difficult to estimate, but may be in the range of 10^5-10^6 tons annually. A fraction of this would undoubtedly escape into the atmosphere and influence the air quality of the region.

The Chemical Composition of the Maltene.

The composition of maltene, the deasphaltened bitumen, has been investigated by the API-60 procedure, a somewhat simplified version thereof and also by microanalytical procedures developed in organic geochemical studies [5-7].

The elemental compositions of Peace River, Wabasca, Athabasca and Cold Lake bitumens are given in Table I, and Table II gives their gross composition according to classes.

TABLE I

Overall Composition of Alberta Oil Sand Bitumens [5-7]

Fraction	Source*	C	H	N	O	S	H/C	mw
		%						
Whole Bitumen	A	83.98	10.22	0.65	1.97	4.57	1.46	620
	P	81.68	9.98	0.14	2.08	5.60	1.46	520
	C	83.93	10.46	0.23	0.94	4.70	1.49	490
	W	82.44	10.32	0.42	0.82	5.51	1.49	600
Asphaltenes	A	81.31	7.88	1.06	2.79	7.53	1.16	5,920
	P	79.87	8.15	0.78	2.08	8.82	1.21	3,500
	C	80.54	7.39	1.15	1.78	6.51	1.10	8,140
	W	80.46	8.20	0.99	1.16	8.40	1.21	5,760
Deasphaltened Oil	A	84.38	10.63	0.07	0.87	3.91	1.51	435
	P	82.89	10.61	0.08	1.27	5.41	1.53	440
	C	84.19	11.01	0.27	0.61	3.89	1.54	430
	W	82.99	10.74	0.28	1.04	5.25	1.54	530

* A=Athabasca, P=Peace River, C=Cold Lake, W=Wabasco bitumen.

TABLE II

Gross Composition of Alberta Oil Sand Bitumens [5-7]

	% of whole bitumen*			
	Cold Lake	Athabasca	Peace River	Wabasca
Asphaltenes	15.3	16.9	19.8	18.6
Deasphalted oil	84	83.1	80.0	81.2
Acids	15.2	13.69	12.0	10.3
Bases	6.38	6.5	6.8	6.38
neutral N-compounds	1.15	1.35	1.41	2.67
Saturates	21.3	18.3	15.15	14.60
Monoaromatics	8.3	8.1	8.57	7.61
Diaromatics	3.6	3.8		
Polyaromatics+ non-defined polar compounds	24.35	23.8		
Thiopheno-aromatics from mono- and diaromatic fractions	0.8	0.88		

*Bitumen = 100%

As indicated by these data and the results of computerized gc-ms multiple ion cross scans [8] and matrix analyses, the bulk and fingerprint composition of these bitumens are quite similar and the minor differences which appear seem to signify some subtle trends in diagenetic history.

The similarity in chemical composition points to a common origin for the four major oil sand deposits. The distribution pattern of steranes (C_{26}, 5%; C_{27}, 44%; C_{28}, 33%; C_{29}, 17%; C_{30}, 1%) is reminiscent of the ratio obtained from the reduction of marine sterol mixtures and therefore suggests a marine origin. From microbiological model studies it can be concluded that AOSB is a microbiologically degraded residue. Microbiological simulation experiments showed that the end product of the microbiological degradation of a conventional oil is a bitumen-like material, low in paraffins and small ring, less alkylated aromatics and rich in polycondensed polyalkylated aromatics and polar materials, including asphaltene.

The Peace River and Athabasca bitumens seem to represent the end product of biodegradation. The Cold Lake bitumen contains slightly more paraffins, including small amounts of *n*-alkanes, and less asphaltene, and the Lloydminster heavy crude which is the least biodegraded material contains more paraffins and even less asphaltene.

The *n*-paraffins of the Cold Lake bitumen range from C_{13} to C_{33} with a maximum at C_{19} and a carbon preference index of 1.0. The isoprenoid fraction contains both pristane and phytane along with lower and higher mw homologues of the series.

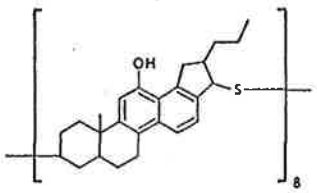
The gc-ms fingerprint spectra of the hopane series in all the oil sand bitumens showed the presence of the 17α(H), 21β isomer of C_{27}, C_{29}-C_{35} hopanes with a maximum at C_{30}. Both C_{22} diastereoisomers were present in approximately equal quantities. Living organisms synthesize the 17β(H), 21β isomer and the high conversion to the 17α(H), 21β isomer is indicative of some geothermal history of the oil sand, although the role of the mineral matter as a catalyst for the isomerization cannot be readily assessed.

Steranes and hopanes comprise only a small fraction of the saturates, the bulk of which still has not been identified. The compositions of the other separated fractions, the acids, bases, neutral nitrogen compounds, olefins, mono, di and polycycloaromatics, etc., are not well established either at present, although recent gc-ms multiple cross scan and matrix analyses have yielded valuable estimates.

As was pointed out above, the combined microbiological simulation and chemical analytical studies leave little doubt as to the severity of microbial degradation which the oil sand bitumen has suffered, nonetheless the question of whether the oil sand bitumen is a primary degraded kerogen without a significant thermal history, similar in character to the precursors of conventional oils, or whether it is the microbiological end product of a conventional oil has not been settled unambiguously.

The Chemistry of Bitumen Asphaltene.

The asphaltene fraction of the Athabasca bitumen has recently been subjected to detailed chemical studies for the elucidation of its molecular structure [9]. Here the essential question was to establish the role of sulfur in the polymeric framework of the asphaltene molecule. It was possible to show that in the Athabasca asphaltene the sulfur is present largely in the form of sulfides holding together dicycloaromatic carbon units of average molecular formula



$C_{200}H_{240}N_2S_7O_4$ (mw 3,000)

Two of these molecules are joined together by hydrogen bonding. In reality, asphaltene is a complex mixture of molecules with a broad distribution of molecular weight and with significant deviations in the structures of monomeric units from the average dicycloaromatic unit above.

Esr studies of the Athabasca asphaltene showed the presence of paramagnetic species, approximately one free spin per 80-90 molecules and most of the molecules carrying free spin appear to carry more than one [10-12]. The resonance at g=2.0036 is thought to be associated with polycycloaromatic doublet radicals and singlet and triplet state diradicals. The relative distribution of these radicals can be influenced by temperature, the addition of paramagnetic gases such as O_2 and NO, and by other means. The effects are reversible. Since the minimum degree of condensation required in a cycloaromatic system for the formation of stable free radicals is about six, it seems that at least 1.1% of the asphaltene molecules contain at least one hexacycloaromatic sheet.

After completion of the initial phase of these studies on the molecular structure of the Athabasca asphaltene, current research has been extended in three directions:

a) It appeared to be desirable to compare the elemental composition, mw and the number of sulfide linkages in the polymeric framework of various asphaltenes from different sources. The results suggest that Athabasca asphaltene contains the highest percentage of bridged sulfur, as will be shown in a separate article [13,14].

b) More precise information on the chemical nature of the monomeric units liberated in the reductive cleavage of the sulfur-carbon bonds or in the mild thermolysis of the asphaltene will be obtained using a combination of hplc, gel permeation chromatography and organic geochemical microanalytical techniques.

c) Taking the synthetic approach, the feasibility of making asphaltene-like materials from simple or complex organic base materials by treatment with sulfur has been explored. In this way it was possible to prepare asphaltene-like materials with molecular weights approaching those of natural asphaltene in which the carbon monomeric units are joined together with sulfide linkages. Details of these studies will be given in another article [15].

Acknowledgements.

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CLINICAL RESEARCH

Road traffic noise and stroke: a prospective cohort study

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Aims Epidemiological studies suggest that long-term exposure to road traffic noise increases the risk of cardiovascular disorders. The aim of this study was to investigate the relation between exposure to road traffic noise and risk for stroke, which has not been studied before.

Methods and results In a population-based cohort of 57 053 people, we identified 1881 cases of first-ever stroke in a national hospital register between 1993–1997 and 2006. Exposure to road traffic noise and air pollution during the same period was estimated for all cohort members from residential address history. Associations between exposure to road traffic noise and stroke incidence were analysed in a Cox regression model with stratification for gender and calendar-year and adjustment for air pollution and other potential confounders. We found an incidence rate ratio (IRR) of 1.14 for stroke [95% confidence interval (CI): 1.03–1.25] per 10 dB higher level of road traffic noise (L_{den}). There was a statistically significant interaction with age ($P < 0.001$), with a strong association between road traffic noise and stroke among cases over 64.5 years (IRR: 1.27; 95% CI: 1.13–1.43) and no association for those under 64.5 years (IRR: 1.02; 95% CI: 0.91–1.14).

Conclusion Exposure to residential road traffic noise was associated with a higher risk for stroke among people older than 64.5 years of age.

Keywords Stroke • Traffic noise • Epidemiology

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Introduction

Increasing noise from traffic occurs in parallel with urbanization. Acute exposure to noise is believed to activate the sympathetic and endocrine systems, thereby causing changes in blood pressure and heart rate and release of stress hormones.[1–3] Furthermore, exposure to noise during the night at normal urban levels has been associated with sleep disturbances.[4]

Persistent exposure to noise is believed to increase the risk of cardiovascular disorders. An overview from 2006 of 61 epidemiological studies of the effects of exposure to transport noise (road, air, and rail) on cardiovascular health showed associations with hypertension and ischaemic heart disease in adults.[5] Recently, a meta-analysis indicated that the risk for myocardial infarction with road traffic noise increased in a dose–effect manner[6]; this finding was supported by those of a case–control study that, as the first study of its kind, adjusted for exposure to air pollution.[7]

Stroke is a major cause of disability and death worldwide.[8] There has been no investigation of the relation between exposure to transport noise and risk for stroke, although some of the suspected effects of noise, e.g. increased blood pressure, are associated with risk for stroke.[9] One study of the relation between exposure to road traffic noise and overall cerebrovascular mortality showed no association.[10]

The aim of the present study was to investigate the association between exposure to transport noise and risk for stroke in a

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cohort after adjustment for air pollution and other important risk factors for stroke.

Methods

Study population

The study was based on the Diet, Cancer, and Health cohort study in which 160 725 people were invited to participate between 1993 and 1997. The 160 725 people was a random sample of all eligible people living in the Copenhagen or Aarhus area that was free of cancer and between 50 and 64 years of age at the time of invitation. Participants had to have been born in Denmark.[11] All in all, 57 053 people accepted the invitation and were enrolled into the cohort. Participation was based on written informed consent. The study was conducted in accordance with the Helsinki Declaration and approved by the local Ethics Committees.

At enrolment, each participant completed self-administered, interviewer-checked, questionnaires regarding lifestyle habits, diet, health status, and social factors. The average amount of tobacco smoked each day (smoking intensity) was calculated by equating a cigarette to 1 g, a cheroot or a pipe to 3 g, and a cigar to 5 g of tobacco. In the food frequency questionnaire, participants were asked how often on average they had consumed different types of foods during the preceding 12 months. The frequency consumption was categorized into 12 groups ranging from never to 8 or more times daily. A mean daily intake of foods (g/day) was calculated by multiplying the frequencies of intake by a gender-specific portion size using the software Foodcalc version 1.3.[12] Participants were also asked to state their average amount of alcohol consumption as the intake of specific amounts of each beverage type: light, normal, and fortified beer; red, white, and fortified wine; and spirits. On the basis of ethanol content in the different beverage types, these categories were converted into number of standard drinks (12 g alcohol) and added to yield a measure of average gram alcohol per day. Coffee consumption (filter coffee) was also stated and based on this we defined four categories: $\leq$1, 2–3, 4–5, and $\geq$6 cups of coffee per day. Trained staff measured height, weight, and diastolic and systolic blood pressures.

Identification of outcome

Stroke events among participants were identified by linking the unique personal identification number of each cohort member to the Danish National Hospital Registry, which has collected nationwide data on all non-psychiatric hospital admissions since 1977. Since 1995, patients discharged from emergency departments and outpatient clinics have also been registered.[13] The Danish National Board of Health maintains the register and assures the quality of the data. We identified cohort members who were registered with a discharge diagnosis of stroke (International Classification of Diseases, revision 8 codes 431.0, 431.9, 432.0, 432.9, 433.09, 433.99, 434.09, 434.99, 436.0, and 436.9 and International Classification of Diseases, revision 10 codes DI61, DI63, and DI64). We only included cases for whom stroke was the primary reason for hospitalization, also when the primary stroke resulted in death. Participants with a discharge diagnosis of stroke before enrolment into the Diet, Cancer, and Health cohort were excluded ($n = 542$). The first hospitalization for stroke was used as the outcome.

Exposure assessment

A complete residential address history was collected between enrolment and event or censoring date for 53 162 of the 57 053 cohort members. Exposure to road traffic noise was calculated for the years 1990, 1995, 2000, and 2005 using Soundplan (version 6.5, http://www.soundplan.dk/) for all 61 873 residential addresses at which these 53 162 cohort members had lived between enrolment and event/censoring. This noise calculation programme implements the joint Nordic prediction method for road traffic noise, which has been the standard method for noise calculation in Scandinavia during many years.[14,15]

The input variables for the noise model were point for noise estimation (geographical coordinates and height of the floor of the residence); road lines, with information on yearly average daily traffic, traffic composition, traffic speed, and road type (motorways, rural highways, roads wider than 6 m, and other roads); and building polygons for all buildings, including information on height. We assumed that the terrain was flat, which is a reasonable assumption in Denmark, and that urban areas, roads, and areas with water were hard surfaces whereas all other areas were acoustically porous. No information was available on noise barriers.

Road traffic noise was calculated as the equivalent continuous A-weighted sound pressure level (L_{Aeq}) at the most exposed facade of the dwelling at each address for the day (L_d; 07:00–19:00 h), evening (L_e; 19:00–22:00 h), and night (L_n; 22:00–07:00 h) and expressed as L_{den} (as an indicator of the overall noise level during the day, evening, and night) by applying a 5 dB penalty for the evening and a 10 dB penalty for the night.[16]

We aimed at investigating effects of relatively recent exposure in our main analyses. Therefore, the noise level used in the analyses was the yearly mean exposure at a residence at a given age.

Exposure to railway noise was calculated with the joint Nordic prediction method based on general information about traffic in 1993–2000. Screening by designated noise screens and buildings was not considered. The noise impact from airports and airfields was determined from information about noise zones obtained from local environmental authorities. The programmes DANSIM and INM3, which fulfil the joint Nordic criteria for air traffic noise calculations, were used.

The curves for railway and aircraft noise were transformed into digital maps, and noise levels were linked to each address by geocodes. In the statistical analysis, exposure to noise from railways and airports was entered categorically as above and below L_{Aeq} 60 dB and L_{den} 55 dB, respectively. Different exposure thresholds were chosen because annoyance and sleep disturbance are greater with airport noise than with railway noise.[4,17] About 17% of the cohort members were exposed to railway noise in excess of 60 dB, and 1% was exposed to aircraft noise above 55 dB.

The concentration of NO_x in the air was calculated with the Danish AirGIS modelling system for each year (1993–2006) at each address at which the cohort members had lived. AirGIS allows calculation of air pollution at a location as the sum of: local air pollution from traffic in the streets, with the Operational Street Pollution Model; the urban background contribution, calculated with a simplified area source dispersion model (SUBmodel); and a regional background contribution.[18] Input data for the AirGIS system included traffic data for the period 1960–2005, emission factors for the Danish car fleet, street and building geometry, building height, and meteorological data.[19] The AirGIS system has been successfully validated in several studies.[18–20]

We calculated the yearly mean exposure for noise and NO_x from 1993 to 2006 for all addresses for which we had information on. Based on this, we determined the difference in noise and NO_x means, respectively, over time.

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Statistical methods

The analyses were based on a Cox proportional hazards model with age as the underlying time.[21] This ensured comparison of individuals of the same age. We used left truncation at the age of enrolment, so that people were considered at risk from enrolment into the cohort, and right censoring at the age of stroke (event), death, emigration, or end of follow-up (27 June 2006), whichever came first (event/censoring). All analyses were stratified by gender and calendar-year. Exposure to road traffic noise was modelled as a time-dependent variable using the value of the yearly mean exposure at the residence at a given age.

The incidence rate ratios (IRRs) for stroke in association with road traffic noise at the time of diagnosis were calculated as crude estimates and adjusted for *a priori* defined potential confounders. Information on most potential confounders was based on the baseline questionnaire: smoking status (never, former, current), smoking intensity (g tobacco/day), intake of fruit (g/day), intake of vegetables (g/day), education (<8, 8–10, >10 years), alcohol intake (yes/no; g/day among drinkers), body mass index (BMI, kg/m^2), and physical activity (yes/no; h/week of sport during leisure time among active). The remaining covariates were based on address-specific information on: municipality income (data from 1995; 10th and 90th percentiles of the mean gross income of the 275 Danish municipalities as cut-off points); railway and airport noise [mainly based on data from the late 1990s (railway noise >60 dB (yes/no) and airport noise >55 dB (yes/no))]; and air pollution (NO_x, μg/m^3, data on exposure at the residence in the period from 1993 to 2006). Information on all the selected potential confounders was available for 51 485 of the 53 162 cohort members with complete residential histories. Estimates were calculated both with and without further adjustment for systolic and diastolic blood pressures and use of antihypertensive medicine at the time of enrolment.

We also conducted an analysis in which we calculated the IRRs for stroke in association with road traffic noise at the residential address at the time of enrolment (baseline).

The assumption of linearity of the variables (traffic noise, air pollution, smoking intensity, intake of fruits, intake of vegetables, alcohol consumption, BMI, and blood pressure) in relation to risk for stroke was evaluated both visually and by formal testing with linear spline models with boundaries placed at the nine deciles for cases.[22] Traffic noise, smoking intensity, and diastolic and systolic blood pressures did not deviate significantly from linearity and were entered as linear variables. Air pollution was included as a linear variable after logarithmic transformation. Intake of fruits, intake of vegetables, intake of alcohol, and BMI were included as linear variables after allowance for different slopes below and above 100, 150, and 15 g/day and 24 kg/m^2, respectively. These cut-points were determined by visual examination of the linear spline models, followed by a formal testing of whether we could assume linearity of the variables below and above the cut-points.

In addition to the linear analyses described earlier, we performed a categorical analysis with seven noise exposure categories (55–58, 58–61, 61–64, 64–67, 67–70, 70–73, and >73 dB) and a reference category (≤55 dB). We used 55 dB as the reference because this is often the limit value for noise in outdoor residential areas, and we used exposure categories of 3 dB because this difference corresponds to a doubling in acoustical energy. IRRs above and below 64.5 years of age, corresponding to the median age at stroke diagnosis among the cases, were calculated. IRRs were also calculated separately for men and women and for cases with and without co-morbidity of acute myocardial infarction and of diabetes at the time of the stroke diagnosis.

All tests were based on the likelihood ratio test statistic. Two-sided 95% confidence intervals (CIs) were calculated on the basis of the Wald test of the Cox regression parameter, that is, on the log ratio scale. The procedure PHREG in SAS version 9.1 (SAS Institute, Cary, NC, USA) was used for the statistical analyses.

Results

Out of the study population of 51 485 participants, 1881 (3.7%) were admitted to hospital for stroke for the first time between baseline and censoring, with an average follow-up of 6.0 years among cases and 10.1 among cohort members. Distribution of baseline covariates and air pollution among the cohort according to exposure to road traffic noise below and above 60 dB can be seen in *Table 1*.

The distribution of road traffic noise exposure (L_{den}) at enrolment into the cohort is shown in *Figure 1*. There was a significant correlation between L_{den} and NO_x at enrolment ($r = 0.62$, $P < 0.0001$). During the period from 1993 (inclusion) to 2006 (end of follow-up), there was a small but steady increase over time in L_{den} for the addresses used in this study (approximately 0.5 dB increase per 5 years). For NO_x, the concentration decreased approximately 25% from 1993 to 2000 after which it stabilized.

A 10 dB higher level of road traffic noise was associated with a 1.14 times (95% CI: 1.03–1.25) higher risk for stroke after adjustment for various risk factors (*Table 2*). The association was still present after further adjustment for systolic and diastolic blood pressures and use of antihypertensive medicine at enrolment (IRR: 1.12; 95% CI: 1.01–1.24). For participants above 64.5 years of age, the IRR was 1.23 (95% CI: 1.09–1.39) and for participants below 64.5 years of age, the IRR was 1.01 (95% CI: 0.89–1.15) after adjustment for blood pressure and antihypertensive medicine. Similar IRRs were found for men and women (*Table 2*). There was a significant interaction with age ($P < 0.001$), with a strong association between road traffic noise and stroke among older cases (≥64.5 years) and no association in younger cases. The exposure to noise was similar in participants below and above 64.5 years of age (mean: 57.8 and 58.2 dB, respectively). There were no statistically significant interactions with co-morbidity, neither acute myocardial infarction nor diabetes (*Table 2*). Also, no interactions with co-morbidity were found in participants below or above 64.5 years, respectively (all P for the interactions >0.64, data not shown).

In an analysis in which baseline exposure was used as noise exposure variable, we found an IRR of 1.07 (95% CI: 0.98–1.18) per 10 dB higher noise exposure at the residence at enrolment (adjusted analysis).

Figure 2 shows the IRRs for seven exposure categories in comparison with a reference group of ≤55 dB for participants below and above 64.5 years of age. For the younger participants, there were no associations between road traffic noise and risk for stroke, except in the highest exposure group (>73 dB) in which there seemed to be an association (IRR: 1.48; 95% CI: 0.98–2.24; *Figure 2A*). Among the older participants, there were some indications of an increase in the risk for stroke at road traffic noise levels <60 dB. At exposures >60 dB, the risk for stroke increases in what seemed to be a dose-dependent manner

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Table 1 Baseline characteristics for the Diet, Cancer, and Health cohort according to exposure to road traffic noise below and above 60 dB (L_{den})

Characteristics at enrolment	$L_{den} \leq 60$ ($n = 33\,843$)		$L_{den} > 60$ ($n = 17\,642$)	
	n (%)	Median (5–95th percentiles)	*n* (%)	Median (5–95th percentiles)
Gender				
Male	16 279 (48)		8029 (46)	
Female	17 564 (52)		9613 (54)	
Age (years)		56.1 (50.7–64.1)		56.3 (50.8–64.2)
Smoking				
Current	11 404 (34)		7288 (41)	
g tobacco/day		15.0 (4.8–31.7)		15.3 (5.3–32.3)
Former	9523 (28)		4651 (26)	
Never	12 916 (38)		5703 (32)	
Intake of fruits (g/day)		172 (29–517)		163 (24–537)
Intake of vegetables (g/day)		165 (51–366)		155 (45–369)
Years of education				
≤7	10 624 (31)		6368 (36)	
8–10	15 774 (47)		8080 (46)	
>10	7445 (22)		3194 (18)	
Municipality income				
Low	4414 (13)		6385 (36)	
Medium	20 954 (62)		6756 (38)	
High	8475 (25)		4501 (26)	
Physical activity				
No	14 779 (44)		8813 (50)	
Yes	19 064 (56)		8829 (50)	
Among active (h/week)		2.0 (0.5–6.5)		2.0 (0.5–7.0)
BMI (kg/m^2)		25.4 (20.5–33.1)		25.7 (20.32–33.9)
Drink alcohol				
No	680 (2)		484 (3)	
Yes	33 163 (98)		17 158 (97)	
Among active drinkers (g/day)		13.3 (1.2–62.2)		13.2 (1.0–69.3)
Intake of coffee (cups/day)				
≤1	5646 (17)		3074 (17)	
2–3	9126 (27)		4723 (27)	
4–5	9685 (29)		4819 (27)	
≥6	9386 (28)		5026 (28)	
Air pollution (NO_x, μg/m^3)		18.5 (14.1–28.3)		34.3 (16.9–137)

(*Figure 2B*). At enrolment, 35% of the cohort members lived at addresses with noise levels >60 dB.

We found that a doubling in NO_x was associated with an IRR of 1.04 (95% CI: 0.98–1.10) when adjusted for the selected potential confounders (lifestyle confounders, education, and municipality income) except traffic, railway, and airport noise and an IRR of 0.96 (95% CI: 0.88–1.04) when adjusted by all the selected potential confounders including noise. A 10 dB higher level of road traffic noise was associated with a 1.10 times (95% CI: 1.03–1.18) higher risk for stroke after adjustment for all the selected potential confounders except NO_x.

Exposure to railway and airport noise was not associated with a higher risk for stroke (IRR railway: 1.04; 95% CI: 0.92–1.17 and IRR airport: 0.73; 95% CI: 0.39–1.37). The IRR for stroke mortality (survival 30 days or less after a stroke; 146 of 1881 cases) was 1.09 (95% CI: 0.86–1.39).

Discussion

In this study, residential exposure to road traffic noise was associated with risk for stroke, with a 14% higher risk per 10 dB higher

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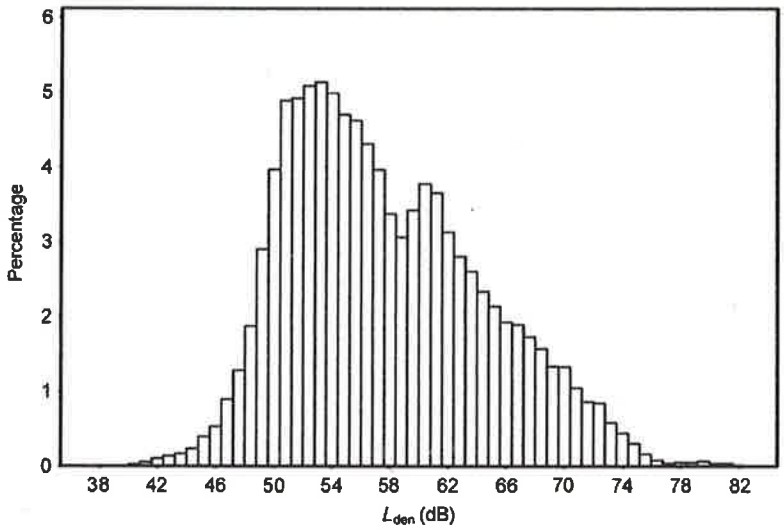

Figure 1 Distribution of residential exposure to road traffic noise (L_{den}) at the time of enrolment into the cohort.

Table 2 Incidence rate ratios (IRRs) of stroke per 10 dB higher level of exposure to road traffic noise

Exposure to road traffic noise, L_{den} (per 10 dB)	No. of cases	Crude IRR (95% CI[a])[b]	*P*-value	Adjusted IRR (95% CI[a])[c]	*P*-value	*P*-interaction
All	1881	1.18 (1.11–1.26)	<0.0001	1.14 (1.03–1.25)	0.008	
Gender						0.96
Male	1109	1.20 (1.10–1.30)	<0.0001	1.14 (1.02–1.27)	0.02	
Female	772	1.17 (1.05–1.29)	0.003	1.13 (1.00–1.28)	0.04	
Age at stroke (years)						0.001
<64.5	952	1.06 (0.97–1.16)	0.22	1.02 (0.91–1.14)	0.77	
≥64.5	929	1.32 (1.20–1.45)	<0.0001	1.27 (1.13–1.43)	<0.0001	
Co-morbidity, acute myocardial infarction[d]						0.80
Yes	143	1.22 (0.97–1.53)	0.10	1.17 (0.92–1.48)	0.21	
No	1738	1.18 (1.10–1.26)	<0.0001	1.13 (1.03–1.24)	0.01	
Co-morbidity, diabetes[d]						0.75
Yes	244	1.20 (1.01–1.43)	0.04	1.16 (0.96–1.40)	0.12	
No	1637	1.17 (1.09–1.26)	<0.0001	1.13 (1.02–1.24)	0.02	

[a]CI, confidence interval.
[b]Stratified by gender.
[c]Stratified by gender and calendar-year and adjusted for lifestyle confounders (smoking status, smoking intensity, intake of fruits, intake of vegetables, intake of coffee, BMI, alcohol intake, and physical activity), education, municipality income, exposure to noise from railways and airports, and exposure to air pollution (NO_x).
[d]A previous diagnosis at the time of the stroke diagnosis.

exposure to noise for all participants and a 27% higher risk per 10 dB higher exposure to noise for participants above 64.5 years.

This is the first study on the association between transport noise and risk for stroke, as previous studies on transport noise focused mainly on hypertension and ischaemic heart disease.[5] Exposure to noise is suspected to cause hypertension and ischaemic heart disease through a stress response, with changes in stress hormones and blood pressure,[1–3] which are also related to the risk for stroke.[9] Our results show that the risk for stroke increases in a dose-dependent manner at exposure levels > 60 dB among the oldest participants. These results are in accordance with the results of a meta-analysis of case–control and cohort studies on road traffic noise and myocardial infarction, which showed that there appeared to be a dose–response relation starting at noise levels > 60 dB.[6]

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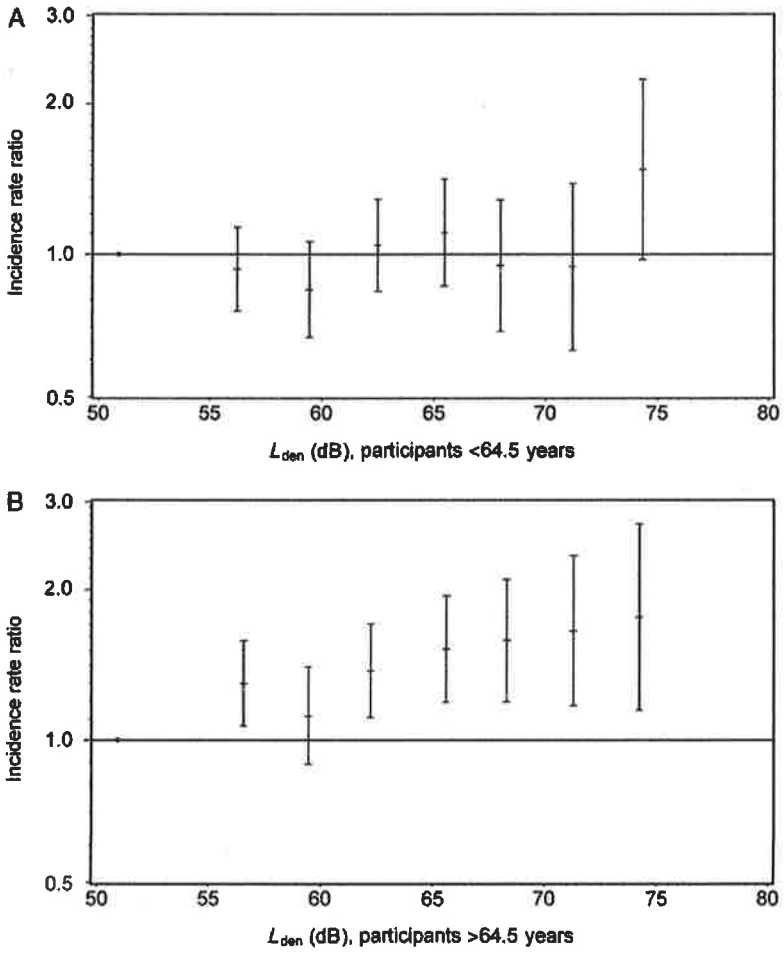

Figure 2 Dose–response relation between exposure to road traffic noise (L_{den}) and incidence rate ratio (IRR) for stroke based on a Cox proportional hazards model with age as the underlying timescale among participants below (*A*) and above (*B*) 64.5 years of age. The analyses were stratified by gender and calendar-year and adjusted for smoking status and intensity, intake of fruits, intake of vegetables, intake of coffee, body mass index, alcohol intake, physical activity, education, municipality income, exposure to noise from railways and airports, and exposure to air pollution (NO_x). The vertical whiskers show the IRRs with 95% confidence intervals at the median of seven exposure categories (55–58, 58–61, 61–64, 64–67, 67–70, 70–73, and >73 dB) when compared with the reference category of ≤55 dB. The black dot shows the median of the reference category. The horizontal solid line shows the neutral value (IRR = 1.0).

This value may therefore be a threshold with regard to both cerebro- and cardiovascular effects of road traffic noise.

Although one of the most important risk factors for stroke is high blood pressure,[9] the association with road traffic noise persisted after adjustment for systolic and diastolic blood pressures and use of antihypertensive medicine, indicating that other pathways are involved in the effect of traffic noise on risk for stroke.

If we assume that the association in the categorical analyses among all participants is causal, an estimated 8% of all stroke cases (19% of stroke cases more than 64.5 years of age) in this population could be attributed to exposure to road traffic noise. The population in this study, however, lived mainly in urban areas and is thus not representative of the whole population in terms of exposure to road traffic noise.

In the present study, we focused on relatively recent exposure as we used annual means of noise at the residences in our main analyses. In another analysis, we found that exposure to noise more distant in time (at enrolment) was associated with a 7% higher risk for stroke per 10 dB higher exposure to noise, which suggests that recent exposures are more strongly associated to stroke than more distant exposure to noise. However, as 72% of the participants in our study did not move during the follow-up period, it is difficult to separate the effect of recent and distant noise exposures, and, thus, exposure to noise more distant in time might also affect the risk for stroke.

The relation between exposure to road traffic noise and risk for stroke was strongest among the oldest participants in the present study. Sleep disturbances can contribute to cerebro- and cardiovascular risks,[23,24] leading to the hypothesis that nocturnal exposure to noise might be more harmful than daytime exposure.[25] The sleep structure generally becomes more fragmented with age, and elderly people are thus more susceptible to sleep

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disturbances.[26,27] This could explain why the association between road traffic noise and risk for stroke was mainly seen for the oldest participants. As exposure to road traffic noise during the night (L_n) was highly correlated with L_{den}, we could not separate the two effects.

The study indicated no association between risk for stroke and noise from railways and airports; however, the risk estimate for exposure to airport noise has a very wide CI (0.39–1.37) probably because <1% of the cohort was exposed. Furthermore, the calculations of railway noise were subject to some degree of uncertainty because of missing information on screening by buildings and noise screens. Thus, we cannot rule out the possibility that these types of noise also affect the risk for stroke.

An important strength of our study is the adjustment for air pollution, which is known to correlate with road traffic noise and, thus, could confound the association between noise and stroke. We used NO_x levels as an indicator of air pollution, because NO_x is a good marker of traffic-related air pollution and correlates closely with particulate matter in Danish streets: $r = 0.93$ for total particle number concentration [size 10–700 nm (ultrafine particles)] and $r = 0.70$ for PM_{10}.[28,29] The role of air pollution as a trigger of stroke following short-term exposures is well established,[30–33] whereas still limited and mixed evidence exists on the effect of long-term exposures to air pollution on the risk of developing stroke.[34,35] We found that exposure to air pollution was not associated with risk for stroke neither before nor after adjustment for traffic noise. Our definition of air pollution exposure as annual-mean NO_x levels at the time of the stroke admission likely represents a mix of short- and long-term effects of air pollution. Exposure to road traffic noise, however, was significantly associated with risk for stroke both before and after adjustment by air pollution, suggesting an independent effect of road traffic noise.

The strengths of our study also include the prospective design, follow-up for stroke in a nationwide register, the large number of cases, and access to residential address histories. Furthermore, we considered only the first hospitalization for stroke, reducing any influence of preventive medication on the risk estimates.

The estimation of noise was based on modelled and not measured values. In addition to practical and economical advantages of the modelling approach in large-scale epidemiological studies, the modelling approach might provide the best noise estimate. The level of traffic noise varies over very short time due to, for example, the movement of vehicles relative to the observer and strong influence of the propagation of traffic noise by weather. It is therefore extremely difficult, if not impossible, to get reliable long-term noise exposure data by use of direct measurements, and during the last four decades, still more accurate and reliable prediction methods for traffic noise have, thus, been developed. However, although the Nordic prediction method has been used for many years, estimation of noise is inevitably associated with some degree of uncertainty. One reason could be inaccurate input data, which would result in exposure misclassification. As the noise model does not distinguish between cases and the cohort, such misclassification is believed to be non-differential, and, in most situations, this would influence the relative risk estimate towards the neutral value. Air pollution levels were calculated with dispersion models that have been successfully validated and applied.[18,20,36] Nevertheless, as for noise, such estimates are associated with some degree of uncertainty; it is possible that some of the effects found for exposure to road traffic noise can be explained by residual confounding from air pollution.

Another limitation is that we had information only on residential addresses and not, for example, work or holiday home addresses. Such an imprecision is, however, believed to have been similar for cases and the cohort and might therefore have attenuated the risk estimates. We also had no information on bedroom location, window opening habits, noise from neighbours, or hearing impairment, all of which might influence exposure to noise. Other studies have found that the association with noise is stronger when these factors are considered,[5,7] suggesting that the effect of noise might be underestimated in the present study.

The stroke cases were identified from a national register instead of from medical records, which is another limitation. Previous validation studies of the register have showed, however, that approximately 80% of persons with a diagnosis of stroke in the Danish National Hospital Registry are confirmed when medical records are reviewed.[37,38] The predictive value of the register is thus high, and we believe that diagnostic errors would have affected the risk estimates only marginally.

In the present study, the analyses were adjusted by various potential lifestyle and socio-economic confounders, as well as by air pollution. We cannot, however, rule out that confounders not accounted for or residual confounding could affect the results. For example, we find a higher proportion of participants with low income among the highly exposed when compared with participants exposed to <60 dB, and socio-economic status has been found to be a predictor for stroke.[39] Therefore, we cannot exclude the possibility of residual confounding by socio-economic differences.

Conclusions

The present study shows a positive association between residential exposure to road traffic noise and risk for stroke in a general Danish population among people older than 64.5 years of age. As this is the first study of its kind, the results need to be confirmed by other studies before any conclusions can be drawn.

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GEOCHEMICAL STUDIES - 1

TRACE ELEMENTS IN ALBERTA CRUDE OILS

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1983-04-30

Alberta Research Council Open File Report 1983-02

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"GEOCHEMICAL STUDIES"

This series of reports, under the general title "Geochemical Studies", *makes available geochemical data on the formation fluids and rocks of Alberta which would otherwise remain unpublished. Two types of data fall in this category. First,* "Geochemical Studies" *will act effectively as a document desposi-tory in cases where a formal publication is available, but without the raw data having been published; addi-tional interpretations may be included if pertinent. Second,* "Geochemical Studies" *will include both the raw data and a minimal descriptive report in the case where no formal publication is planned.*

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ABSTRACT

Twenty-two trace elements, determined by neutron activation techniques, are reported for 86 conventional crude oils and two crude oils from the Athabasca oil sand deposit, Alberta. When these crude oils are grouped into three families, based on previously studied hydrocarbon characteristics, analysis of variance indicates that S, Se, V, Ni, Br, Co and Zn can be used to differentiate, at a 90 percent confidence level, at least one of the families. These elements may therefore be an additional basis for distinguishing the crude oil families. Further study using multivariate methods are justified.

It is suggested, tentatively, that crude oils with alkaline associated formation waters may be relatively depleted in Na, when compared to those with acidic associated formation waters. The contents of both Zn and V in crude oils may be related to the content of these elements in the source rocks. A geochemical model is presented for the occurrence of Br and I in formation waters and I in crude oils. These three geochemical observations, as well as the possible use of trace elements for the classification of crude oils into families, may be of value to explorationists and more study is justified.

INTRODUCTION

Trace elements have been known to be present in crude oils for many decades. Although the amounts found are generally small their impact on successful industrial processing is immense, especially with respect to petroleum refining and environmental concerns. In addition, they play a role in elucidating the origin of petroleum. Early studies on trace elements in Alberta crude oils comprise mainly the publications of Hodgson and his co-workers (Scott et al., 1954; Hodgson 1954; Hodgson and Baker, 1957; Baker and Hodgson, 1959). These dealt mainly with the trace elements V, Ni and Mg.

The more recent work of Hitchon et al. (1975) represents the first published account of a multi-element study of trace elements in Alberta crude oils. Their study reported the general statistics for 22 elements in 86 conventional crude oils and two crude oils from the Athabasca oil sand depost. Eleven elements (S, V, Cl, Na, Ni, Zn, Co, Mn, Se, Br and As) were studied by both Q-mode and R-mode statistical factor analysis. A separate R-mode analysis using the biquartimin solution was provided for V, Fe, Ni, Co, Mn and Cr in 20 selected crude oils. Recognizing the limitations imposed by the determination of trace elements in the total crude oil, when their content is sometimes essentially confined to a variable fraction such as the asphaltenes, Hitchon et al. (1975) concluded that S, V, Cl, Na, Ni, Zn, Co, Mn, Se, Br and As are controlled by maturation processes rather than migration processes, though this control may simply reflect maturation of the asphaltene fraction of the crude oils. More than one quarter of the cumulative variance is accounted for by S, V, Se and Ni, representing metalorganic complexes. The factor analyses further demonstrated not only the efficacy of their separation system between crude oil and entrained formation water, but also that it is most unlikely that the Fe present in the crude oils originates from corrosion products incorporated during production. Four elements, Fe, Mn, As and Co represent essentially unique factors.

Additional analytical data on the same suite of crude oils studied by Hitchon et al. (1975) can be found in Elofson et al. (1977), including the ESR properties of the crude oils, the percent asphaltenes, and both the ESR properties and elemental composition of the asphaltenes. The molecular weight (in benzene) of the asphaltenes was reported for selected samples.

This Open File Report will act effectively as a document depository for the raw data used by Hitchon et al. (1975). Additional analytical information on the same suite of crude oils is also provided. Hitchon et al. (1975) considered the 86 conventional crude oils and two crude oils from the Athabasca oil sand deposit as a single population, based on the results of the Q-mode analysis, and found maturation to be the controlling process for eleven elements. Additional information may be derived from the same data if the conventional crude oils are divided into the three major crude oil families distinguished by Deroo et al. (1977), and

the trace elements compared between families. For reasons to be explained elsewhere, the two crude oils from the Athabasca oil sand deposit are treated separately.

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TRACE ELEMENTS IN ALBERTA CRUDE OILS - DATA SHEETS

The raw data for the 22 elements in 86 conventional crude oils and two crude oils from the Athabasca oil sand deposit studied by Hitchon et al. (1975) is found in the Appendix. Also included is information on the field, pool, stratigraphic unit, well name and location, source of the sample and method of production, and the date sampled. The "Remarks" section of the data sheet shows any unusual features of the sample. For each stratigraphic unit a map is provided showing the distribution of crude oils sampled from that unit. All sample numbers correspond with those published in Elofson et al. (1977).

ADDITIONAL ANALYTICAL INFORMATION

The Kinematic viscosity at 70°F (21.1°C) and 100°F (37.8°C), and the Ramsbottom carbon residue were determined on most of the crude oils reported in the Appendix, and this data is presented in Table 1 without further comment. In addition to the determination of V in the crude oils by neutron activation (see Appendix), tetravalent V (V^{4+}) was determined on many of the same samples by ESR techniques; this data is also shown in Table 1 and will be considered briefly elsewhere in this report.

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Table 1. Viscosity, carbon residue and V^{4+} for 88 Alberta crude oils

SAMPLE NUMBER	KINEMATIC VISCOSITY (Centistokes) 70°F	100°F	RAMSBOTTOM CARBON RESIDUE (wt %)	V^{4+} (BY ESR) (µg/g)
1	6.44	3.64	1.72	*
2	15.2	6.85	2.54	*
3	10.3	4.07	2.11	*
4	5.32	3.53	0.70	*
5	17.4	8.43	1.89	4.6
6	3.44	2.41	0.29	*
8	2.15	1.68	0.32	*
9	14.6	-	1.76	0.25
10	6.65	4.23	0.66	*
11	0.53	-	0.08	*
12	23.7	11.2	-	6.2
14	36.2	14.8	-	3.1
15	9.4	5.58	1.38	*
16	13.6	7.47	1.17	*
17	6.6	4.69	0.74	*
18	9.85	5.97	-	*
19	4.73	3.35	-	*
20	10.6	-	1.10	*
21	6.27	4.26	0.59	*
22	9.90	5.86	-	0.12
24	241	-	7.22	108
26	165	57.5	6.65	82
27	8.94	5.78	1.65	2.7
28	-	-	3.05	8.1
29	12.3	-	2.34	4.4
30	37.2	-	4.32	26
31	6.00	4.10	0.98	*
32	8.58	5.26	2.21	2.5
33	2.89	2.72	0.30	*
34	29.3	15.8	4.68	28.9
35	59.3	-	4.75	29.3
36	-	-	4.31	15.9
37	0.91	0.71	0.05	*
38	4.50	3.23	1.30	*
39	86.6	23.1	4.43	23.6

(Table continued)

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Table 1. (continued)

SAMPLE NUMBER	KINEMATIC VISCOSITY (Centistokes) 70°F	100°F	RAMSBOTTOM CARBON RESIDUE (wt %)	V^{4+} (BY ESR) (µg/g)
40	5.49	3.79	2.30	5.3
41	23.4	13.3	4.64	37.6
42	3.82	2.72	0.42	*
43	-	-	-	279
94	-	-	12.6	-
44	-	-	5.21	39.6
45	33.3	-	5.22	33.5
46	77.8	36.6	7.67	88.4
47	38.8	8.48	3.25	35.1
48	6.88	4.48	1.85	15.9
49	7.76	5.84	2.35	18.7
50	6.16	4.19	0.74	0.17
52	-	-	1.01	1.4
53	4.57	3.34	1.51	1.15
54	10.3	5.96	1.24	*
55	4.03	2.86	0.58	*
56	4.91	3.44	0.98	*
57	38.0	18.2	4.14	14.4
58	13.2	-	4.02	12.2
59	4980	1070	-	211
60	35.8	12.8	2.61	5.1
61	2.46	2.09	0.44	*
62	4.75	3.30	1.37	1.1
63	4.98	3.41	1.05	*
64	1040	270	7.62	61.2
65	3.20	2.38	0.58	*
66	23.9	11.2	3.83	9.1
67	8.98	5.73	3.76	26
68	6.99	4.61	2.01	1.1
69	4.36	3.07	1.48	1.3
70	6.88	4.48	0.98	0.2
71	-	-	-	*
72	1.60	1.28	0.30	*
73	-	-	3.13	8.3
74	11.0	6.77	3.94	6.3

(Table continued)

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Table 1. (continued)

SAMPLE NUMBER	KINEMATIC VISCOSITY (Centistokes) 70°F	100°F	RAMSBOTTOM CARBON RESIDUE (wt %)	V^{4+} (BY ESR) (µg/g)
75	22.2	8.88	1.56	*
76	8.71	4.91	1.97	*
77	4.12	2.99	0.86	*
78	3.56	2.57	0.79	*
79	6.49	4.25	1.54	0.75
80	3.04	2.24	0.46	*
81	20.8	11.3	5.01	5.4
82	-	-	-	-
83	6.22	4.19	2.65	1.1
84	4.29	2.93	1.19	*
85	5.56	3.76	1.17	*
86	4.12	2.70	0.66	*
87	42.4	15.1	3.63	5.2
88	-	-	-	7.4
89	11.7	5.61	4.38	0.9
90	4.86	3.37	1.92	*
91	9.31	5.05	2.33	*
93	5.34	3.71	2.51	0.98

Note: Samples in same order as in the Appendix
* = Below detection
- = Not determined

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GEOCHEMISTRY OF TRACE ELEMENTS IN ALBERTA CRUDE OILS

INTRODUCTION

Based on a very comprehensive study, Deroo et al. (1977) distinguished three crude oil families in Alberta. The most important criteria are summarized next.

Group 1 (Upper Cretaceous Colorado and post-Colorado reservoirs)
These crude oils have the lowest contents of sulphur and aromatic hydrocarbons, are richest in saturated hydrocarbons, acyclic alkanes and acyclic isoprenoids but have n-alkane distributions similar to those of Group 2. They can be distinguished from the Group 3 crude oils only by the lower proportion of cyclo-alkanes with one and two rings, and also on the basis of their benzothiophene content, which is small and limited to the lower carbon number range.

Group 2 (Lower Cretaceous Mannville Group, Jurassic, Carboniferous and Upper Devonian Wabamun Group reservoirs)
These crude oils are distinguished clearly from those of the other two groups on the basis of gross composition and sulphur content. They are richest in sulphur and aromatics but are the poorest in saturated hydrocarbons, acyclic alkanes, and acyclic isoprenoids relative to n-alkanes. Their very high sulphur contents are attributed to large amounts of benzothiophenes and dibenzothiophenes, with the latter having a wide carbon number range (C_{21} to C_{30}).

Group 3 (Upper Devonian Winterburn and Woodbend Groups and Beaverhill Lake Formation reservoirs)
These crude oils contain a higher proportion of thiophenic compounds than Group 1 and they have a smooth distribution over a wide range of carbon numbers. The crude oils from Beaverhill Lake Formation reservoirs form Group 3b and generally contain a smaller proportion of thiophenic compounds than those in Group 3a from the overlying Woodbend and Winterburn Groups.

Deroo et al. (1977) noted some exceptions to this classification but because their exceptions are not among the crude oils studied in this report no further comment will be made on any exceptions to their broad classification given above.

Preliminary appraisal of the trace element data for the 86 conventional crude oils analyzed indicated that when they were grouped into the three families distinguished by Deroo et al. (1977) some of the trace element distribution trends might be of value for classification purposes. It was also observed that the two crude oils from the Athabasca oil sand deposit (Nos. 43 and 94) exhibited trace element content characteristics

markedly different from the Group 2 crude oils, even though they appear to be correctly and unequivocally part of that group. The strict conditions under which all the conventional crude oils were collected in the field, and their preliminary laboratory treatment are outlined by Hitchon et al. (1975); following the preliminary laboratory treatment they were further cleaned-up, as described by Filby and Shah (1975). The two crude oils from the Athabasca oil sand deposit had first to be extracted from the oil sand in a Soxhlet extractor with benzene, centrifuged and evaporated down, and even with extreme care, the possibility of contamination from entrained fine-grained mineral matter cannot be ruled out. For this reason, the two crude oils from oil sands are treated separately from the conventional crude oils in this report.

On the basis of the report of Deroo et al. (1977) the conventional crude oils analyzed in this study may be classified as follows:

GROUP 1:

U. Cretaceous, post-Colorado Supergroup, Belly River Fm. (Nos. 1, 2, 3 and 4)
U. Cretaceous, U. Colorado Gp., Cardium Fm. (Nos. 5, 6, 8 and 9)
U. Cretaceous, U. Colorado Gp., other (Nos. 10, 11 and 12)
U. Cretaceous, L. Colorado Gp., Viking Fm. (Nos. 14, 15, 16, 17, 18, 19, 20, 21 and 22)

GROUP 2:

U. Cretaceous, L. Colorado Gp., Basal Colorado Ss. (No. 24)
L. Cretaceous, Mannville Gp., (Nos. 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40 and 41)
Jurassic (Nos. 44, 45 and 46)
Carboniferous (Nos. 54, 56, 57, 58 and 59)

GROUP 3:

U. Devonian, Wabamun Gp. (Nos. 60 and 61)
U. Devonian, Winterburn Gp. (Nos. 62, 63, 64, 65, 66 and 67)
U. Devonian, Woodbend Gp. (Nos. 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81 and 82)
U. Devonian, Beaverhill Lake and Slave Point Fms. (Nos. 83, 84, 85 and 86)
M. Devonian, Elk Point Gp. (Nos. 87, 88, 89, 90 and 91)
Granite Wash (No. 93)

The total number of samples in Groups 1, 2 and 3 are 20, 25 and 33, respectively. For some of the samples, the group classification in this report differs from that of Deroo et al. (1977). They did not analyze crude oils from the Basal Colorado sandstone, but preliminary and subsequent study shows, quite clearly, that sample No. 24 is correctly classified with Group 2 and not Group 1. All samples from Carboniferous and Upper Devonian Wabamun Group reservoirs studied by Deroo et al, (1977) were from situations close to, or immediately underlying, the pre-Cretaceous unconformity, and hence were classified under Group 2. In

this study, two samples from Carboniferous strata (Nos. 52 and 53), and two from the U. Devonian Wabamun Group (Nos. 60 and 61) were situated far below the pre-Cretaceous unconformity. In the absence of definitive data on the hydrocarbon content of samples 52 and 53 they were omitted from this study, and samples 60 and 61 were included, as it turned out correctly, in Group 3. The Turner Valley field lies in the Disturbed Belt and was sampled from both the Lower Cretaceous Blairmore Group and Carboniferous reservoirs (samples 42 and 55, respectively). Because of the structural separation and the fact that both crude oils showed trace element characteristics unlike those of the other Group 2 crude oils they were not used in this study. No Triassic crude oils were analyzed by Deroo et al. (1977), and because samples 47, 48, 49 and 50 showed ambiguous trace element characteristics they also were not used in this study. Where pertinent, an attempt is made to classify those crude oils excluded from this study.

Hitchon et al. (1975) concluded that for their total population of 88 crude oils, eleven trace elements were controlled by maturation processes. It is therefore important to know, for at least these eleven trace elements, whether the grouping of crude oils according to the distinguishing criteria of Deroo et al. (1977) will also reflect a maturation trend. In the absence of data on the maximum temperature to which each reservoir has been subjected the present reservoir temperature can serve as a guide to the degree of thermal maturation. Based on published data from the Alberta Energy Resources Conservation Board the following general statistics can be calculated:

Present reservoir temperature (°C)

	Minimum	Median	Average	Maximum	Range
Group 1 crude oil reservoirs	36	59	56	89	53
Group 2 crude oil reservoirs	24	64	57	92	68
Group 3 crude oil reservoirs	34	66	70	110	76

These statistics suggest that although, on average, the Group 3 crude oil reservoirs are 15°C hotter than crude oil reservoirs in the two other groups, and there is an increase in both the range (23°C) and maximum temperature (21°C) between Group 1 and Group 3 reservoirs, any significant differences in trace element characteristics between the three groups of crude oils can be considered in terms of differences in source or of different processes operative after the crude oils were accumulated, bearing in mind that both source differences and processes can be affected by temperature differences. In simpler terms, the range in temperature of the reservoirs within each group is so large that the trace element characteristics within each group should exhibit the maturation trends demonstrated by Hitchon et al. (1975), but differences

in the trace element characteristics among the groups should reflect processes (other than maturation) or source differences.

As noted in the Introduction, Hitchon et al. (1975), thought that the control exerted by maturation processes on the eleven trace elements they studied may simply reflect maturation of the asphaltene fraction of the crude oils. As indicated in the previous paragraph, the temperature range of the reservoirs within each group of crude oils is so large that each group should exhibit a similar suite of maturation criteria, including similar statistics for the content of asphaltenes within each group. Based on the percent asphaltenes for these crude oils published by Elofson et al. (1977) the following general statistics can be calculated:

Asphaltene content (% w/w)

	Number of samples	Number of samples with no detectable asphaltenes (%)	Median (all samples)	Average (all samples)	Maximum
Group 1 crude oils	20	11 (55)	0.0	0.34	2.8
Group 2 crude oils	25	2 (8)	2.6	3.23	15.5
Group 3 crude oils	32	3 (9)	0.46	1.83	14.6

These statistics show that Group 1 crude oils are generally deficient in asphaltenes, in contrast to Groups 2 and 3, which have similar high average contents of asphaltenes and a much wider range, by almost an order of magnitude compared to those in Group 1. This suggests that differences in trace element characteristics between the groups, especially between Groups 2 and 3, should reflect source differences or differences in processes operative after the crude oils accumulated.

ANALYSIS OF VARIANCE

Introduction

Analysis of variance (ANOVA) is a univariate statistical technique that compares the variance of a variable (trace element content in this study) within each population (Groups 1, 2 and 3, as designated previously) with the variance between the populations to determine if the distribution of that variable is significantly different in one or more of the populations. The level of significance is obtained from a statistical term called the F statistic. Because ANOVA is a univariate method, it considers only one variable at a time, but is of value in such a study as this because it enables the selection, through examination of the F statistic, of only those variables which are statistically significant at a chosen level, and which can then be examined by a multivariate method if so desired.

Dummy Values

A persistent problem in statistical analysis is the lack of complete sets of variables for all samples studied. Among the reasons for incomplete sets of variables are non-determination of some variables in some samples for a variety of reasons, and variables below detection limits; the latter is especially pertinent for geochemical data. As far as the authors are aware, there are no rigid criteria which can be followed respecting the substitution of dummy values to fill "data holes", and much depends on the experience of the operator. In the past, the senior author has either limited statistical analysis to complete data sets where a large number of samples were available (Hitchon and Horn, 1974), or arbitrarily substituted a value of one tenth the detection limit where the number of samples was more limited and less than ten percent of the samples for any variable required the substitution of a dummy value (Hitchon et al., 1971).

In the present study, the problem of dummy values was approached in two ways. First, for crude oils from the same stratigraphic unit where the regional distribution of samples was relatively close, an isoconcentration map for the pertinent trace element was drawn and the best value based on concentration gradients was used as a dummy value. Second, where the sample was relatively isolated with respect to other samples from the same stratigraphic unit, an arbitrary value of one tenth the detection limit was substituted for the missing data. A further general restriction placed on the selection of samples for statistical analysis was that no more than one dummy value could be used in each sample; this criterion was relaxed for 12 of the samples studied in which an additional dummy value was allowed provided the other dummy value was from a pool in the same field or from an immediately contiguous field in the same stratigraphic unit. The only exception to this last restriction was the sample from the Rundle Group at Turner Valley, where four dummy values were used because of the desire to include this sample in the ungrouped population. As a result of all the above restrictions, 75 crude oils were subject to statistical analysis, with eleven variables each.

Logarithmic Transforms

One of the restrictions to the use of ANOVA is that the variable in each population be normally distributed. Because many geochemical data approximate a lognormal distribution, the eleven variables were tested for normality, within groups, using both the raw data and their logarithmic transforms. Ten exhibited a lognormal distribution and Mn a normal distribution, and it is these data which were used in the ANOVA, the results of which are shown in Figure 1.

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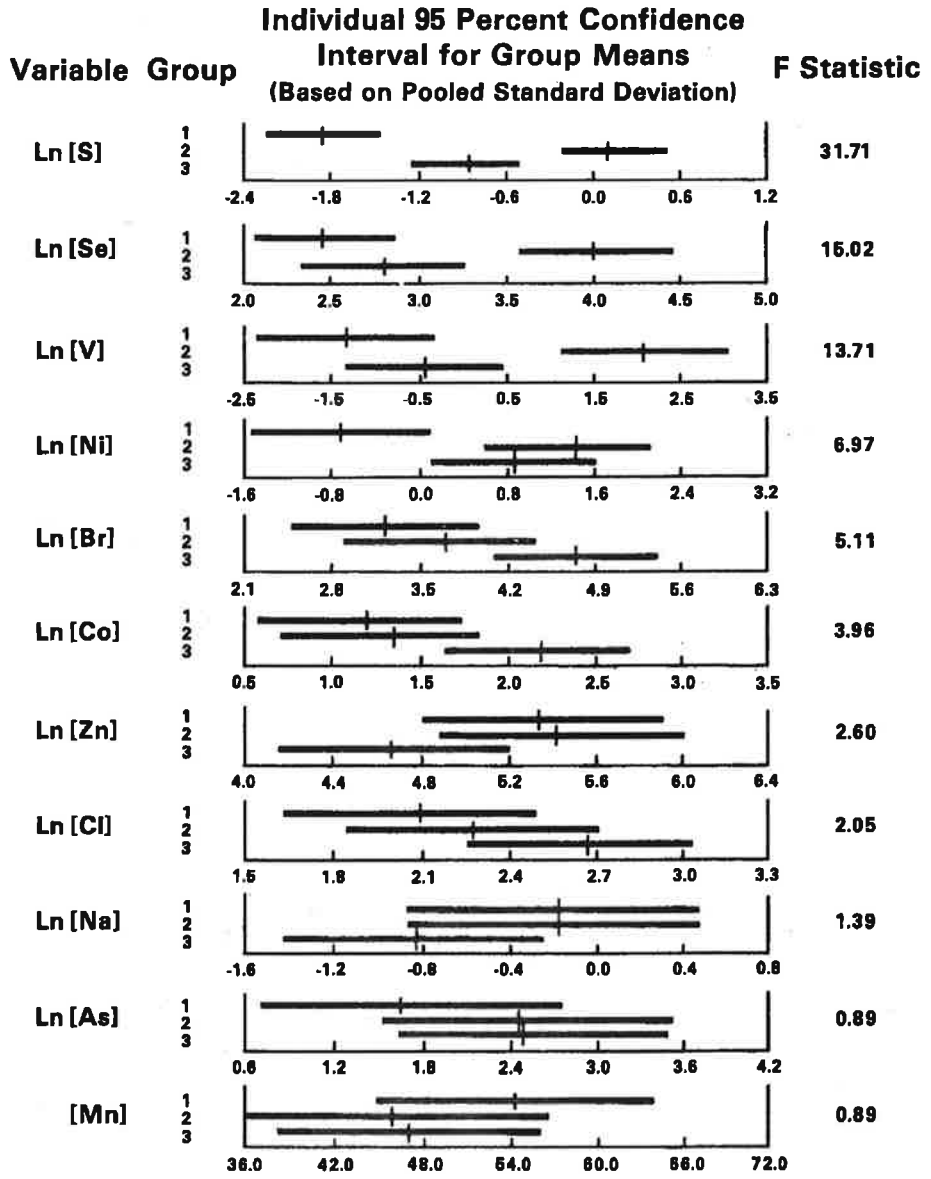

Figure 1. Bar charts showing 95 percent confidence interval, based on ANOVA for Alberta crude oils grouped according to the crude oil families distinguished by Deroo et al. (1977).

Results

For the data analyzed, values of the F statistic greater than about 2.5 are significant at the 90 percent level. Thus, for the logarithmic transforms of S, Se, V, Ni, Br, Co and Zn we can be more than 90 percent certain that there is a difference in the content of these elements in at least one of the groups. The bar charts in Figure 1 illustrate this point. It must be emphasized, however, that because three populations were studied, not all of these seven elements are, a priori, discriminators between the three crude oil families; only a multivariate statistical technique such as discriminant function analysis is capable of solving the intergroup correlation problem.

ALKALI METALS – SODIUM, RUBIDIUM AND CESIUM

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Na (ppm)	20	0.046	0.73	1.54	13.0
Rb (ppb)	3	1.870	5.43	24.9	67.5
Cs (ppb)	12	0.310	0.88	1.05	2.27
Group 2 crude oils (n=25)					
Na (ppm)	22	0.018	0.83	2.94	19.3
Rb (ppb)	2	28.1	43.0	43.0	57.8
Cs (ppb)	13	0.32	1.395	1.80	9.03
Group 3 crude oils (n=33)					
Na (ppm)	33	0.0174	0.632	4.61	64.7
Rb (ppb)	2	15.58	37.8	37.8	60.0
Cs (ppb)	13	0.641	1.71	3.78	19.45

Geochemistry

Effectively all samples contained Na, although the concentration range was wide (0.0174 to 64.7 ppm). Rubidium was detected in 9 percent of the samples and Cs in nearly 50 percent of the samples. In every sample in which all three alkali metals were detected, the order was always Na >> Rb > Cs, with the general proportions being 1000:10:1, respectively (in ppb). In 24 of the fields and pools, the co-produced formation water had been analyzed previously by Hitchon et al. (1971), including the determination of Na and Rb. Even if all the Rb in the associated crude oils had originated from entrained formation water, calculations show that it would be below detection in 17 of the crude oils and close to detection in the remainder. Therefore, the Rb contents reported in

the crude oils are not due to contamination by entrained formation water. Hitchon et al. (1975) have also shown that Na is unrelated to Cl, and therefore concluded that their sample preparation technique was effective in removing entrained formation water. With respect to relations of the alkali metals among the three groups of crude oils, although there are systematic changes in the average and maximum contents of both Na and Cs from Group 1 to Group 3 the ranges of concentration between the groups are so similar that neither of these alkali metals can be used to distinguish the crude oil groups. There are insufficient determinations of Rb to make a meaningful comment. The low value of the F-statistic for Na in the ANOVA confirms this observation.

Based on experiments with a Tertiary crude oil from California, Filby (1975) showed that 92 percent of the Na in that oil is present as an oil-soluble compound that is also water soluble or is hydrolyzed, perhaps as the Na salt of a petroleum acid. For the same 24 fields and pools cited in the previous paragraph, the pH of the co-produced formation water was determined in the field, and therefore as close to reservoir temperature as possible and with minimal loss of dissolved gases. The data on pH of the co-produced formation water and Na in the associated crude oil may be grouped as follows:

Number of samples	Formation water	Na in crude oil (ppm)		
		Minimum	Average	Maximum
10	pH > 7.0	0.13	4.1	19.3
14	pH < 7.0	0.02	5.8	57.8

If the Na in crude oils occurs as the Na salt of a petroleum acid then, in general, alkaline formation waters (pH > 7.0) should be relatively enriched in these Na organic salts, which would preferentially pass into the aqueous phase, and the crude oil relatively depleted. The grouped data above seem to support the suggestion of Filby (1975). Work carried out some years ago at the Alberta Research Council on the surface tension of formation waters showed a wide range in this property; formation waters with very low surface tensions and correspondingly high pH values also contained the largest amounts of salts of petroleum acids. Figure 2 shows the results of a portion of this work on surface tension in formation waters, with the data grouped on the basis of the study of Deroo et al. (1977). There is a wide range in surface tension within each group although it is not possible to distinguish the three groups on the basis of surface tension. The genetic relations among surface tension, pH and surfactants (such as Na salts of petroleum acids) in formation waters, and the content and form of bonding of Na in their associated crude oils justifies considerably more study. The results of such a

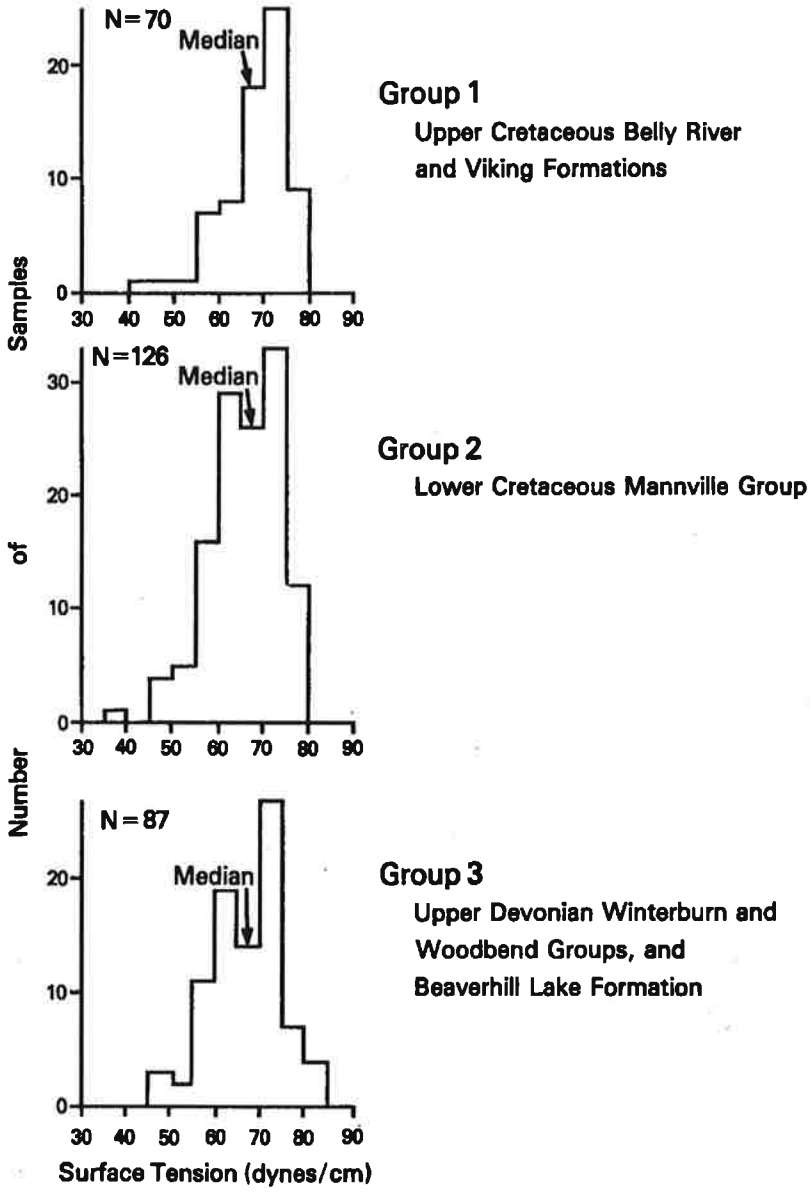

Figure 2. Histograms of frequency distribution of surface tension in formation waters from Alberta grouped according to the crude oil families distinguished by Deroo et al. (1977).

study have the potential of being of value to explorationists through the use of the salts of petroleum acids in formation waters as indicators for crude oil occurrence.

GOLD

Gold was detected in only three conventional crude oils (samples 12, 73 and 74) at levels of 0.078, 0.149 and 0.204 ppb, respectively. These crude oils appear to have no common characteristic and so this data on gold in Alberta crude oils is presented without further comment.

ZINC AND MERCURY

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Zn (ppb)	20	11.51	270	272	669.7
Hg (ppb)	11	1.60	8.05	32.6	202.2
Group 2 crude oils (n=25)					
Zn (ppb)	24	17.5	401	456	1277.0
Hg (ppb)	9	1.93	6.69	27.6	138.3
Group 3 crude oils (n=33)					
Zn (ppb)	28	13.40	140	573	5921.0
Hg (ppb)	15	1.34	6.57	85.6	398.6

Geochemistry

Zinc was detected in more than 90 percent of the crude oils and Hg in about 45 percent of the samples. Thirty-seven crude oils contained detectable amounts of both Zn and Hg, and in only three (Nos. 19, 31 and 77) was Hg > Zn; these three appear to have no other common feature, though it can be noted that sample 31 from the Lower Cretaceous Basal Quartz B pool at Niton was singled out by Deroo et al. (1977) as being unique in having a large content of heavy n-alkanes, which is characteristic of crude oils derived from nonmarine organic matter. The Zn/Hg ratio ranged from 0.127 to 406, with the average proportion of Zn:Hg being about 8:1. With respect to the relations of Zn and Hg among the three groups of crude oils, although there are systematic changes in the average and maximum contents of Zn from Group 1 to Group 3, and the statistics for Hg show that Groups 1 and 2 are similar, with Group 3 crude oils containing on average more than twice as much Hg as the first two groups, the ranges of concentration for both elements do not appear to show sufficiently significant differences to allow the use of Zn and Hg to distinguish the three crude oil groups. A value of 2.6 for the F

statistic for Zn in the ANOVA indicates that Zn may be marginally useful in crude oil correlation, however; only a multivariate analysis can confirm this indication. Within each group the Zn/Hg ratios exhibit similar ranges, but as can be expected from the above general statistics for the individual elements, the average Zn/Hg ratio for Groups 1 and 3 are similar (about 50), with Group 2 crude oils having a much higher average Zn/Hg ratio (130). Any suggestion that this higher Zn/Hg ratio in the extensively biodegraded crude oils of Group 2 is related to the biodegradation, can only be regarded as speculative at this time.

Hitchon (1977) has discussed the geochemical links between oil fields and ore deposits in sedimentary rocks with special reference to Alberta, and drew an analogy between the features of high salinity and high metal content of geothermal brines and the deeper, hotter, more saline formation waters from Alberta, which have leached evaporites to produce some of the solutes, possibly assisted by membrane filtration, and shales to produce the enhanced content of metals. A further analogy was developed between the generation and leaching of hydrocarbons from shale source rocks, and their subsequent migration and accumulation as oil fields, and the leaching of shales by hot, saline formation waters, with their subsequent migration and accumulation as ore deposits. This analogy is very well illustrated by the statistics presented in Table 2, in which data on Zn and Cl in formation waters from Alberta (Hitchon et al. (1971), Zn and Hg in crude oils (this report) and Zn in some Alberta shales (Hitchon, in press) is grouped according to the crude oil families distinguished by Deroo et al. (1977). In broad terms, the statistics in Table 2 may be summarized as follows:

	Group 1	Group 2	Group 3
Zn in shales	High	Intermediate	Low
Cl in formation waters	Low	Intermediate	High
Zn in formation waters	Low	Low	High
Zn in crude oils	Low	Intermediate	High
Hg in crude oils	Low	Low	High

This summary supports the thesis that the Zn in crude oils, and probably also Hg, as well as Zn in formation waters, have originated from shales which yielded the crude oil hydrocarbons through the specific action of hot, chloride-rich formation waters, with the consequent depletion of those shales in Zn (and probably also Hg).

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Table 2. Statistics for Zn and Cl in formation waters, Zn and Hg in crude oils, and Zn in shales, Alberta, grouped according to the crude oil families distinguished by Deroo et al. (1977)

		GROUP 1 U. CRETACEOUS	GROUP 2 L. CRETACEOUS, JURASSIC AND CARBONIFEROUS	GROUP 3 U. AND M. DEVONIAN
FORMATION WATERS				
No. of samples		15	25	34
Zn (mg/ℓ)	min.	0.00	0.03	0.05
	avg.	0.36	0.32	1.43
	max.	3.05	3.05	27.5
Cl (mg/ℓ)	min.	798	320	15 800
	avg.	12 200	25 700	69 700
	max.	38 200	95 800	172 000
CRUDE OILS				
No. of samples		20	24	28
Zn (ppb)	min.	11.51	17.5	13.40
	avg.	272	456	573
	max.	669.7	1277.0	5921.0
No. of samples		11	9	15
Hg (ppb)	min.	1.60	1.73	1.34
	avg.	32.6	27.6	85.6
	max.	202.2	138.3	398.6
SHALES				
No. of samples		7	12	7
Zn (ppm)	min.	99	65	19
	avg.	270	132	61
	max.	943	261	100

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SCANDIUM, EUROPIUM AND GALLIUM

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Sc (ppb)	8	0.013	0.13	0.126	0.241
Eu (ppb)	13	0.073	0.305	0.270	0.641
Ga (ppb)	1	-	1.13	-	-
Group 2 crude oils (n=25)					
Sc (ppb)	14	0.020	0.16	0.185	0.764
Eu (ppb)	16	0.050	0.40	0.373	0.661
Ga (ppb)	2	5.7	8.35	8.35	11.0
Group 3 crude oils (n=33)					
Sc (ppb)	23	0.014	0.094	0.090	0.178
Eu (ppb)	15	0.091	0.255	0.281	0.604
Ga (ppb)	13	0.07	15.82	63.4	445.0

Geochemistry

Scandium and Eu were each detected in nearly 60 percent of the crude oils, and Ga was detected in 20 percent of the samples. Of the 86 conventional crude oils analyzed, only 24 reported both Sc and Eu, in 22 of which Sc > Eu (Sc/Eu ratio ranged from 0.035 to 0.774). The similarities between the statistics for Sc and Eu among the three groups of crude oils means that these two elements cannot be used to distinguish between the groups. It is possible that the determination Al in crude oils could be used to indicate the degree of contamination by entrained clay minerals, and in the absence of data on the Al content, Ga contents might be substituted as indicators of clay mineral contamination. One would expect the most viscous crude oils, which are the most difficult to clean-up, to contain the highest contents of Ga (or Al, if that element was determined). If the viscosity data in Table 1 are grouped and treated statistically like the trace element data no relation is found between the statistical characteristics of viscosity and Ga. This finding is equivocal, but could be interpreted in terms of satisfactory procedures for the elimination of clay mineral contamination, and that Ga occurs only in the form of organic associations. This latter suggestion is supported by recent studies by the junior author.

ARSENIC AND ANTIMONY

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
As (ppb)	15	0.610	3.694	11.4	56.72
Sb (ppb)	6	0.10	0.25	0.343	0.810
Group 2 crude oils (n=25)					
As (ppb)	19	0.020	9.9	196	1988.0
Sb (ppb)	6	0.530	2.4	5.42	22.8
Group 3 crude oils (n=33)					
As (ppb)	28	0.63	12.8	104	1219.0
Sb (ppb)	9	0.190	2.55	6.15	34.8

Geochemistry

Arsenic was detected in 80 percent of the conventional crude oils, and Sb in 25 percent. Only 19 samples (22 percent) reported both As and Sb, which exhibited a random distribution when plotted together (correlation coefficient < 0.001). With respect to the relations of As and Sb among the three groups of crude oils, the Group 1 crude oils are distinct in having a much narrower range in the content of both elements than Groups 2 and 3, with an average content of As an order of magnitude less, and Sb about one fifteenth that of the other groups. Despite this, the ANOVA shows that As cannot be used in crude oil correlation. Study of As and Sb in individual samples may still be of interest, however; for example, sample No. 24 from the Basal Colorado sandstone, with As 79.8 ppb and Sb 1.07 ppb, is clearly a Group 2 crude oil, even though it occurs in a reservoir classified by Deroo et al. (1977) as belonging to Group 1. In this instance, migration of the crude oil from the underlying Mannville Group is strongly indicated. Group 2 crude oils cannot be distinguished, unequivocally, from Group 3 crude oils even though their average content of As is twice that of those in Group 3.

Filby (1975) has suggested that in crude oils, As and Sb appear to be present partly as low-molecular-weight compounds such as alkyl or aryl arsines and stibines, and that in the case of Sb, these compounds are strongly associated with the asphaltenes. As noted in the introduction to this section of the report, Group 1 crude oils are generally deficient in asphaltenes, in contrast to Groups 2 and 3, which have similar high average contents of asphaltenes and a much wider range, by almost an order of magnitude compared to those in Group 1. It seems likely, therefore, that the differences in the statistical distribution of As, and particularly Sb, between the crude oil groups is directly related to

their differences in asphaltene content, and also supports the suggestion of Filby (1975) on the occurrence and association of these elements in crude oils. Recent work by the junior author on As in oil sand bitumen supports the theory of As occurring in small modelcules; Sb behaves quite differently from As, however. In the study of Hitchon et al. (1975) As represented an essentially unique factor, with a low (0.34) positive loading for Br; bearing in mind the work of Filby (1975), there is no obvious explanation for the weak association of As and Br in the Alberta crude oils, other than the speculation that Br also occurs in small organic modecules.

Evans et al. (1971) and Milner et al. (1977) have shown that the undersaturated oil systems of the Upper Devonian Beaverhill Lake Formation of west-central Alberta and the Middle Devonian Keg River Formation of north-western Alberta have both been subjected to gas deasphalting. The former comprise the Group 3b crude oils of Deroo et al. (1977). Samples from this study which have been subjected to gas deasphalting include Nos. 84, 85, 86 and 87. Although some of these have lower contents of asphaltenes and As (Sb was not detected) than both the average and median for all Group 3 crude oils, the small population precludes drawing a statistically valid conclusion respecting the effect of gas deasphalting on As and Sb in crude oils.

VANADIUM

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
V (ppm)	20	0.003	0.21	0.834	6.553
Group 2 crude oils (n=25)					
V (ppm)	23	0.068	20.88	28.1	138.8
Group 3 crude oils (n=33)					
V (ppm)	32	0.014	1.0	6.28	52.67

Geochemistry

Effectively all conventional crude oils contained detectable amounts of V, with a total range in content from 0.003 to 138.8 ppm. All groups of crude oils contained some samples with very low contents of V; the percentage of crude oils within each group with less than 1 ppm V were 85, 9 and 50, for Groups 1, 2 and 3, respectively. Among the three groups, the smallest median, average and maximum contents of V are found in Group 1, and the largest median, average and maximum values in Group 2; as a consequence, Group 3 exhibits statistical trends intermediate between Groups 1 and 2. The high value (13.71) of the F statistic in the ANOVA strongly suggests that V is of value in distinguishing crude

oil families. There is a high correlation between the contents of V and asphaltenes in Groups 1 and 2, with lower correlation for Group 3 (Table 3); lower correlation coefficients but similar relations exist between V and API gravity (Table 3).

Table 3. Correlation among V, asphaltenes, API gravity and V^{4+} for Alberta crude oils

Crude oil group	Correlation coefficient (r^2) between		
	V and % asphaltenes	V and API gravity	V and V^{4+}
1	0.87	0.59	0.60
2	0.86	0.68	0.98
3	0.56	0.16	0.75
All crude oils	0.66	0.52	0.96

The relation between V and asphaltenes is shown in Figure 3; both the general distribution charts and histograms are based on numerical data presented elsewhere in this report. Group 1 crude oils are characterized by low contents of both asphaltenes and V. As noted in the introduction to this section of the report, the frequency distribution of asphaltene content between Groups 2 and 3 is similar (as can be seen in figure 3), but their frequency distribution in V content is different. As suggested earlier, this would imply either different processes (other than maturation) operating after accumulation of the crude oils, or source differences. Bearing in mind the small number of samples involved, the removal of crude oils from reservoirs subjected to gas deasphalting does little to alter the profile of the frequency distribution diagram for V in the Group 3 crude oils (figure 3). The crude oils in Group 2 are known to be extensively biodegraded and this might account for the generally higher contents of V in these crude oils. Vanadium was determined in the Alberta shales studied by Hitchon (in press), and if the contents of V are grouped according to the crude oil families distinguished by Deroo et al. (1977) the statistics may be summarized as follows:

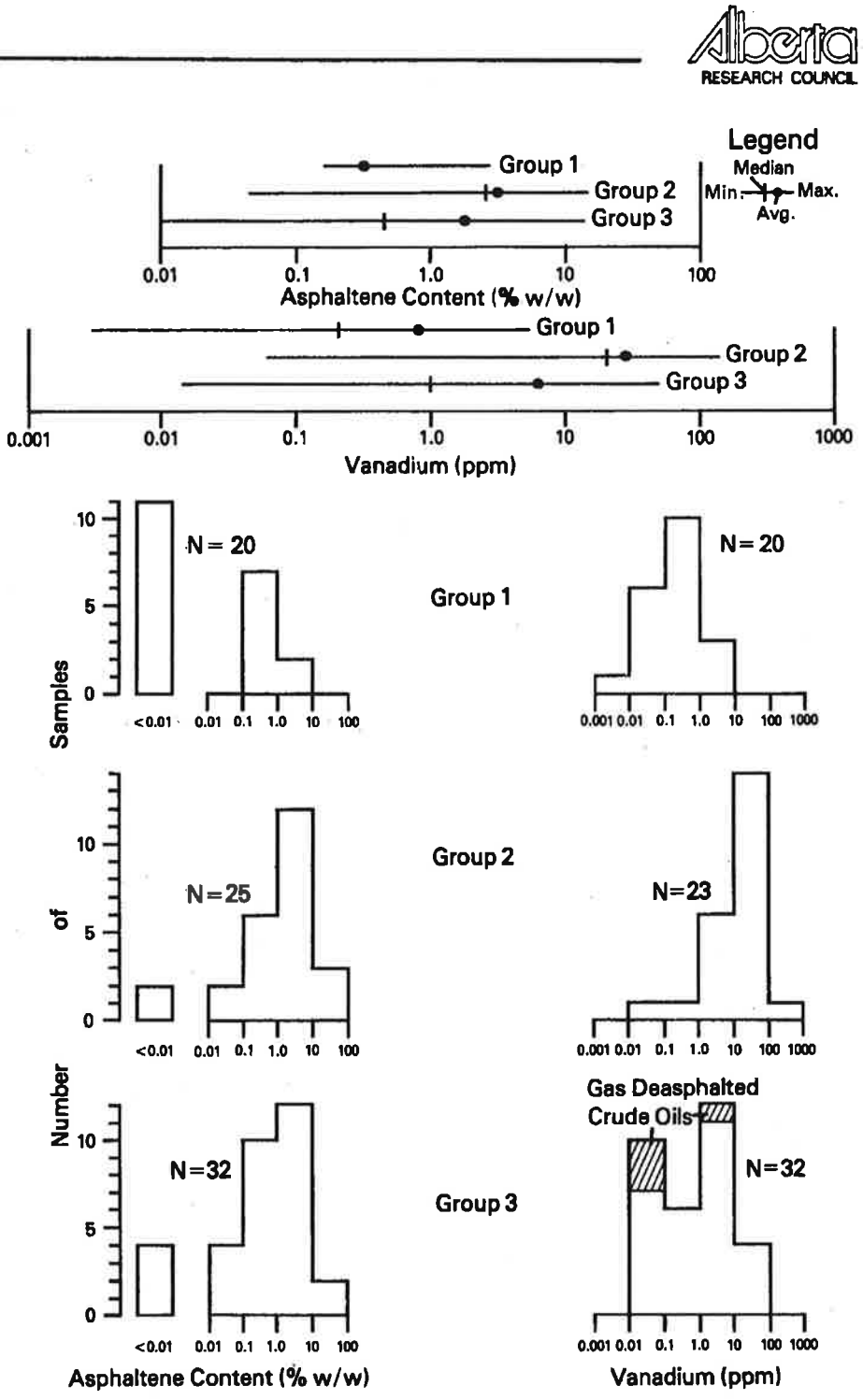

Figure 3. Statistical distribution and frequency histograms for asphaltenes and V in Alberta crude oils grouped according to the crude oil families distinguished by Deroo et al. (1977).

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	Group 1 (U. Cretaceous)	Group 2 (L. Cretaceous, Jurassic and Carboniferous)	Group 3 (U. and M. Devonian)
No. of samples	7	12	7
V (ppm) min.	46	55	35
median	87	75	71
avg.	86	116	68
max.	127	421	101

This information does not preclude a source difference but extreme caution must be used in any extrapolation from such a small and varied population. If the collection of further data supports the suggestion that the higher content of V in Group 2 crude oils is a result of higher V contents of the source sediments, then the differences in V content of the Group 1 and 3 crude oils may be similarly related to source differences, probably coupled with different degrees of maturation of the source sediments. In all cases it will be necessary to determine the form (inorganic and organic) of V in the shales and to obtain a high correlation between V in the crude oils and V in extracted organic matter from the presumed source sediments. Such a study would be of considerable significance for the explorationist.

On the basis of its content of As and Sb, crude oil No. 24 from the Basal Colorado sandstone at Cessford was classified with Group 2; a V content of 59.82 ppm adds support to this classification. Two crude oils were sampled from the Turner Valley field, No. 42 from the Lower Cretaceous Blairmore Group (0.017 ppm V) and No. 55 from the Carboniferous Turner Valley Formation (0.028 ppm V). Both values fall outside the range for other Group 2 crude oils and this fact, plus the structural separation of the Turner Valley field from other Group 2 reservoirs in the Alberta plains, suggests that these two crude oils may have a common origin that is different from other Group 2 crude oils. The two crude oils from the Carboniferous reservoir at Eaglesham (samples 52 and 53) have V contents more closely related to those from Turner Valley than the rest of the Group 2 crude oils. It seems likely that a detailed study of Carboniferous crude oils might show two distinct groups, those immediately adjacent to the pre-Cretaceous unconformity and having affinities with Group 2 crude oils, and a second group with a source within Carboniferous rocks, stratigraphically, and sometimes structurally, separate and unrelated to the pre-Cretaceous unconformity. Deroo et al. (1977) did not analyze crude oils from Triassic reservoirs, but the stratigraphic position of samples 47, 48 and 49 is close to the pre-Cretaceous unconformity with only a thin intervening cover of Jurassic strata; all have V contents consistant with Group 2 crude oils. Crude

oil No. 50, with a V content of 0.185 ppm, also lies at the Triassic-Jurassic boundary but is overlain by more than 75 m of dark L. Jurassic Fernie shales and a local source cannot be ruled out.

Because essentially all V in crude oils occurs in the tetravalent form (V^{4+}) it is not surprising that there is a generally high correlation between the total V content as determined by neutron activation and the content of tetravalent V as determined by ESR techniques; such a correlation is without geochemical significance, however. The data in Table 3 show that this is true whether the crude oils are all considered together, or are divided into the three groups distinguished by Deroo et al. (1977).

SULPHUR AND SELENIUM

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
S (%)	20	0.040	0.135	0.214	1.244
Se (ppb)	18	3.74	8.96	12.9	48.1
Group 2 crude oils (n=25)					
S (%)	25	0.04	1.391	1.31	3.355
Se (ppb)	19	5.26	55.22	78.7	252.3
Group 3 crude oils (n=33)					
S (%)	33	0.101	0.443	0.717	2.991
Se (ppb)	16	4.0	14.4	22.3	73.6

Geochemistry

All crude oils analyzed contained S, and Se was detected in 20 percent of the samples. As noted by Deroo et al. (1977) the biodegraded crude oils of Group 2 are characteristically rich in S, although all groups contain some crude oils with both high and low contents of S. The detrimental effect of biodegradation is well illustrated when Groups 1 and 2 are compared; both are from clastic source facies. The higher median and average values for S in Group 3, compared to Group 1, are most likely a reflection of the higher S in crude oils from carbonate-evaporite source facies (Evans et al., 1971). Hitchon et al. (1975) showed a high correlation between S and Se and therefore, as might be expected, all the above features can be demonstrated also for Se. The high inter-correlation among S, Se and V (Hitchon et al., 1975) and the fact that V has been shown to be a useful tool in crude oil correlation, means that S and Se should also be of value for correlating crude oils. For S, a complete set of data is available and the importance of this

element for classification of crude oils into families can be demonstrated quite clearly by the high value of the F statistic in the ANOVA. Thus even on the basis of the very simple statistical approach used in this report its importance is apparent. Selenium also has a high value of the F statistic in the ANOVA, indicating it also is of value in crude oil correlation.

Figure 4 shows the range, median and average values for S in each of the three crude oil groups, together with the concentration range (thick bar) for 80 percent of the samples distributed about the mean values. For Groups 1 and 2 there is no overlap of S contents for 80 percent of the samples, and Groups 2 and 3 are reasonably distinct, with some overlap for Group 2 crude oils which have below average contents of S and Group 3 crude oils which have above average contents of S. In most cases consideration of the stratigraphic situation will allow correct classification. For example, with one exception (sample No. 67 from the Arcs Member, Winterburn Group at Youngstown), all Group 3 crude oils with above average contents of S are from carbonate reefs or carbonate structural-stratigraphic traps far below the pre-Cretaceous unconformity in positions which make it most unlikely that the crude oils came from the Mannville Group. At Youngstown the stratigraphic position (95 m below the pre-Cretaceous unconformity) and hydraulic regime might indicate a Mannville Group source for the crude oil, but evidence based on only one trace element is not conclusive. Clearly, a multivariate trace element approach is needed for this case. Another example is sample No. 24 which is again definitely classified with the Group 2, as are the three Triassic crude oils, Nos. 47, 48 and 49. Triassic sample No. 50 is indeterminate and, as for V, a local source cannot be ruled out.

CHROMIUM

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Cr (ppb)	7	3.71	7.80	8.39	15.51
Group 2 crude oils (n=25)					
Cr (ppb)	14	4.66	24.5	41.6	105.8
Group 3 crude oils (n=33)					
Cr (ppb)	16	3.63	14.8	32.6	205.8

Geochemistry

Just over 45 percent of the crude oils contained detectable amounts of Cr. Group 1 crude oils exhibited the lowest median and average values as well as the narrowest range in Cr content. Crude oils from Groups 2 and 3 have effectively similar median and average contents of Cr, and

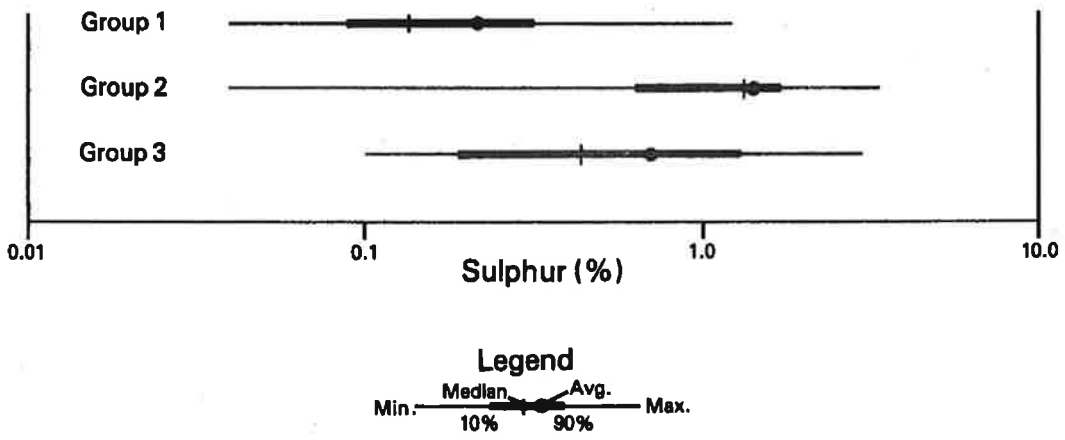

Figure 4. Statistical distribution of S in Alberta crude oils grouped according to the crude oil families distinguished by Deroo et al. (1977). The thick bar represents the range of 80 percent of the samples distributed about the mean.

maximum amounts an order of magnitude greater than those from Group 1. The fact that more than half the crude oils analyzed showed no detectable Cr means that this element is of minimal value in classifying crude oils. Hitchon et al. (1975) found a high correlation among Cr, V and Ni in their study of 20 selected crude oils and this suggests that if the detection limit can be lowered that Cr could be of use in classifying crude oils. This contention is supported by the fact that sample No. 24, with 95.2 ppb Cr has clear affinities with the Group 2 crude oils, as probably do samples 47, 48 and 49 from Triassic rocks.

HALOGENS - CHLORINE, BROMINE AND IODINE

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Cl (ppm)	20	2.16	6.91	9.48	38.74
Br (ppb)	20	5.641	19.2	32.3	127.4
I (ppb)	11	7.771	293.5	410	1364.0
Group 2 crude oils (n=25)					
Cl (ppm)	25	0.634	8.992	24.1	230.3
Br (ppb)	20	1.75	37.3	477	7529.0
I (ppb)	14	8.735	72.6	720	9005.0
Group 3 crude oils (n=33)					
Cl (ppm)	33	0.50	13.35	77.5	1014.0
Br (ppb)	32	4.27	87.5	923	12 470.0
I (ppb)	20	10.46	316	1077	7 396.0

Geochemistry

All crude oils contained detectable amounts of Cl, nearly 95 percent reported Br, and 60 percent showed I above detection limits. The values for the F statistic in the ANOVA indicate that Br, but not Cl, is of value in crude oil correlation. On initial appraisal the trends for Cl between the groups appear to be directly related to the trends for Cl in the co-produced formation waters, as shown in Table 2. This is fortuitous, however, because not only have Hitchon et al. (1975) shown that Na and Cl in crude oils are statistically unrelated, but plots of the ratios Na/Cl and Br/I between crude oils and their associated formation waters are effectively random, thus confirming that none of these elements (Na, Cl, Br or I) originate from entrained formation water. For 46 of the fields and pools represented by the 86 conventional crude oils analyzed, the contents of Cl and Br are available for the co-produced formation water, and calculations show that there is insufficient Cl reported in the crude oil to account for the Br content in more than half the samples, assuming all the Br originated from entrained formation water. Similar calculations for 32 pairs of crude oils and co-produced formations waters show enrichments of I in the crude oils of up to nearly 860 times that to be expected if all the I originated from

entrained formation water. This latter observation is particularly pertinent in view of the work of Hitchon and Horn (1974) on the use of I as a petroleum indicator in Paleozoic formation waters from Alberta. If the I-enrichment factors are classified into Mesozoic and Paleozoic groups, as were the formation waters studied by Hitchon and Horn (1974), the following statistics can be calculated:

Iodine-enrichment factor

	Minimum	Median	Average	Maximum
Mesozoic crude oils (n=16)	1.24	16.7	74.4	858
Paleozoic crude oils (n=16)	15.2	84.3	167	746

Although both groups of crude oils are enriched in I, those from Paleozoic rocks are particularly enriched in this element. A geochemical model for the occurrence of Br and I in formation waters and I in crude oils is shown in Figure 5. In non-producing situations the Br and I in formation waters essentially represents background Br and I from clays and I from organic matter in the adjacent (upflow) shales. In producing situations the background values of Br and I are still present but the higher content of organic matter in the source rock results in enhanced contents of I in the formation water, which is reflected in the I content of the crude oil. Thus although I was not a discriminator for the Mesozoic formation waters studied by Hitchon and Horn (1974) it nevertheless is relatively enriched in Mesozoic crude oils. Clearly, for certain biophile elements like I their distribution and geochemical occurrence in the fluids and rocks of sedimentary basins justifies more study and attention than they have received heretofor.

MANGANESE

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Mn (ppb)	20	1.0	50.6	54.9	119.2
Group 2 crude oils (n=25)					
Mn (ppb)	19	1.70	49.8	47.1	79.28
Group 3 crude oils (n=33)					
Mn (ppb)	31	17.93	49.2	52.6	176.6

Geochemistry

Manganese was detected in nearly 90 percent of the conventional crude oils analyzed. The grouped general statistics indicate that of all the

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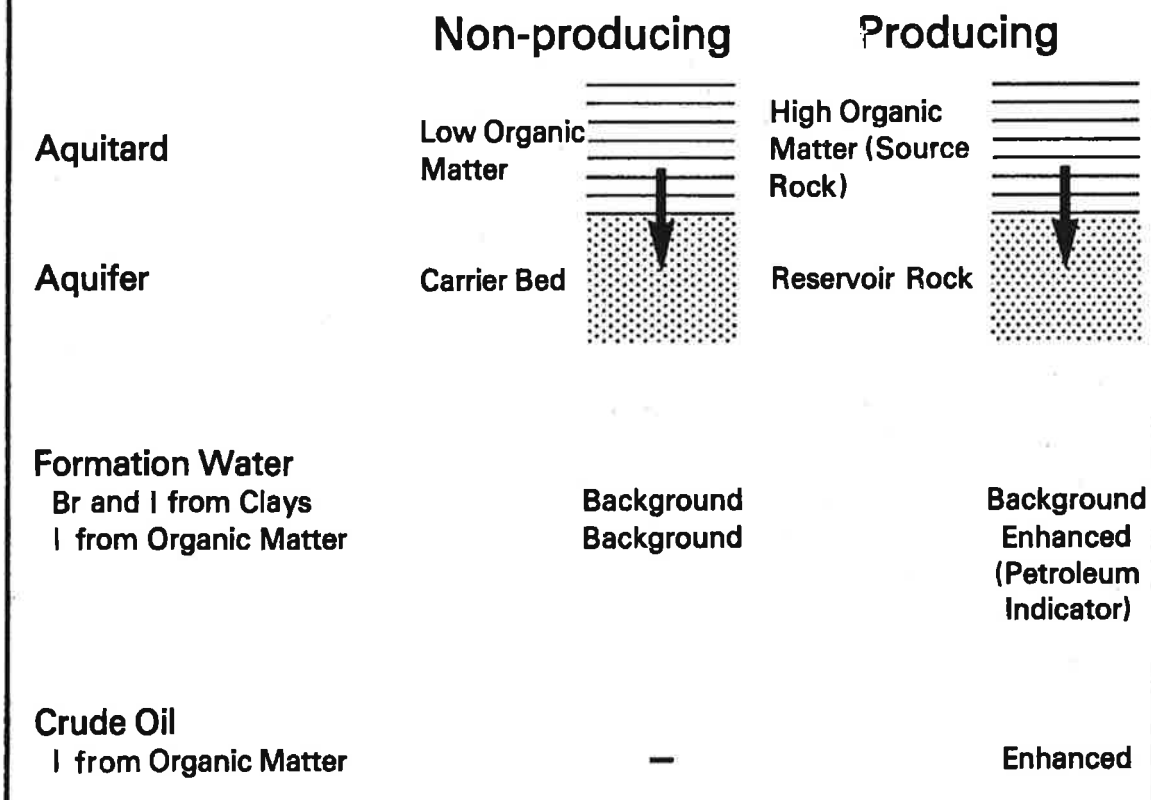

Figure 5. Geochemical model for the occurrence of Br and I in formation waters and I in crude oils, for non-producing and producing situations.

trace elements determined it is probably the most evenly distributed, not only among the three crude oil groups distinguished by Deroo et al. (1977) but also in those conventional crude oils not considered in the general statistics. Clearly, Mn cannot be used in the classification of crude oils, a fact confirmed by the ANOVA results. Hitchon et al. (1975) found that Mn was the only element forming a unique factor, without significant loadings of any other element. Even in their study of 20 selected crude oils Mn was effectively a unique factor, with only a minor loading for Cr. The factor study, together with the general statistics reported here, make it difficult to determine the manner of occurrence of Mn in crude oils, bearing in mind its factorially unique nature and its very even distribution across a suite of crude oils with widely differing properties from at least three distinct sources and in crude oils that have been subjected to maturation, biodegradation and gas deasphalting. The only distinct difference between the 86 conventional crude oils with their very uniform contents of Mn, and the sample from the Athabasca oil sand (No. 94) with an extremely high content of Mn (3853 ppb) is in sample preparation. This may imply some form of general contamination of the conventional crude oils by Mn during sample preparation, as yet unidentified.

IRON, COBALT AND NICKEL

General Statistics

	Number of samples reporting element	Minimum	Median	Average	Maximum
Group 1 crude oils (n=20)					
Fe (ppm)	10	0.071	0.466	0.545	1.671
Co (ppb)	18	0.261	2.71	8.05	29.07
Ni (ppm)	15	0.022	0.520	0.939	4.03
Group 2 crude oils (n=25)					
Fe (ppm)	12	0.286	0.587	0.94	2.19
Co (ppb)	25	0.397	4.484	10.2	60.22
Ni (ppm)	22	0.136	8.53	11.5	57.48
Group 3 crude oils (n=33)					
Fe (ppm)	16	0.216	0.566	2.04	19.77
Co (ppb)	32	0.516	8.66	22.7	202.6
Ni (ppm)	24	0.085	3.51	7.96	70.1

Geochemistry

For the 86 conventional crude oils studied, Fe was detected in 45 percent, Co in 95 percent and Ni in nearly 80 percent of the samples. With respect to Fe and Co, there is a gradual increase in the range and both the median and average values from Group 1 to Group 3 crude oils. Nickel shows a similar trend in the range of values from Group 1 to Group 3 but the median and average values for Group 2 crude oils are the

highest, with Group 1 crude oils having the lowest median and average contents of this element. With respect to Ni, therefore, the trends are very similar to those for V. Both Ni and V occur in crude oils as porphyrin compounds as well as in a non-porphyrin form, whereas Fe and Co appear to occur only as non-porphyrin material (Filby, 1975), and this may, in part, explain the differences in trends between Ni and V, and Fe and Co. Despite these apparent differences, both Ni and Co have values of the F statistic which suggest their use as crude oil correlation discriminators. The Ni content of sample No. 24 clearly places it in the Group 2 crude oils, as also do the Ni contents of samples 47, 48 and 49 from Triassic reservoirs. The data from the Carboniferous reservoir at the Eaglesham field is equivocal.

TRACE ELEMENTS IN CRUDE OILS FROM THE ATHABASCA OIL SAND DEPOSIT

The two crude oils from the Athabasca oil sand deposit (Nos. 43 and 94) are here treated separately from the 86 conventional crude oils because the nature of the material necessitated different sample preparation techniques and the possibility of contamination from entrained fine-grained mineral matter cannot be ruled out. They are, nevertheless, unequivocally classified as Group 2 crude oils. Table 4 lists the maximum value reported for all trace elements analyzed in Group 2 conventional crude oils and the values reported for samples 43 and 94 (in increasing order of magnitude). A subjective estimate of the probability of the values for the Athabasca oil sand deposit samples being due to contamination is shown in the last column. The dilute formation waters associated with the Athabasca oil sand deposit are essentially of the sodium bicarbonate type and this fact is the reason for the suggested very high and high probability of contamination for the alkali metals (Na, Rb and Cs) compared to the halogens (Cl and Br). In shales, the proportions of Ga:Sc:Eu are of the order 20:10:1; for samples 43 and 94 the proportions of these trace elements are 35:22:1 and 11.5:8:1, respectively. These proportions are strongly suggestive of contamination by entrained fine-grained clay minerals. The possibility of contamination by fine-grained entrained pyrite cannot be ruled out (although its presence was not sought in this study) and this may be the explanation for the very high contents of Fe, Co, Mn, and possibly Cr. Values for the other elements (S, V, Ni, As, Hg, Sb, Se, Cl and Br) probably reflect true amounts associated with the crude oil.

Table 4. Comparison of maximum content of trace elements in Group 2 conventional crude oils with trace elements of two crude oils from Athabasca oil sand deposit.

Element	Maximum in Group 2 conventional crude oils	Athabasca oil sand deposit	
		Reported contents	Probability of contamination
Na (ppm)	19.3	20.78 ; 40.33	High
Rb (ppb)	57.8	377.5 ; 720.2	Very high
Cs (ppb)	9.03	25.9 ; 68.53	Very high
Zn (ppb)	1277.0	-	
Hg (ppb)	138.3	81.7 ;	Low
Sc (ppb)	0.764	190.7 ; 199.0	Very high
Eu (ppb)	0.661	9.0 ; 23.2	Very high
Ga (ppb)	11.0	267.0 ; 315.0	Very high
As (ppb)	1988.0	320.9 ; 400.3	Low
Sb (ppb)	22.8	27.7 ; 30.61	Low
V (ppm)	138.8	176.5 ;	Low
S (%)	3.355	3.881;	Low
Se (ppb)	252.3	286.4 ; 517.1	Low
Cr (ppb)	105.8	1014.0 ; 1682.0	Intermediate/high
Cl (ppm)	230.3	7.961;	Low
Br (ppb)	7529.0	103.6 ; 155.3	Low
I (ppb)	9005.0	-	
Mn (ppb)	79.28	3853.0 ;	Very high
Fe (ppm)	2.19	141.7 ; 254.2	Very high
Co (ppb)	60.22	1349.0 ; 1998.0	Very high
Ni (ppm)	57.48	71.88 ; 74.11	Low
Asphaltenes (%w/w)	15.5	17.1 ;	

- = Not reported

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SUMMARY

This preliminary study shows that when Alberta crude oils are grouped into families, based on their hydrocarbon characteristics, some trace elements may also be a basis for distinguishing the groups. Initial indications are that S, Se, V, Ni, Br, Co and Zn are likely to be the most important discriminators. The data have been treated in a univariate manner using only the most simplistic statistical approach and a more thorough, multivariate statistical analysis is clearly justified. In addition to the use of trace elements to classify crude oils into families the following geochemical observations were made:

1. It is suggested, tentatively, that crude oils with alkaline associated formation waters may be relatively depleted in Na, when compared to those with acidic associated formation waters. The genetic relations among surface tension, pH and surfactants (such as Na salts of petroleum acids) in formation waters, and the content and form of bonding of Na in their associated crude oils justifies considerably more study, and may be of value to explorationists through the use of salts of petroleum acids in formation waters as indicators for crude oil occurrence.

2. Comparison of Zn in shales, Cl and Zn in formation waters, and Zn and Hg in crude oils, all grouped on the basis of the work of Deroo et al. (1977), supports the thesis that the Zn in crude oils, and probably also Hg, as well as Zn in formation waters, have originated from shales which yielded the crude oil hydrocarbons through the specific action of hot, chloride-rich formation waters, with the consequent depletion of those shales in Zn (and probably also Hg).

3. Preliminary evidence is presented to suggest that the V content of crude oils may be related to the V content of the source rocks. A study to determine the form (inorganic and organic) of V in shales, and to seek the relationship between V in crude oils and V in extracted organic matter from the presumed source sediments, could be of considerable significance to the explorationist.

4. Some crude oils show enrichment of I of up to nearly 860 times that to be expected if all the I originated from entrained formation waters. Although almost all crude oils are enriched in I, those from Paleozoic rocks are particularly enriched, compared to those from Mesozoic rocks. A geochemical model is presented for the occurrence of Br and I in formation waters and I in crude oils; in non-producing situations the Br and I in formation waters essentially represents Br and I from clays and I from organic matter in the adjacent (upflow) shales, whereas in producing situations the background values of Br and I are still present but the higher content of organic matter in the source rock results in enhanced

contents of I in the formation water, which is reflected in the I content of the crude oil. Thus although I was not a discriminator for the Mesozoic formation waters studied by Hitchon and Horn (1974) it nevertheless is relatively enriched in Mesozoic crude oils. Clearly, for certain biophile elements like I their distribution and geochemical occurrence in the fluids and rocks of sedimentary basins justifies more study and attention than they have received heretofor.

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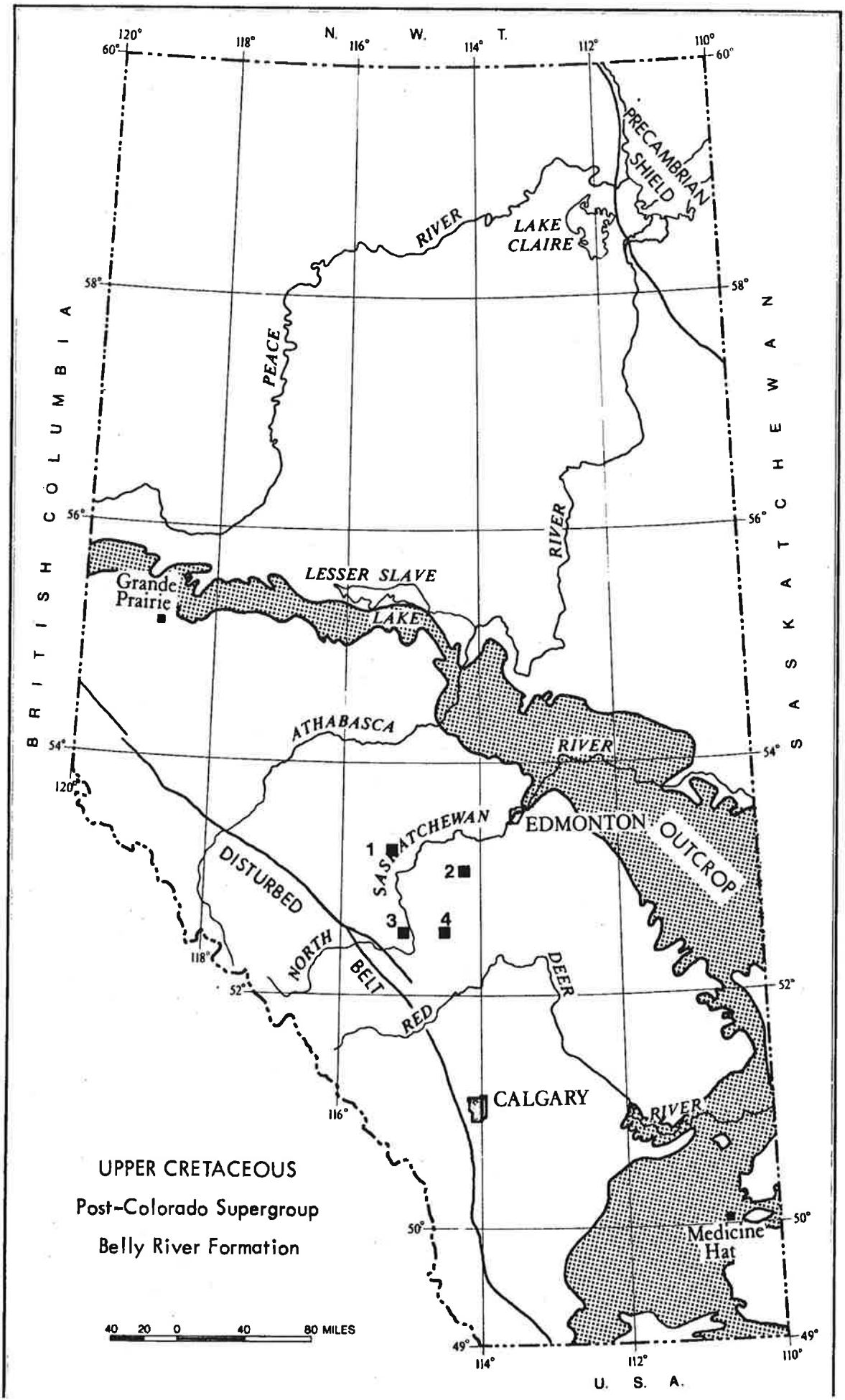
APPENDIX

Trace Elements in Alberta Crude Oils - Data Sheets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RIVER
PEACE
BRITISH COLUMBIA
SASKATCHEWAN
Grande Prairie
LESSER SLAVE LAKE
ATHABASCA RIVER
SASKATCHEWAN
EDMONTON
OUTCROP
1
2
3
4
DISTURBED
NORTH
BELT
DEER
RED
RIVER
CALGARY
Medicine Hat
UPPER CRETACEOUS
Post-Colorado Supergroup
Belly River Formation
40 20 0 40 80 MILES
U. S. A.

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TRACE ELEMENTS IN ALBEPTA CRUDE OILS -- DATA SHEET

Sample number: 1

Field (pool): Pembina (Belly River J)

Stratigraphic unit: U. Cretaceous, Belly River Fm.

Well name: Pan Am HB J-35 Pem 10-21-49-9

Location: Lsd 10 Sec 21 Twp 49 Rge 9 W 5 Mer

Source of sample: Wellhead - tubing Pressure: 150 psig Temperature: cold °F

Method of production: Pump Depth: 4050-4064 ft. (perforations)

Date sampled: 4th Feb. 1970

Remarks:

S	(%)						
V	(ppm)	0.461	(0.0143)				
Cl	(ppm)	8.860	(0.4840)				
Na	(ppm)	0.810	(0.0140)				
Fe	(ppm)						
Ni	(ppm)	0.574	(0.0356)				
Zn	(ppb)	172.0	(10.2)	Au	(ppb)		
Co	(ppb)	7.564	(0.43)	Sb	(ppb)	0.129	(0.079)
I	(ppb)	116.20	(18.19)	Cr	(ppb)		
Mn	(ppb)	73.40	(1.375)	Rb	(ppb)		
Se	(ppb)	12.4	(1.90)	Sc	(ppb)	0.19	(0.0211)
Hg	(ppb)	8.05	(0.813)	Eu	(ppb)		
Cs	(ppb)	1.3	(0.472)	Ga	(ppb)		
Br	(ppb)	58.6	(1.12)				
As	(ppb)	34.0	(0.531)				

* Relative standard deviation (counting statistics) in parenthesis

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TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 2

Field (pool): Pembina (Keystone Belly River M)

Stratigraphic unit: U. Cretaceous, Belly River Fm.

Well name: Calstan Pembina 14-34BR-47-2

Location: Lsd 14 Sec 34 Twp 47 Rge 2 W 5 Mer

Source of sample: Wellhead-tubing Pressure: nil psig Temperature: cold°F

Method of production: Pump Depth: 3154 ft. (perforations)

Date sampled: 2nd Feb. 1970

Remarks: Prior to sample preparation contained some silty material.

S	(%)	0.193	(0.0196)				
V	(ppm)	0.636	(0.0091)				
Cl	(ppm)	8.773	(0.2838)				
Na	(ppm)	0.307	(0.0050)				
Fe	(ppm)						
Ni	(ppm)	1.26	(0.245)				
Zn	(ppb)	130.0	(9.31)	Au	(ppb)		
Co	(ppb)	12.43	(0.90)	Sb	(ppb)		
I	(ppb)	1101.0	(16.08)	Cr	(ppb)	6.23	(1.80)
Mn	(ppb)	67.47	(1.640)	Rb	(ppb)		
Se	(ppb)	12.0	(1.97)	Sc	(ppb)		
Hg	(ppb)	13.45	(1.17)	Eu	(ppb)	0.212	(0.063)
Cs	(ppb)	2.15	(0.490)	Ga	(ppb)		
Br	(ppb)	12.62	(0.50)				
As	(ppb)	4.35	(0.194)				

* Relative standard deviation (counting statistics) in parenthesis

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TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 3

Field (pool): Ferrier (Belly River A)

Stratigraphic unit: U. Cretaceous, Belly River Fm.

Well name: Amerada Cdn-Sup Ferrier 4-7-41-8

Location: Lsd 4 Sec 7 Twp 41 Rge 8 W 5 Mer

Source of sample: Test separator-sight glass Pressure: 120 psig Temperature: 62 °F

Method of production: Pump Depth: 5545-5571 ft. (perforations)

Date sampled: 17th Sept. 1970

Remarks:

S	(%)	0.141	(0.0169)				
V	(ppm)	0.554	(0.0100)				
Cl	(ppm)	7.470	(0.3121)				
Na	(ppm)	0.561	(0.0098)				
Fe	(ppm)	0.357	(0.092)				
Ni	(ppm)	0.718	(0.0281)				
Zn	(ppb)	286.9	(10.2)	Au	(ppb)		
Co	(ppb)	29.07	(0.548)	Sb	(ppb)	0.810	(0.312)
I	(ppb)	43.23	(6.147)	Cr	(ppb)	7.80	(1.18)
Mn	(ppb)	77.87	(2.088)	Rb	(ppb)		
Se	(ppb)	14.23	(1.20)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.169	(0.0848)
Cs	(ppb)	1.0	(0.26)	Ga	(ppb)		
Br	(ppb)	39.5	(1.79)				
As	(ppb)	3.694	(0.326)				

* - Relative standard deviation (counting statistics) in parenthesis

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TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 4

Field (pool): Gilby (Belly River A)

Stratigraphic unit: U. Cretaceous, Belly River Fm.

Well name: Hamilton Gilby 12-33-41-4

Location: Lsd 12 Sec 33 Twp 41 Rge 4 W 5 Mer

Source of sample: Wellhead-tubing Pressure: 38 psig Temperature: 37 °F

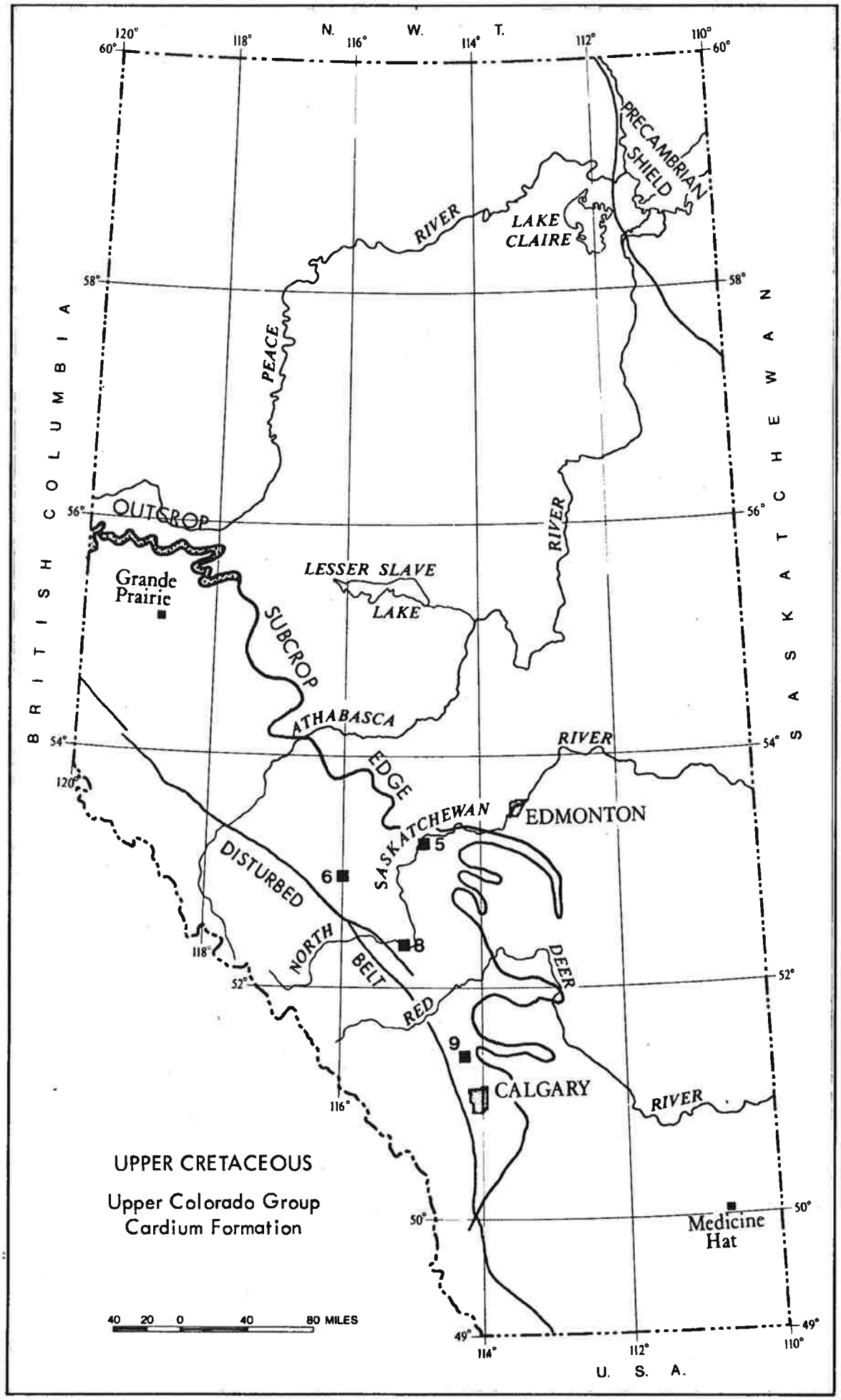
Method of production: Flowing Depth: 4201-4205 ft. (perforations)
4209-4216

Date sampled: 12th Feb. 1971

Remarks:

S	(%)	0.109	(0.0167)				
V	(ppm)	0.176	(0.0060)				
Cl	(ppm)	8.645	(0.2988)				
Na	(ppm)	0.572	(0.043)				
Fe	(ppm)	0.107	(0.0293)				
Ni	(ppm)	0.277	(0.0093)				
Zn	(ppb)	186.6	(4.52)	Au	(ppb)		
Co	(ppb)	2.81	(0.487)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	3.71	(0.570)
Mn	(ppb)	73.06	(1.061)	Rb	(ppb)		
Se	(ppb)	10.72	(0.68)	Sc	(ppb)	0.016	(0.004)
Hg	(ppb)	6.54	(0.579)	Eu	(ppb)	0.073	(0.031)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	23.4	(0.776)				
As	(ppb)	24.42	(0.530)				

* Relative standard deviation (counting statistics) in parenthesis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PRECAMBRIAN SHIELD
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RIVER
PEACE
BRITISH COLUMBIA
SASKATCHEWAN
OUTCROP
Grande Prairie
LESSER SLAVE LAKE
SUBCROP
ATHABASCA
RIVER
EDGE
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH
BELT
DEER
RED
CALGARY
RIVER
Medicine Hat
5
6
8
9
UPPER CRETACEOUS
Upper Colorado Group
Cardium Formation
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 5

Field (pool): Pembina (Cardium)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Cardium Fm.

Well name: Imp. Cdn-Sup Pembina 14-22C-49-6

Location: Lsd 14 Sec 22 Twp 49 Rge 6 W 5 Mer

Source of sample: Separator Pressure: 225psig Temperature: 64 °F

Method of production: - Depth: 4345.5 to 4382.5 ft. (perforations)

Date sampled: 14th July 1969

Remarks:

S	(%)	0.233	(0.0595)		
V	(ppm)	1.107	(0.0188)		
Cl	(ppm)	17.44	(0.8181)		
Na	(ppm)	2.23	(0.014)		
Fe	(ppm)	0.457	(0.081)		
Ni	(ppm)	1.961	(0.044)		
Zn	(ppb)	177.7	(8.212)	Au	(ppb)
Co	(ppb)	25.0	(0.530)	Sb	(ppb)
I	(ppb)	367.4	(13.95)	Cr	(ppb)
Mn	(ppb)	62.36	(1.535)	Rb	(ppb)
Se	(ppb)	30.63	(1.97)	Sc	(ppb)
Hg	(ppb)			Eu	(ppb)
Cs	(ppb)	0.562	(0.2173)	Ga	(ppb)
Br	(ppb)	48.2	(1.63)		
As	(ppb)	8.24	(0.661)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 6

Field (pool): Brazeau River (Cardium A)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Cardium Fm.

Well name: HB Texaco BrazR 10-21-46-14

Location: Lsd 10 Sec 21 Twp 46 Rge 14 W 5 Mer

Source of sample: Storage tank Pressure: atmos. psig Temperature: 32°F

Method of production: Flowing Depth: 7772-7777 ft (perforations)

Date sampled: 19th Feb. 1970

Remarks:

S	(%)	0.061	(0.0146)				
V	(ppm)	0.016	(0.0013)				
Cl	(ppm)	4.789	(0.2039)				
Na	(ppm)	0.647	(0.0053)				
Fe	(ppm)						
Ni	(ppm)	0.022	(0.005)				
Zn	(ppb)	34.3	(2.03)	Au	(ppb)		
Co	(ppb)	0.442	(0.047)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	44.91	(0.9198)	Rb	(ppb)		
Se	(ppb)	4.0	(0.46)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.102	(0.027)
Cs	(ppb)	0.532	(0.0711)	Ga	(ppb)		
Br	(ppb)	13.70	(0.642)				
As	(ppb)	3.04	(0.276)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 8

Field (pool): Ferrier (Cardium B)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Cardium Fm.

Well name: Sinclair Pacific Ferrier 8-28MU-39-8

Location: Lsd 8 Sec 28 Twp 39 Rge 8 W 5 Mer

Source of sample: Separator Pressure: 255 psig Temperature: 97 °F

Method of production: Flowing Depth: 7382-7390 ft. (perforations)

Date sampled: 16th Jan. 1971

Remarks:

S	(%)						
V	(ppm)	0.013	(0.0015)				
Cl	(ppm)	3.702	(0.0292)				
Na	(ppm)	0.328	(0.0027)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	315.0	(5.40)	Au	(ppb)		
Co	(ppb)	0.264	(0.045)	Sb	(ppb)	0.290	(0.061)
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	32.0	(0.77)	Rb	(ppb)	1.870	(0.913)
Se	(ppb)	3.74	(0.418)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.10	(0.0261)
Cs	(ppb)	0.430	(0.081)	Ga	(ppb)		
Br	(ppb)	9.22	(0.367)				
As	(ppb)	0.755	(0.124)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 9

Field (pool): Crossfield (Cardium A)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Cardium Fm.

Well name: Baysel Shell Crossfield 8-21-28-2

Location: Lsd 8 Sec 21 Twp 28 Rge 2 W 5 Mer

Source of sample: Wellhead Pressure: 45 psig Temperature: 73°F

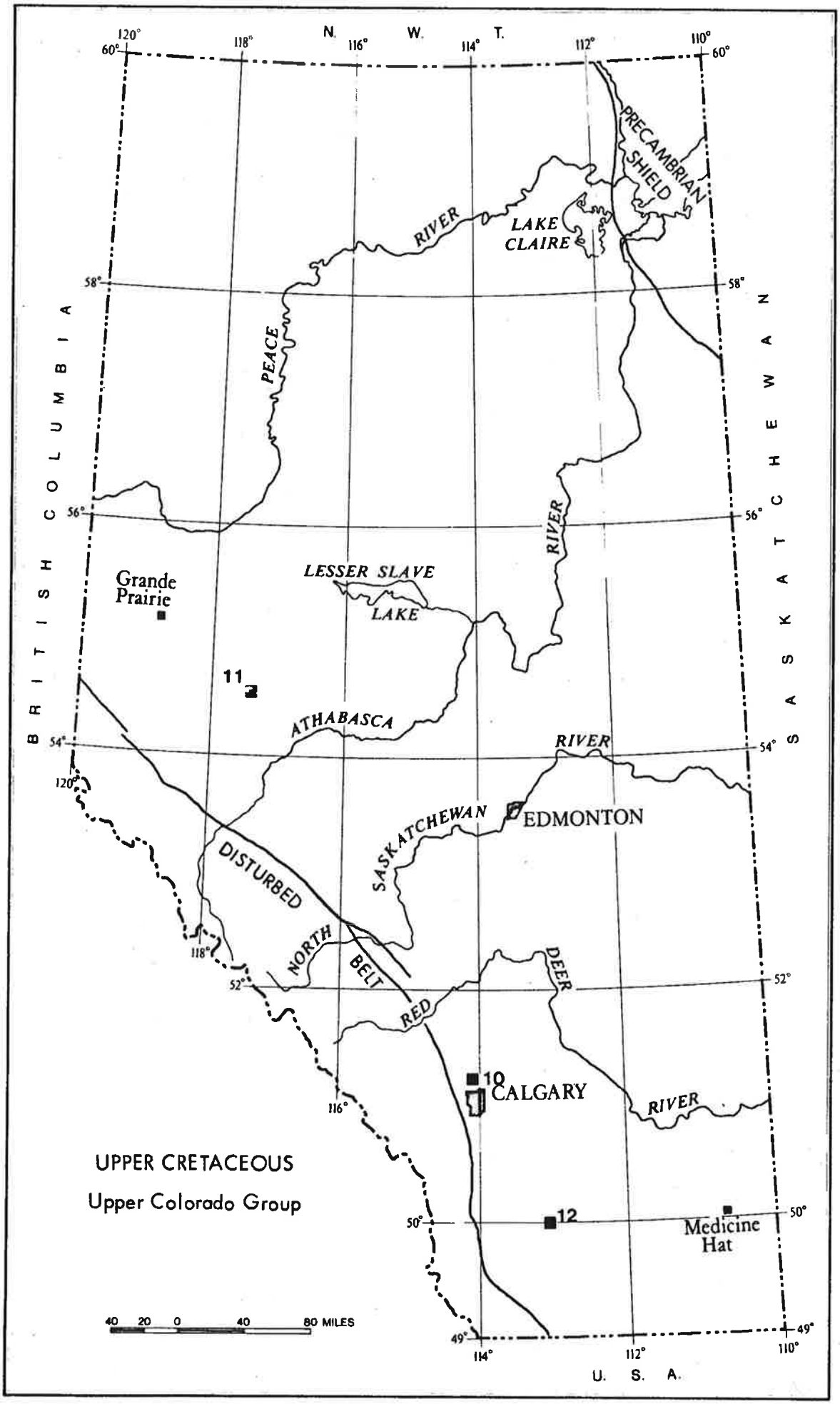
Method of production: Pump Depth: 6728.5-6739.5 ft. (perforations)

Date sampled: 23rd June 1970

Remarks:

S	(%)	0.198	(0.0258)				
V	(ppm)	0.381	(0.0079)				
Cl	(ppm)	8.860	(0.4840)				
Na	(ppm)	0.284	(0.0045)				
Fe	(ppm)	0.475	(0.150)				
Ni	(ppm)	0.520	(0.033)				
Zn	(ppb)	36.8	(3.79)	Au	(ppb)		
Co	(ppb)	17.12	(0.814)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	8.91	(1.50)
Mn	(ppb)	73.40	(1.375)	Rb	(ppb)		
Se	(ppb)	8.51	(1.53)	Sc	(ppb)		
Hg	(ppb)	17.7	(1.22)	Eu	(ppb)	0.641	(0.10)
Cs	(ppb)	0.75	(0.35)	Ga	(ppb)		
Br	(ppb)	65.83	(2.45)				
As	(ppb)	6.69	(0.557)				

* Relative standard deviation (counting statistics) in parenthesis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PRECAMBRIAN SHIELD
LAKE CLAIRE
RIVER
PEACE
BRITISH COLUMBIA
SASKATCHEWAN
Grande Prairie
LESSER SLAVE LAKE
11
ATHABASCA
RIVER
EDMONTON
SASKATCHEWAN
DISTURBED
NORTH
BELT
DEER
RED
10
CALGARY
RIVER
12
Medicine Hat
UPPER CRETACEOUS
Upper Colorado Group
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 10

Field (pool): Crossfield (Jumping Pound A)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Jumping Pound Ss.

Well name: Homestead et al Airdrie 10-31-26-1

Location: Lsd 10 Sec 31 Twp 26 Rge 1 W 5 Mer

Source of sample: Tank Pressure: nil psig Temperature: 80 °F

Method of production: Pump Depth: 6828-6844 ft. (perforations)

Date sampled: 23rd June 1970

Remarks: Well shut in 19th June 1970

S	(%)						
V	(ppm)	0.090	(0.0051)				
Cl	(ppm)	6.723	(0.2624)				
Na	(ppm)	0.646	(0.0082)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	303.2	(8.60)	Au	(ppb)		
Co	(ppb)	0.803	(0.210)	Sb	(ppb)		
I	(ppb)	31.06	(6.707)	Cr	(ppb)		
Mn	(ppb)	119.2	(1.879)	Rb	(ppb)		
Se	(ppb)	8.09	(1.95)	Sc	(ppb)	0.060	(0.013)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	0.692	(0.180)	Ga	(ppb)		
Br	(ppb)	24.4	(2.00)				
As	(ppb)	21.3	(0.99)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 11

Field (pool): Waskahigan (Dunvegan A)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Dunvegan Fm.

Well name: Mobil Amoco Waskahigan 6-26-64-23

Location: Lsd 6 Sec 26 Twp 64 Rge 23 W 5 Mer

Source of sample: Gas plant Pressure: 10 psig Temperature: 48 °F

Method of production: Flowing Depth: 5094-5113 ft. (perforations)

Date sampled: 24th July 1970

Remarks: Well fractured with Beaverhill Lake crude oil, therefore there may be traces of other oil in this condensate.

S	(%)						
V	(ppm)	0.003	(0.0007)				
Cl	(ppm)	2.16	(0.125)				
Na	(ppm)	0.051	(0.0008)				
Fe	(ppm)	0.071	(0.029)				
Ni	(ppm)						
Zn	(ppb)	11.51	(1.85)	Au	(ppb)		
Co	(ppb)	0.261	(0.059)	Sb	(ppb)	0.20	(0.077)
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	1.0	(0.20)	Rb	(ppb)	5.43	(T.101)
Se	(ppb)			Sc	(ppb)	0.013	(0.0034)
Hg	(ppb)	3.310	(0.325)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	7.002	(0.257)				
As	(ppb)	0.610	(0.0731)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 12

Field (pool): Barons (Colorado)

Stratigraphic unit: U. Cretaceous, Upper Colorado Gp., Fishscales Ss.

Well name: Barons 1

Location: Lsd 10 Sec 15 Twp 12 Rge 23 W 4 Mer

Source of sample: Tank Pressure: nil psig Temperature: 88°F

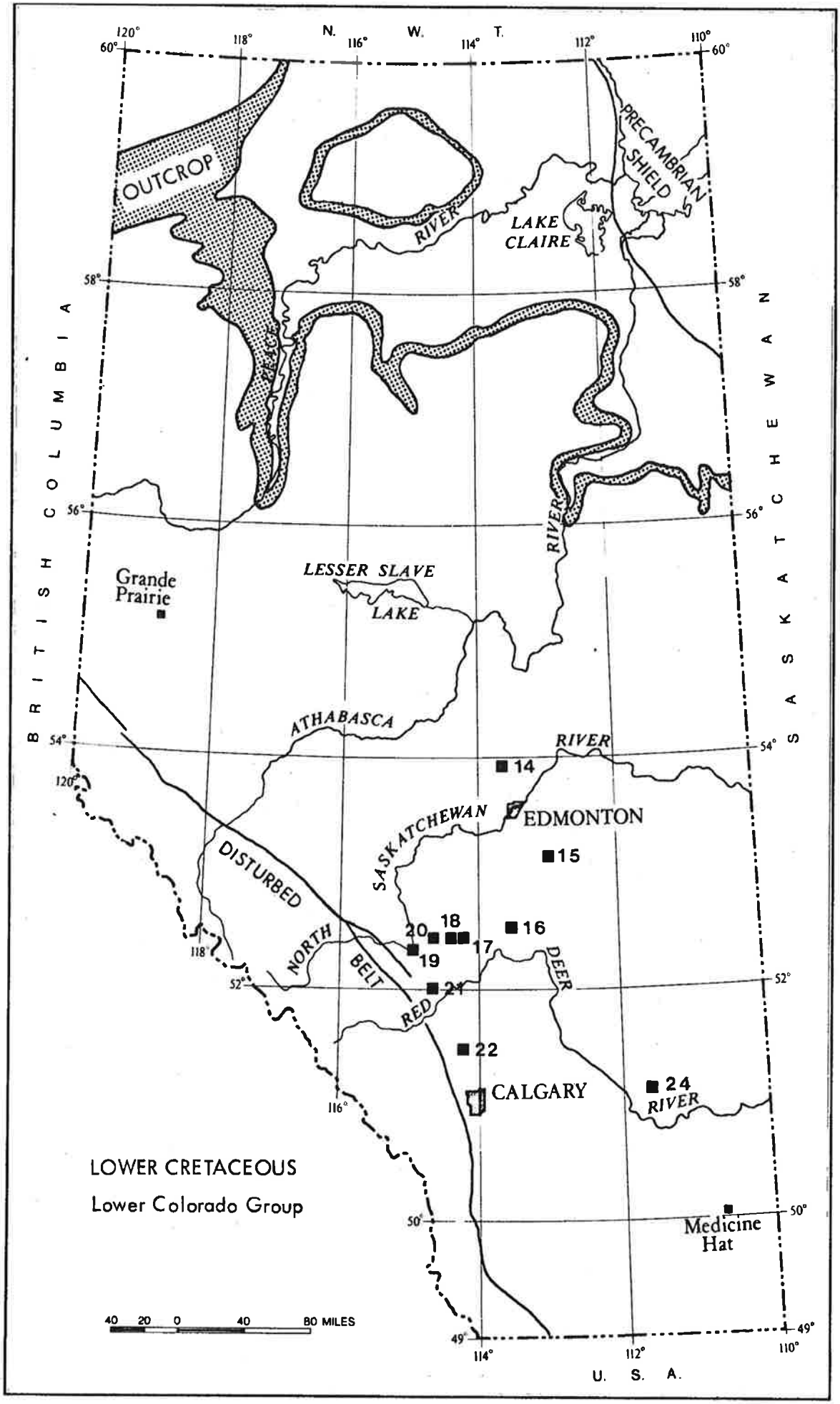
Method of production: Pump Depth: Open hole ft.
completion. Barons sand 4105 ft.
T. D. 4114 ft.

Date sampled: 25th June 1970

Remarks:

S	(%)	0.314	(0.0347)				
V	(ppm)	4.992	(0.0686)				
Cl	(ppm)	3.654	(0.2101)				
Na	(ppm)	0.046	(0.0011)				
Fe	(ppm)	0.768	(0.1002)				
Ni	(ppm)	3.20	(0.068)				
Zn	(ppb)	324.8	(11.1)	Au	(ppb)	0.078	(0.0077)
Co	(ppb)	20.20	(0.460)	Sb	(ppb)		
I	(ppb)	79.01	(7.273)	Cr	(ppb)		
Mn	(ppb)	48.90	(1.305)	Rb	(ppb)		
Se	(ppb)	27.7	(2.05)	Sc	(ppb)		
Hg	(ppb)	3.08	(1.20)	Eu	(ppb)	0.305	(0.0727)
Cs	(ppb)			Ga	(ppb)	1.13	0.070
Br	(ppb)	38.74	(1.48)				
As	(ppb)	1.860	(0.148)				

* Relative standard deviation (counting statistics) in parenthesis


N. W. T.
OUTCROP
PRECAMBRIAN SHIELD
RIVER
LAKE CLAIRE
BRITISH COLUMBIA
SASKATCHEWAN
Grande Prairie
LESSER SLAVE LAKE
ATHABASCA RIVER
SASKATCHEWAN RIVER
EDMONTON
CALGARY
Medicine Hat
DISTURBED BELT
NORTH RED DEER RIVER
14
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24
LOWER CRETACEOUS
Lower Colorado Group
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 14

Field (pool): Legal (Viking)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: LWS 3 Legal 3-21-57-25

Location: Lsd 3 Sec 21 Twp 57 Rge 25 W 4 Mer

Source of sample: Wellhead Pressure: 200 psig Temperature: - °F

Method of production: Pump Depth: 2790-2792 ft. (perforations)

Date sampled: September, 1971

Remarks:

S	(%)	1.244	(0.0631)				
V	(ppm)	6.553	(0.0579)				
Cl	(ppm)	38.74	(0.9333)				
Na	(ppm)	13.0	(0.217)				
Fe	(ppm)	1.671	(0.123)				
Ni	(ppm)	4.03	(0.085)				
Zn	(ppb)	266.8	(11.07)	Au	(ppb)		
Co	(ppb)	19.9	(0.513)	Sb	(ppb)		
I	(ppb)	488.6	(13.28)	Cr	(ppb)	6.20	(2.0)
Mn	(ppb)	52.23	(1.167)	Rb	(ppb)		
Se	(ppb)	48.1	(2.814)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	2.27	(0.30)	Ga	(ppb)		
Br	(ppb)	127.4	(8.85)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 15

Field (pool): Joarcam (Viking)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Imperial Gulf HB Armena 9-34-48-21

Location: Lsd 9 Sec 34 Twp 48 Rge 21 W 4 Mer

Source of sample: Satellite manifold Pressure: - psig Temperature: - °F

Method of production: Pump Depth: 3254-3259 ft. (perforations)

Date sampled: 14th July, 1969

Remarks: Sample bottle contained sediment on bottom

S	(%)	0.145	(0.018)				
V	(ppm)	0.682	(0.0032)				
Cl	(ppm)	25.5	(0.528)				
Na	(ppm)	2.921	(0.0247)				
Fe	(ppm)	0.696	(0.0976)				
Ni	(ppm)	0.609	(0.0236)				
Zn	(ppb)	669.7	(14.34)	Au	(ppb)		
Co	(ppb)	2.75	(0.087)	Sb	(ppb)		
I	(ppb)	1364.0	(24.0)	Cr	(ppb)		
Mn	(ppb)	48.0	(0.80)	Rb	(ppb)		
Se	(ppb)	9.402	(1.19)	Sc	(ppb)		
Hg	(ppb)	84.1	(2.79)	Eu	(ppb)	0.405	(0.096)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	72.01	(1.961)				
As	(ppb)	2.433	(0.522)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 16

Field (pool): Chigwell (Viking B) .

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Pamoil et al Chigwell 10-3-41-25

Location: Lsd 10 Sec 3 Twp 41 Rge 25 W 4 Mer

Source of sample: Treater inlet Pressure: 20 psig Temperature: 50 °F

Method of production: Pump Depth: 4592-4595 ft. (perforations)

Date sampled: 24th June 1969

Remarks: Sediment on bottom of sample bottle

S	(%)	0.126	(0.022)				
V	(ppm)	0.250	(0.0072)				
Cl	(ppm)	6.96	(0.512)				
Na	(ppm)	1.324	(0.010)				
Fe	(ppm)						
Ni	(ppm)	0.500	(0.0217)				
Zn	(ppb)	413.6	(10.8)	Au	(ppb)		
Co	(ppb)	2.67	(0.186)	Sb	(ppb)		
I	(ppb)	615.5	(71.5)	Cr	(ppb)		
Mn	(ppb)	41.9	(0.77)	Rb	(ppb)	67.5	(5.80)
Se	(ppb)	6.51	(1.30)	Sc	(ppb)		
Hg	(ppb)	14.74	(0.723)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	14.90	(0.710)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 17

Field (pool): Gilby (Viking A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Colstan Gilby 10-33V-40-2

Location: Lsd 10 Sec 33 Twp40 Rge 2 W 5 Mer

Source of sample: Separator-sight glass Pressure: 27 psig Temperature: 40 °F

Method of production: Pump Depth: 6002-6075 ft. (open hole)

Date sampled: 4th July 1969

Remarks:

S	(%)						
V	(ppm)	0.061	(0.0029)				
Cl	(ppm)	3.585	(0.1267)				
Na	(ppm)	0.874	(0.0068)				
Fe	(ppm)						
Ni	(ppm)	0.053	(0.0138)				
Zn	(ppb)	664.3	(14.44)	Au	(ppb)		
Co	(ppb)	0.805	(0.145)	Sb	(ppb)		
I	(ppb)	7.771	(2.9660)	Cr	(ppb)		
Mn	(ppb)	45.39	(0.8260)	Rb	(ppb)		
Se	(ppb)	4.29	(1.238)	Sc	(ppb)		
Hg	(ppb)	3.473	(0.532)	Eu	(ppb)	0.351	(0.094)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	5.641	(0.693)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS — DATA SHEET

Sample number: 18

Field (pool): Gilby (Viking C)

Stratigraphic unit: Upper Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: HB Richfield Eckville 1

Location: Lsd 10 Sec 2 Twp 40 Rge 3 W 5 Mer

Source of sample: Separator Pressure: 45 psig Temperature: 70°F

Method of production: Flowing Depth: 6270-6280 ft. (perforations)

Date sampled: 18th June 1970

Remarks:

S	(%)	0.128	(0.0163)				
V	(ppm)	0.148	(0.0043)				
Cl	(ppm)	6.524	(0.2861)				
Na	(ppm)	0.945	(0.0168)				
Fe	(ppm)	0.357	(0.161)				
Ni	(ppm)	0.064	(0.0303)				
Zn	(ppb)	480.0	(16.9)	Au	(ppb)		
Co	(ppb)			Sb	(ppb)	0.10	(0.039)
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	55.45	(0.8311)	Rb	(ppb)		
Se	(ppb)	5.75	(2.1540)	Sc	(ppb)	0.241	(0.025)
Hg	(ppb)			Eu	(ppb)	0.324	0.18
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	13.7	(0.778)				
As	(ppb)	1.78	(0.293)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 19

Field (pool): Ferrier (Viking A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Seafort et al Ferrier 10-18-39-7

Location: Lsd 10 Sec 18 Twp 39 Rge 7 W 5 Mer

Source of sample: Tubing Pressure: 400 psig Temperature: 38 °F

Method of production: Flowing Depth: 7927-7930 ft. (perforations)

Date sampled: 29th Oct. 1970

Remarks:

S	(%)	0.040	(0.011)				
V	(ppm)	0.018	(0.0024)				
Cl	(ppm)	4.54	(0.56)				
Na	(ppm)	0.709	(0.0138)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	63.0	(9.3)	Au	(ppb)		
Co	(ppb)			Sb	(ppb)		
I	(ppb)			Cr	(ppb)	10.38	(2.79)
Mn	(ppb)	44.71	(0.77)	Rb	(ppb)		
Se	(ppb)	8.13	(1.952)	Sc	(ppb)	0.24	(0.026)
Hg	(ppb)	202.2	(11.0)	Eu	(ppb)	0.31	(0.164)
Cs	(ppb)	1.32	(0.50)	Ga	(ppb)		
Br	(ppb)	6.67	(0.846)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 20

Field (pool): Willesden Green (Viking A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Mobil Leafland 16-16 MU-40-5

Location: Lsd 16 Sec 16 Twp 40 Rge 5 W 5 Mer

Source of sample: Tubing Pressure: 350 psig Temperature: 39°F

Method of production: Flowing Depth: 7019-7029 ft. (perforations)

Date sampled: 18th Nov. 1970

Remarks:

S	(%)						
V	(ppm)	0.042	(0.0030)				
Cl	(ppm)	5.08	(0.1871)				
Na	(ppm)	0.751	(0.0126)				
Fe	(ppm)						
Ni	(ppm)	0.070	(0.0294)				
Zn	(ppb)	178.0	(10.1)	Au	(ppb)		
Co	(ppb)	0.722	(0.274)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	15.51	(4.43)
Mn	(ppb)	47.23	(0.8345)	Rb	(ppb)		
Se	(ppb)	9.52	(3.0)	Sc	(ppb)	0.22	(0.021)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	6.34	(0.412)				
As	(ppb)	0.831	(0.160)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 21

Field (pool): Caroline (Viking A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Altana Caroline 4-18-35-5

Location: Lsd 4 Sec 18 Twp 35 Rge 5 W 5 Mer

Source of sample: Separator Pressure: 40 psig Temperature: 52 °F

Method of production: Pump Depth: 8383-8389; 8404-8408 ft. (perforations)

Date sampled: 24th April 1970

Remarks:

S	(%)	0.102	(0.0194)				
V	(ppm)	0.365	(0.0103)				
Cl	(ppm)	6.905	(0.1955)				
Na	(ppm)	1.680	(0.018)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	453.0	(10.9)	Au	(ppb)		
Co	(ppb)	1.070	(0.170)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	85.04	(1.658)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.367	(0.072)
Cs	(ppb)	1.24	(0.281)	Ga	(ppb)		
Br	(ppb)	12.1	(0.917)				
As	(ppb)	56.72	(0.947)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 22

Field (pool): Crossfield (Viking A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Viking Fm.

Well name: Cdn-Sup Canberra Cross 10-13-29-2

Location: Lsd 10 Sec 13 Twp 29 Rge 2 W 5 Mer

Source of sample: Tubing Pressure: 140 psig Temperature: 40°F

Method of production: Pump Depth: 7123-7153 ft. (perforations)

Date sampled: 22nd Oct. 1970

Remarks:

S	(%)	0.091	(0.015)				
V	(ppm)	0.128	(0.0063)				
Cl	(ppm)	10.57	(0.3161)				
Na	(ppm)	2.735	(0.0233)				
Fe	(ppm)	0.495	(0.043)				
Ni	(ppm)	0.230	(0.011)				
Zn	(ppb)	273.1	(6.67)	Au	(ppb)		
Co	(ppb)	1.06	(0.084)	Sb	(ppb)	0.530	(0.096)
I	(ppb)	293.5	(8.10)	Cr	(ppb)		
Mn	(ppb)	3.55	(0.154)	Rb	(ppb)		
Se	(ppb)	8.20	(0.832)	Sc	(ppb)	0.028	(0.005)
Hg	(ppb)	1.60	(0.436)	Eu	(ppb)	0.150	(0.034)
Cs	(ppb)	0.310	(0.10)	Ga	(ppb)		
Br	(ppb)	46.0	(1.78)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 24

Field (pool): Cessford (Basal Colorado A)

Stratigraphic unit: U. Cretaceous, Lower Colorado Gp., Basal Colorado Ss.

Well name: HB Unit 2 Cess 10-14-25-12

Location: Lsd 10 Sec 14 Twp 25 Rge 12 W 4 Mer

Source of sample: Wellhead-tubing Pressure: - psig Temperature: - °F

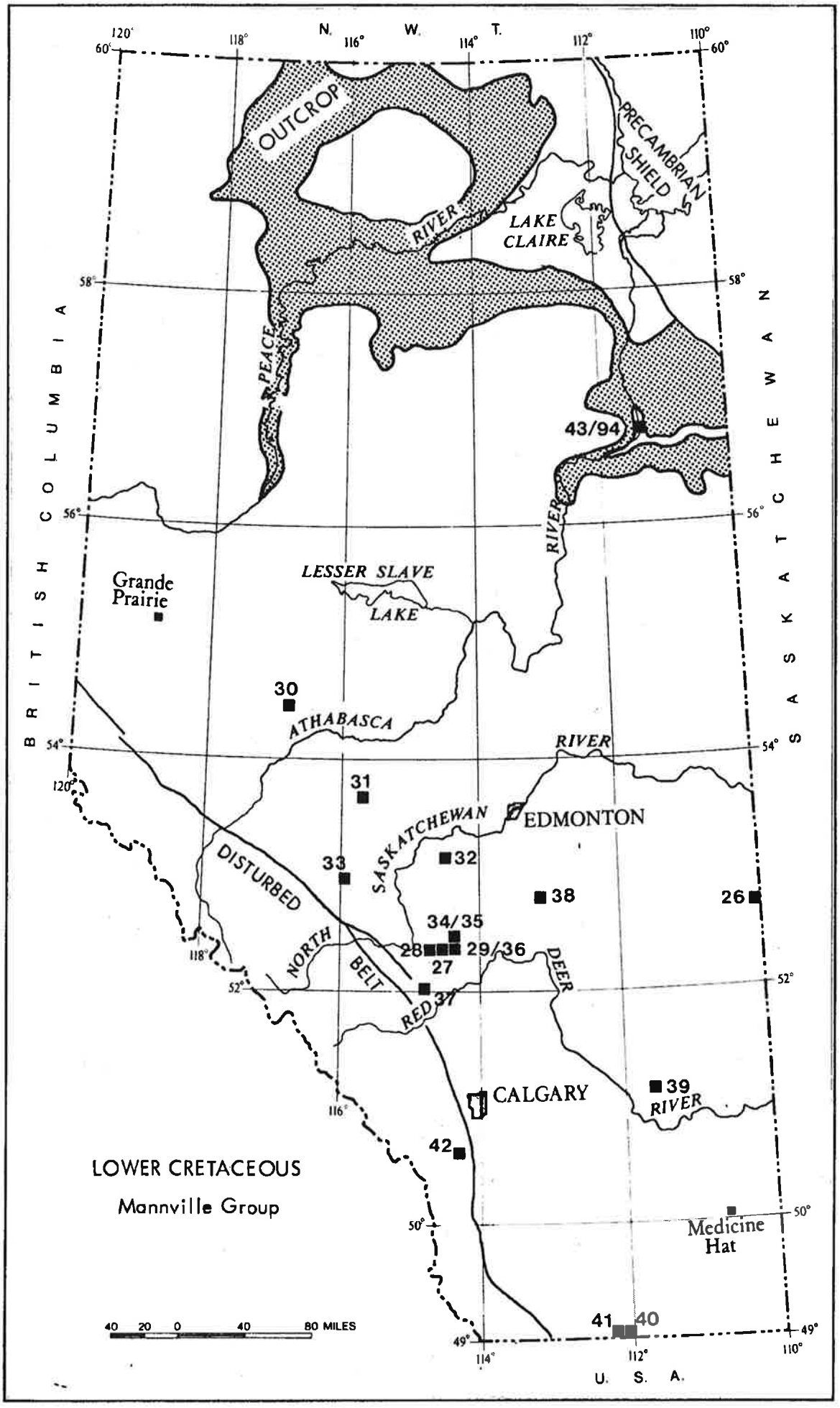
Method of production: Pump Depth: 3046-3052 ft. (perforations)

Date sampled: 12th Aug. 1969

Remarks:

S	(%)	1.514	(0.1519)				
V	(ppm)	59.82	(0.5340)				
Cl	(ppm)	29.21	(0.5583)				
Na	(ppm)	19.3	(0.125)				
Fe	(ppm)	0.39	(0.063)				
Ni	(ppm)	25.2	(0.261)				
Zn	(ppb)	793.5	(10.87)	Au	(ppb)		
Co	(ppb)	58.3	(0.444)	Sb	(ppb)	1.07	(0.103)
I	(ppb)	147.7	(9.806)	Cr	(ppb)	95.2	(2.28)
Mn	(ppb)	42.35	(1.0170)	Rb	(ppb)		
Se	(ppb)	162.0	(4.53)	Sc	(ppb)	0.304	(0.010)
Hg	(ppb)			Eu	(ppb)	0.05	0.028
Cs	(ppb)	0.69	(0.178)	Ga	(ppb)		
Br	(ppb)	177.0	(6.09)				
As	(ppb)	79.8	(3.43)				

* Relative standard deviation (counting statistics) in parenthesis


N. W. T.
OUTCROP
PRECAMBRIAN SHIELD
LAKE CLAIRE
RIVER
PEACE
BRITISH COLUMBIA
SASKATCHEWAN
43/94
RIVER
Grande Prairie
LESSER SLAVE LAKE
30
ATHABASCA
RIVER
31
SASKATCHEWAN
EDMONTON
DISTURBED
33
32
38
26
34/35
28
29/36
27
NORTH
BELT
DEER
RED
37
CALGARY
39
RIVER
42
LOWER CRETACEOUS
Mannville Group
Medicine Hat
41
40
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 26

Field (pool): Chauvin (Mannville A)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Sparky Ss

Well name: Trans-Era CPR Chauvin 16-21-C

Location: Lsd 16 Sec 21 Twp 43 Rge 1 W 4 Mer

Source of sample: Wellhead Pressure: - psig Temperature: - °F

Method of production: Pump Depth: 2152-2171 ft. (perforations)

Date sampled: 9th July 1969

Remarks: Original sample contained formation water

S	(%)	2.591	(0.233)				
V	(ppm)	57.40	(0.430)				
Cl	(ppm)	85.50	(2.0)				
Na	(ppm)						
Fe	(ppm)	1.890	(0.1591)				
Ni	(ppm)	25.05	(0.4282)				
Zn	(ppb)	481.0	(12.7)	Au	(ppb)		
Co	(ppb)	28.2	(0.262)	Sb	(ppb)	1.810	(0.336)
I	(ppb)	185.0	(34.0)	Cr	(ppb)	105.8	(5.87)
Mn	(ppb)	66.2	(1.93)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.764	(0.026)
Hg	(ppb)			Eu	(ppb)	0.385	0.0885
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)						
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 27

Field (pool): Medicine River (Glauconitic A)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Glauconitic Ss.

Well name: HB Medicine R 10-8-39-4

Location: Lsd 10 Sec 8 Twp 39 Rge 4 W 5 Mer

Source of sample: Separator Pressure: 45 psig Temperature: 62°F

Method of production: Flowing Depth: 7361-7365; 7368-7369; 7371-7372; 7379-7381 ft. (perforations)

Date sampled: 18th June 1970

Remarks:

S	(%)	0.759	(0.0457)		
V	(ppm)	4.678	(0.0427)		
Cl	(ppm)	4.886	(0.2261)		
Na	(ppm)	0.728	(0.0134)		
Fe	(ppm)				
Ni	(ppm)	2.00	(0.090)		
Zn	(ppb)	1211.0	(29.0)	Au	(ppb)
Co	(ppb)	3.02	(0.44)	Sb	(ppb)
I	(ppb)			Cr	(ppb)
Mn	(ppb)	23.46	(0.9574)	Rb	(ppb)
Se	(ppb)	49.70	(4.20)	Sc	(ppb)
Hg	(ppb)	4.70	(1.38)	Eu	(ppb)
Cs	(ppb)	1.68	(0.562)	Ga	(ppb)
Br	(ppb)				
As	(ppb)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 28

Field (pool): Willesden Green (Glauconitic A)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Glauconitic Ss.

Well name: CPOG Leaf 8-15-39-5

Location: Lsd 8 Sec 15 Twp 39 Rge 5 W 5 Mer

Source of sample: Separator Pressure: 40 psig Temperature: 52 °F

Method of production: Pump Depth: 7550-7553; 7570-7573 ft. (perforations)

Date sampled: 22nd Oct. 1970

Remarks:

S	(%)	0.751	(0.0780)				
V	(ppm)	8.401	(0.0968)				
Cl	(ppm)	10.33	(0.2849)				
Na	(ppm)	1.84	(0.0312)				
Fe	(ppm)						
Ni	(ppm)	2.712	(0.069)				
Zn	(ppb)	392.0	(14.4)	Au	(ppb)		
Co	(ppb)	3.135	(0.336)	Sb	(ppb)		
I	(ppb)	34.22	(6.1210)	Cr	(ppb)		
Mn	(ppb)	79.28	(1.2080)	Rb	(ppb)		
Se	(ppb)	50.72	(3.096)	Sc	(ppb)	0.183	(0.0211)
Hg	(ppb)	63.75	(5.89)	Eu	(ppb)		
Cs	(ppb)	1.395	(0.473)	Ga	(ppb)		
Br	(ppb)						
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 29

Field (pool): Medicine River (Ostracod A)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Ostracod Mbr.

Well name: Atlantic Medicine River 14-10-39-3

Location: Lsd 14 Sec 10 Twp 39 Rge 3 W 5 Mer

Source of sample: Wellhead Pressure: 155 psig Temperature: 50 °F

Method of production: Pump Depth: 6842-6850 ft. (perforations)

Date sampled: 18th June 1970

Remarks: Some silt and formation water in original container

S	(%)	1.115	(0.0777)				
V	(ppm)	8.336	(0.0910)				
Cl	(ppm)	4.497	(0.1889)				
Na	(ppm)	0.226	(0.0036)				
Fe	(ppm)	0.286	(0.122)				
Ni	(ppm)	4.04	(0.273)				
Zn	(ppb)	104.0	(8.45)	Au	(ppb)		
Co	(ppb)	3.392	(0.375)	Sb	(ppb)		
I	(ppb)	18.61	(5.430)	Cr	(ppb)	13.60	(0.30)
Mn	(ppb)	46.21	(1.212)	Rb	(ppb)		
Se	(ppb)	49.20	(3.90)	Sc	(ppb)		
Hg	(ppb)	4.10	(1.30)	Eu	(ppb)	0.430	(0.109)
Cs	(ppb)	1.05	(0.44)	Ga	(ppb)		
Br	(ppb)	3.10	(0.410)				
As	(ppb)	1.17	(0.148)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 30

Field (pool): Kaybob (Cadomin B)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Cadomin Fm.

Well name: Atlantic Kaybob 16-21-63-19

Location: Lsd 16 Sec 21 Twp 63 Rge 19 W 5 Mer

Source of sample: Tubing Pressure: 50 psig Temperature: 55 °F

Method of production: Pump Depth: 5920-5954; 5961-5974 ft. (perforations)

Date sampled: 21st July 1970

Remarks: Some silt and formation water in original container

S	(%)	1.655	(0.1134)				
V	(ppm)	27.68	(0.2754)				
Cl	(ppm)	8.922	(0.2326)				
Na	(ppm)	2.043	(0.0420)				
Fe	(ppm)						
Ni	(ppm)	10.4	(0.124)				
Zn	(ppb)	645.3	(20.5)	Au	(ppb)		
Co	(ppb)	6.93	(0.605)	Sb	(ppb)		
I	(ppb)	103.8	(7.159)	Cr	(ppb)		
Mn	(ppb)	67.73	(1.002)	Rb	(ppb)		
Se	(ppb)	47.4	(5.20)	Sc	(ppb)	0.06	(0.028)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	1.92	(0.57)	Ce	(ppb)		
Br	(ppb)	29.2	(3.04)				
As	(ppb)	10.4	(1.10)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 31

Field (pool): Niton (Basal Quartz B)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Ellerslie Fm.

Well name: IOE: Niton 4-18-54-12

Location: Lsd 4 Sec 18 Twp 54 Rge 12 W 5 Mer

Source of sample: Tubing Pressure: - psig Temperature: - °F

Method of production: Pump Depth: 6515-6524 ft. (perforations)

Date sampled: 20th Feb. 1970

Remarks: Sample may contain emulsion breaker; this was being injected downstream of sampling point; however, treated oil may have flowed back. Some silt and formation water in original container

S	(%)	0.508	(0.036)				
V	(ppm)	1.04	(0.019)				
Cl	(ppm)	43.9	(0.584)				
Na	(ppm)	1.31	(0.0137)				
Fe	(ppm)	1.89	(0.101)				
Ni	(ppm)	0.212	(0.011)				
Zn	(ppb)	41.1	(2.50)	Au	(ppb)		
Co	(ppb)	0.992	(0.0611)	Sb	(ppb)	0.530	(0.063)
I	(ppb)	9005.0	(131.0)	Cr	(ppb)		
Mn	(ppb)	1.70	(0.20)	Rb	(ppb)		
Se	(ppb)	7.07	(0.83)	Sc	(ppb)	0.021	(0.005)
Hg	(ppb)	138.3	(6.79)	Eu	(ppb)	0.103	(0.029)
Cs	(ppb)	0.32	(0.114)	Ga	(ppb)		
Br	(ppb)	1153.0	(15.6)				
As	(ppb)	40.9	(1.65)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 32

Field (pool): Pembina (Keystone Ellerslie A)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Ellerslie Fm.

Well name: Cities Service Keystone 6-32

Location: Lsd 6 Sec 32 Twp 48 Rge 4 W 5 Mer

Source of sample: Separator Pressure: 60 psig Temperature: cold °F

Method of production: Flowing Depth: Ellerslie 5839 ft.
Carboniferous 5910 ft.

Date sampled: 4th Feb. 1970

Remarks: Sample from perforations, but depth not given

S	(%)	0.928	(0.0784)				
V	(ppm)	3.015	(0.0359)				
Cl	(ppm)	7.108	(0.2320)				
Na	(ppm)	0.317	(0.0031)				
Fe	(ppm)	0.50	(0.043)				
Ni	(ppm)	0.80	(0.018)				
Zn	(ppb)	21.6	(2.10)	Au	(ppb)		
Co	(ppb)	1.25	(0.078)	Sb	(ppb)		
I	(ppb)	10.28	(5.686)	Cr	(ppb)		
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)	32.7	(1.47)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.091	(0.030)
Cs	(ppb)	0.355	(0.112)	Ga	(ppb)		
Br	(ppb)	37.62	(0.823)				
As	(ppb)	4.17	(0.23)				

* Relative standard deviation (counting statistics) in parentheses

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 33

Field (pool): Brazeau River (Lower Mannville A)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp

Well name: HB BrazR 10-9-46-14

Location: Lsd 10 Sec 9 Twp 46 Rge 14 W 5 Mer

Source of sample: Separator Pressure: 44 psig Temperature: 30 °F

Method of production: Flowing Depth: 10237-10246; 10247-10249 ft. (perforations)

Date sampled: 19th Feb. 1970

Remarks: Some silt at bottom of original container

S	(%)						
V	(ppm)						
Cl	(ppm)	0.650	(0.074)				
Na	(ppm)	0.249	(0.0022)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	0.397	(0.1451)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)	5.26	(1.303)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.250	(0.0873)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	22.06	(0.633)				
As	(ppb)	33.4	(0.495)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 34

Field (pool): Gilby (Basal Mannville B)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp.

Well name: Richfield Gabriel Lake 7-29

Location: Lsd 7 Sec 29 Twp 40 Rge 3 W 5 Mer

Source of sample: Separator Pressure: 52 psig Temperature: 56°F

Method of production: Pump Depth: 7015-7035 ft. (perforations)

Date sampled: 4th July 1969

Remarks:

S	(%)	1.534	(0.134)				
V	(ppm)	27.6	(0.278)				
Cl	(ppm)	21.8	(0.798)				
Na	(ppm)						
Fe	(ppm)						
Ni	(ppm)	9.0	(0.146)				
Zn	(ppb)	400.0	(16.0)	Au	(ppb)		
Co	(ppb)	5.40	(0.31)	Sb	(ppb)		
I	(ppb)	193.0	(12.12)	Cr	(ppb)	30.0	(3.77)
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)	49.2	(6.74)	Sc	(ppb)	0.20	(0.0161)
Hg	(ppb)	1.93	(0.406)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	5.7	(0.26)
Br	(ppb)	120.0	(2.84)				
As	(ppb)	5.562	(0.356)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 36

Field (pool): Medicine River (Basal Quartz B)

Stratigraphic unit: L. Cretaceous, Mannville Gp., Ellerslie Fm.

Well name: Hudson's Bay Medicine R 10-28-39-3

Location: Lsd 10 Sec 28 Twp 39 Rge 3 W 5 Mer

Source of sample: Tubing Pressure: 1235 psig Temperature: 40 °F

Method of production: Flowing Depth: 6984-6990 ft.

Date sampled: 22nd Oct. 1970

Remarks:

S	(%)	1.567	(0.1163)		
V	(ppm)	20.88	(0.1983)		
Cl	(ppm)	5.034	(0.7802)		
Na	(ppm)	1.766	(0.0122)		
Fe	(ppm)				
Ni	(ppm)	8.06	(0.202)		
Zn	(ppb)	85.31	(21.00)	Au	(ppb)
Co	(ppb)	3.57	(1.00)	Sb	(ppb)
I	(ppb)			Cr	(ppb)
Mn	(ppb)	50.76	(1.094)	Rb	(ppb)
Se	(ppb)	80.2	(9.72)	Sc	(ppb)
Hg	(ppb)		(9.72)	Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	47.4	(2.40)		
As	(ppb)	2.78	(0.510)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 37

Field (pool): Caroline (Basal Mannville B)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp.

Well name: Altana Caroline 4-2-35-6

Location: Lsd 4 Sec 2 Twp 35 Rge 6 W 5 Mer

Source of sample: Separator Pressure: 1050 psig Temperature: 16 °F

Method of production: Flowing Depth: 9454-9459 ft.

Date sampled: 12th Nov. 1970

Remarks:

S	(%)						
V	(ppm)						
Cl	(ppm)	0.634	(0.135)				
Na	(ppm)	0.033	(0.001)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	17.5	(5.56)	Au	(ppb)		
Co	(ppb)	0.776	(0.154)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	4.66	(1.36)
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.290	(0.106)
Cs	(ppb)	0.486	(0.241)	Ga	(ppb)		
Br	(ppb)	1.75	(0.133)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 38

Field (pool): Malmo (Blairmore A)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp.

Well name: Sun M and C Malmo 6-14B-44-22

Location: Lsd 6 Sec 14 Twp 44 Rge 22 W 4 Mer

Source of sample: Tubing Pressure: 35 psig Temperature: 20°F

Method of production: Pump Depth: 4708-4721 ft. (open hole)

Date sampled: 3rd Nov. 1970

Remarks: Formation water and emulsion on bottom of original container

S	(%)	0.736	(0.0550)				
V	(ppm)	1.140	(0.0156)				
Cl	(ppm)	8.552	(0.2572)				
Na	(ppm)	0.927	(0.0070)				
Fe	(ppm)	0.507	(0.116)				
Ni	(ppm)	1.342	(0.0411)				
Zn	(ppb)	31.9	(6.56)	Au	(ppb)		
Co	(ppb)	2.30	(0.244)	Sb	(ppb)		
I	(ppb)	19.01	(5.084)	Cr	(ppb)	9.52	(3.09)
Mn	(ppb)	72.76	(1.276)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.518	(0.1109)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	26.6	(1.19)				
As	(ppb)	3.36	(0.433)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 39

Field (pool): Cessford (Mannville C)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp.

Well name: HB Delhi Cessford 1-27-25-12

Location: Lsd 1 Sec 27 Twp 25 Rge 12 W 4 Mer

Source of sample: Tubing Pressure: 212 psig Temperature: 44°F

Method of production: Pump Depth: 3364-3370 ft. (perforations)

Date sampled: 11th March 1970

Remarks: Formation water and emulsion on bottom of original container

S	(%)	1.553	(0.1276)				
V	(ppm)	28.58	(0.2984)				
Cl	(ppm)	25.50	(1.114)				
Na	(ppm)	0.018	(0.0002)				
Fe	(ppm)	1.135	(0.185)				
Ni	(ppm)	14.96	(0.242)				
Zn	(ppb)	510.0	(20.3)	Au	(ppb)		
Co	(ppb)	17.34	(0.69)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	41.0	(6.18)
Mn	(ppb)	60.11	(1.553)	Rb	(ppb)		
Se	(ppb)	127.2	(7.57)	Sc	(ppb)	0.023	(0.0066)
Hg	(ppb)			Eu	(ppb)	0.661	(0.141)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)						
As	(ppb)	0.020					

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 40

Field (pool): Coutts (Moulton B)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp., Moulton Ss.

Well name: Camac Mav Coutts 3-28-1-16

Location: Lsd 3 Sec 28 Twp 1 Rge 16 W 4 Mer

Source of sample: Tubing Pressure: 25 psig Temperature: cold °F

Method of production: Pump Depth: 2505-2514 ft.

Date sampled: 25th March 1970

Remarks:

S	(%)	1.235	(0.0852)				
V	(ppm)	11.75	(0.1152)				
Cl	(ppm)	4.312	(0.1820)				
Na	(ppm)	0.398	(0.0046)				
Fe	(ppm)						
Ni	(ppm)	1.80	(0.043)				
Zn	(ppb)	821.0	(15.85)	Au	(ppb)		
Co	(ppb)	1.984	(0.1763)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	47.18	(0.9589)	Rb	(ppb)		
Se	(ppb)	25.06	(2.28)	Sc	(ppb)		
Hg	(ppb)	2.02	(0.834)	Eu	(ppb)	0.214	(0.0850)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	2.19	(0.345)				
As	(ppb)	46.6	(0.662)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 41

Field (pool): Red Coulee (Cut Bank B)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp., Cut Bank Ss.

Well name: Salt Dome 2 Milk River 12-3-1-17

Location: Lsd 12 Sec 3 Twp 1 Rge 17 W 4 Mer

Source of sample: Tubing Pressure: 100 psig Temperature: cold °F

Method of production: Pump Depth: 2733-2743 ft. (perforations)

Date sampled: 11th Aug. 1970

Remarks:

S	(%)	1.686	(0.1208)				
V	(ppm)	38.00	(0.4020)				
Cl	(ppm)	6.406	(0.2465)				
Na	(ppm)	0.685	(0.0116)				
Fe	(ppm)	0.534	(0.1194)				
Ni	(ppm)	13.44	(0.232)				
Zn	(ppb)	1277.0	(19.8)	Au	(ppb)		
Co	(ppb)	8.754	(1.086)	Sb	(ppb)	3.030	(0.332)
I	(ppb)			Cr	(ppb)	36.12	3.70
Mn	(ppb)	64.96	(0.9730)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	0.940	(0.40)	Ga	(ppb)		
Br	(ppb)	9.90	(1.27)				
As	(ppb)	1988.0	(12.4)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 42

Field (pool): Turner Valley (-)

Stratigraphic unit: L. Cretaceous, Blairmore Gp.

Well name: Miracle 3

Location: Lsd 11 Sec 4 Twp 19 Rge 2 W 5 Mer

Source of sample: Tank-thief hatch Pressure: nil psig Temperature: 80 °F

Method of production: Pump Depth: 4787 ft.

Date sampled: 25th June 1970

Remarks: This well only produces periodically. The sample came from the top of the tank and should be new oil. Last produced 22nd June, 1970.

S	(%)	0.085	(0.0204)				
V	(ppm)	0.017	(0.0023)				
Cl	(ppm)	3.854	(0.1591)				
Na	(ppm)	0.351	(0.0086)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	44.7	(5.17)	Au	(ppb)		
Co	(ppb)	2.13	(0.322)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	58.67	(0.9010)	Rb	(ppb)		
Se	(ppb)	20.9	(2.0)	Sc	(ppb)	0.056	(0.016)
Hg	(ppb)			Eu	(ppb)	0.377	(0.135)
Cs	(ppb)	1.45	(0.374)	Ga	(ppb)	9.43	(0.783)
Br	(ppb)	15.12	(1.033)				
As	(ppb)	46.18	(1.24)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 43

Field (pool): Athabasca oil sand deposit

Stratigraphic unit: L. Cretaceous, Mannville Gp., McMurray Fm.

Well name: -

Location: Lsd - Sec - Twp - Rge - W -Mer

Source of sample: - Pressure: - psig Temperature: - °F

Method of production: - Depth: - ft.

Date sampled: -

Remarks:

S	(%)						
V	(ppm)						
Cl	(ppm)						
Na	(ppm)	40.33	(0.353)				
Fe	(ppm)	141.7	(4.48)				
Ni	(ppm)	74.11	(1.43)				
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	1349.0	(52.6)	Sb	(ppb)	30.61	(4.77)
I	(ppb)			Cr	(ppb)	1014.0	(31.3)
Mn	(ppb)			Rb	(ppb)	377.5	(91.3)
Se	(ppb)	286.4	(26.72)	Sc	(ppb)	199.0	(2.13)
Hg	(ppb)	81.7	(19.4)	Eu	(ppb)	9.0	(1.3)
Cs	(ppb)	25.9	(5.80)	Ga	(ppb)	315.0	(11.3)
Br	(ppb)	155.3	(23.42)				
As	(ppb)	400.3	(13.4)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 35

Field (pool): Gilby (Basal Mannville B)

Stratigraphic unit: L. Cretaceous, Lower Mannville Gp.

Well name: Richfield Gabriel Lake 7-29

Location: Lsd 7 Sec 29 Twp 40 Rge 3 W 5 Mer

Source of sample: Wellhead Pressure: 90 psig Temperature: 60 °F

Method of production: Pump Depth: 7015-7035 ft. (perforations)

Date sampled: 2nd July 1970

Remarks:

S	(%)	1.391	(0.112)		
V	(ppm)	36.0	(0.32)		
Cl	(ppm)	34.5	(0.840)		
Na	(ppm)	3.545	(0.028)		
Fe	(ppm)	0.593	(0.161)		
Ni	(ppm)	13.32	(0.214)		
Zn	(ppb)	262.2	(13.0)	Au	(ppb)
Co	(ppb)	5.524	(0.395)	Sb	(ppb)
I	(ppb)	179.2	(30.9)	Cr	(ppb)
Mn	(ppb)	52.9	(0.882)	Rb	(ppb)
Se	(ppb)	143.6	(8.31)	Sc	(ppb)
Hg	(ppb)			Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	82.4	(2.8)		
As	(ppb)	9.9	(1.167)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

ADDITIONAL DATA - Sample No. 43

Oil sand sample from Syncrude Canada Ltd. Extracted in Soxhlet extractor with benzene, centrifuged, and evaporated down at room temperature.

Asphaltenes: 17.1%

Composition of asphaltenes:

C	79.41%
H	8.03%
N	1.89%
O (direct)	3.11%
S	7.56%
Ash	7.11%

EPR of total oil:

Number of spins/gm	6.1×10^{17}
g-value	2.00320
Response width	6.4 gauss
Total V^{4+}	483 μg/g

EPR of asphaltenes:

Number of spins/gm	2.6×10^{18}
g-value	2.00305
Response width	6.4 gauss
Total V^{4+}	1740 μg/g

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 44

Field (pool): Gilby (Jurassic B)

Stratigraphic unit: L. Jurassic, Nordegg Fm.

Well name: Uno-Tex Gilby 4-30-40-3

Location: Lsd 4 Sec 30 Twp 40 Rge 3 W 5 Mer

Source of sample: Wellhead Pressure: 150 psig Temperature: 50 °F

Method of production: Pump Depth: 7055-7062; 7066-7076 ft. (perforations)

Date sampled: 2nd July, 1970

Remarks:

S	(%)	1.561	(0.1144)				
V	(ppm)	32.49	(0.3319)				
Cl	(ppm)	12.28	(0.7645)				
Na	(ppm)	2.292	(0.022)				
Fe	(ppm)	0.783	(0.138)				
Ni	(ppm)	11.15	(0.147)				
Zn	(ppb)	401.1	(7.56)	Au	(ppb)		
Co	(ppb)	4.90	(0.367)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	18.95	(3.43)
Mn	(ppb)	28.48	(0.7504)	Rb	(ppb)		
Se	(ppb)	55.22	(4.20)	Sc	(ppb)	0.045	(0.015)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ce	(ppb)	11.0	(0.677)
Br	(ppb)	150.0	(3.75)				
As	(ppb)	23.60	(1.082)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 45

Field (pool): Medicine River (Jurassic A)

Stratigraphic unit: L. Jurassic, Nordegg Fm.

Well name: HB RO Corp Medicine R 7-32-39-3

Location: Lsd 7 Sec 32 Twp 39 Rge 3 W 5 Mer

Source of sample: Wellhead Pressure: 31 psig Temperature: 50 °F

Method of production: Pump Depth: 7036-7056 ft. (perforations)

Date sampled: 18th June 1970

Remarks:

S	(%)	1.438	(0.1252)				
V	(ppm)	33.45	(0.3392)				
Cl	(ppm)	8.967	(0.3249)				
Na	(ppm)	0.725	(0.0121)				
Fe	(ppm)	0.580	(0.19)				
Ni	(ppm)	14.36	(0.317)				
Zn	(ppb)	651.0	(19.2)	Au	(ppb)		
Co	(ppb)	7.69	(0.665)	Sb	(ppb)		
I	(ppb)	44.39	(7.266)	Cr	(ppb)	16.5	(4.13)
Mn	(ppb)	49.80	(0.9502)	Rb	(ppb)		
Se	(ppb)	94.20	(5.29)	Sc	(ppb)	0.090	(0.0277)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	1.59	(0.583)	Ga	(ppb)		
Br	(ppb)	36.94	(1.69)				
As	(ppb)	8.64	(0.530)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 46

Field (pool): Conrad (Ellis)

Stratigraphic unit: L. Jurassic, Ellis Gp., Sawtooth Fm.

Well name: Joe Phillips Conrad 7-4-6-15

Location: Lsd 7 Sec 4 Twp 6 Rge 15 W 4 Mer

Source of sample: Wellhead - tubing Pressure: _ psig Temperature: _ °F

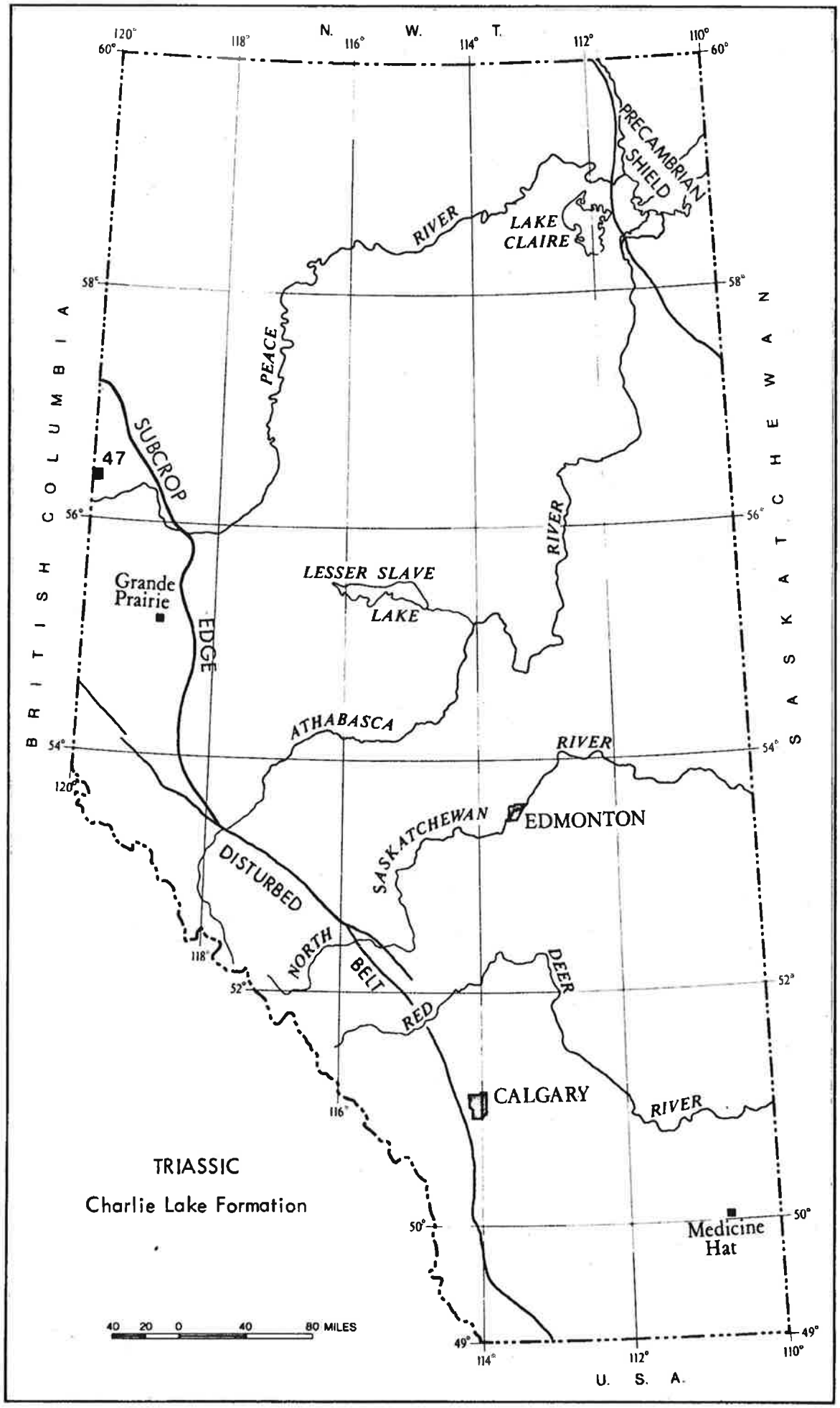
Method of production: Flowing Depth: 2990-2992 ft. (perforations)

Date sampled: 12th August, 1969

Remarks: Considerable amount of sand and silt in original container.

S	(%)	2.445	(0.224)				
V	(ppm)	71.84	(0.572)				
Cl	(ppm)	10.40	(0.308)				
Na	(ppm)	14.52	(0.2801)				
Fe	(ppm)						
Ni	(ppm)	24.87	(0.296)				
Zn	(ppb)	119.0	(10.6)	Au	(ppb)		
Co	(ppb)	16.82	(0.616)	Sb	(ppb)		
I	(ppb)	25.37	(6.63)	Cr	(ppb)	98.11	(7.88)
Mn	(ppb)	38.6	(0.931)	Rb	(ppb)		
Se	(ppb)	139.8	(7.463)	Sc	(ppb)	0.20	(0.0253)
Hg	(ppb)			Eu	(ppb)	0.571	(0.161)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	40.30	(12.61)				
As	(ppb)	235.4	(9.732)				

* Relative standard deviation (counting statistics) in parenthesis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PRECAMBRIAN SHIELD
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Grande Prairie
EDGE
LESSER SLAVE LAKE
RIVER
ATHABASCA
RIVER
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH
BELT
DEER
RED
RIVER
CALGARY
Medicine Hat
TRIASSIC
Charlie Lake Formation
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 47

Field (pool): Boundary Lake South (Triassic E)

Stratigraphic unit: U. Triassic, Charlie Lake Fm., Boundary Mbr.

Well name: Imp. Pan Am Bdy Lk S 6-11-85-13

Location: Lsd 6 Sec 11 Twp 85 Rge 13 W6 Mer

Source of sample: Tubing Pressure: 100 psig Temperature: 50 °F

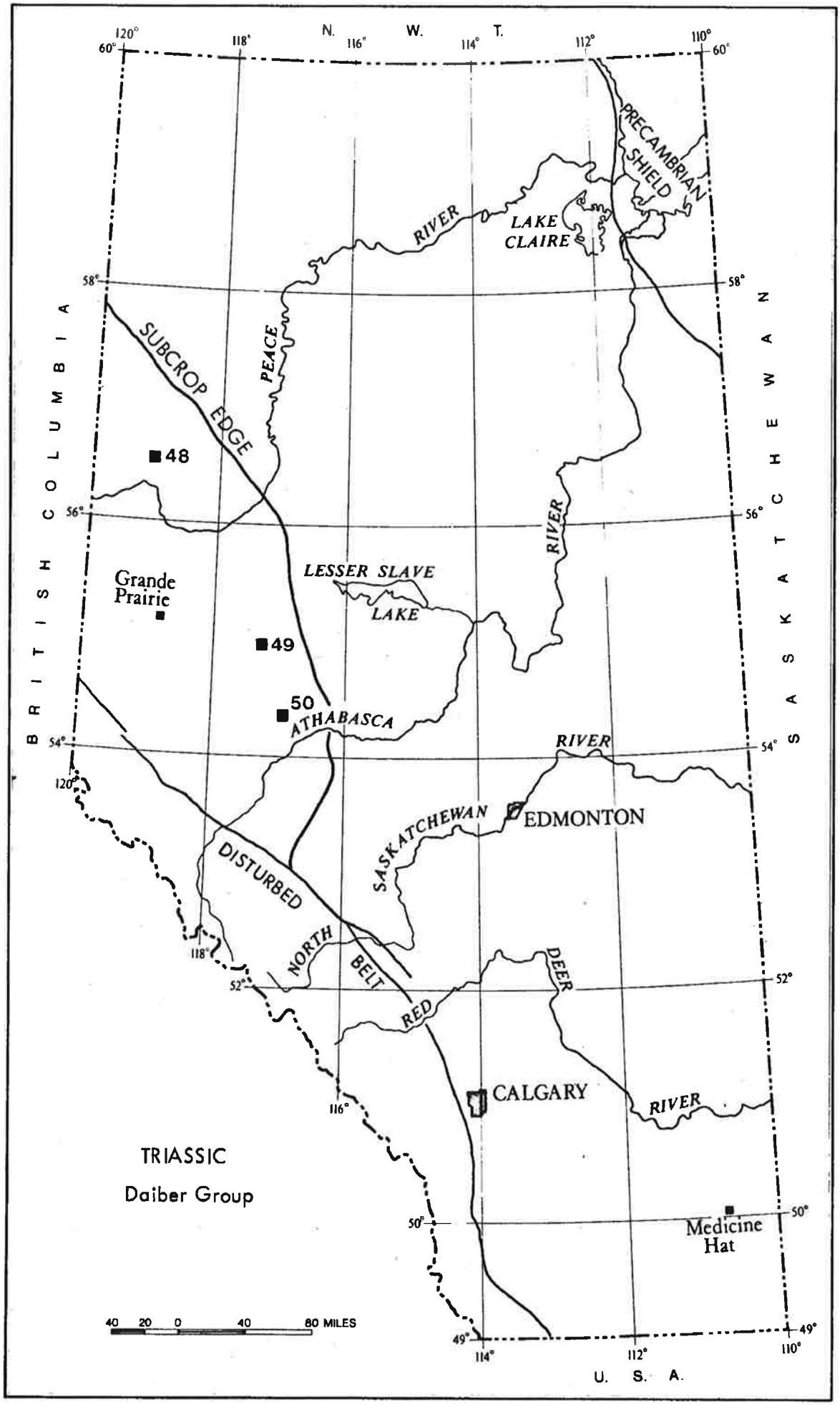
Method of production: Pump Depth: 4354-4360 ft. (perforations)

Date sampled: 23rd July 1970

Remarks:

S	(%)	1.030	(0.0862)				
V	(ppm)	47.39	(0.4054)				
Cl	(ppm)	13.22	(0.4575)				
Na	(ppm)	4.367	(0.0716)				
Fe	(ppm)	2.44	(0.100)				
Ni	(ppm)	14.2	(0.214)				
Zn	(ppb)	924.1	(16.82)	Au	(ppb)		
Co	(ppb)	14.50	(0.118)	Sb	(ppb)	1.03	(0.172)
I	(ppb)	99.64	(8.361)	Cr	(ppb)	31.47	(2.09)
Mn	(ppb)	76.35	(1.448)	Rb	(ppb)		
Se	(ppb)	161.0	(6.87)	Sc	(ppb)	0.112	(0.010)
Hg	(ppb)			Eu	(ppb)	0.28	0.050
Cs	(ppb)	1.232	(0.261)	Ga	(ppb)		
Br	(ppb)	54.15	(3.69)				
As	(ppb)	244.3	(5.20)				

* Relative standard deviation (counting statistics) in parenthesis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LESSER SLAVE LAKE
RIVER
ATHABASCA
RIVER
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH
BELT
RED
DEER
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CALGARY
TRIASSIC
Daiber Group
Medicine Hat
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 48

Field (pool): Worsley (Triassic A)

Stratigraphic unit: Triassic, Daiber Gp.

Well name: Shell Worsley 6-25-87-7

Location: Lsd 6 Sec 25 Twp 87 Rge 7 W 6 Mer

Source of sample: Treater Pressure: 5 psig Temperature: 60 °F

Method of production: - Depth: 3293-3304 ft. (perforations)

Date sampled: 23rd July 1970

Remarks: The well has been shut-in since February, 1970, so this is a February sample, stored for 5 months in the treater. The following chemicals were added to this oil: Treatolite R25, 1 quart per 50 bbls; Alchem 339, 1 bbl per 250 bbls.

S	(%)	1.397	(0.1164)				
V	(ppm)	23.66	(0.2387)				
Cl	(ppm)	10.48	(0.4518)				
Na	(ppm)	0.203	(0.0047)				
Fe	(ppm)						
Ni	(ppm)	10.78	(0.27)				
Zn	(ppb)	965.5	(28.8)	Au	(ppb)		
Co	(ppb)	7.64	(0.723)	Sb	(ppb)		
I	(ppb)	59.51	(9.646)	Cr	(ppb)	24.7	(5.531)
Mn	(ppb)	40.94	(1.274)	Rb	(ppb)		
Se	(ppb)	70.0	(5.94)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	32.43	(1.26)				
As	(ppb)	5.25	(0.360)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 49

Field (pool): Sturgeon Lake South (Triassic A)

Stratigraphic unit: Triassic, Daiber Gp.

Well name: Amerada HB Union Crown Y 5-12

Location: Lsd 5 Sec 12 Twp 69 Rge 22 W 5 Mer

Source of sample: Treater Pressure: 30 psig Temperature: 80 °F

Method of production: Pump Depth: 4934-4944 ft. (perforations)

Date sampled: 10th July 1969

Remarks:

S	(%)	1.138	(0.11)				
V	(ppm)	26.03	(0.27)				
Cl	(ppm)	7.96	(0.301)				
Na	(ppm)	0.347	(0.0032)				
Fe	(ppm)						
Ni	(ppm)	6.66	(0.077)				
Zn	(ppb)	1274.0	(14.4)	Au	(ppb)		
Co	(ppb)	1.53	(0.075)	Sb	(ppb)		
I	(ppb)	150.0	(9.1)	Cr	(ppb)	13.0	(1.88)
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)	36.9	(2.0)	Sc	(ppb)		
Hg	(ppb)	6.34	(1.78)	Eu	(ppb)	0.084	(0.025)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	27.9	(0.867)				
As	(ppb)	8.50	(0.372)				

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 50

Field (pool): Kaybob South (Triassic A)

Stratigraphic unit: Triassic, Daiber Gp.

Well name: HB Union Kaybob S 10-9-62-20

Location: Lsd 10 Sec 9 Twp 62 Rge 20 W5 Mer

Source of sample: Tubing Pressure: - psig Temperature: 60 °F

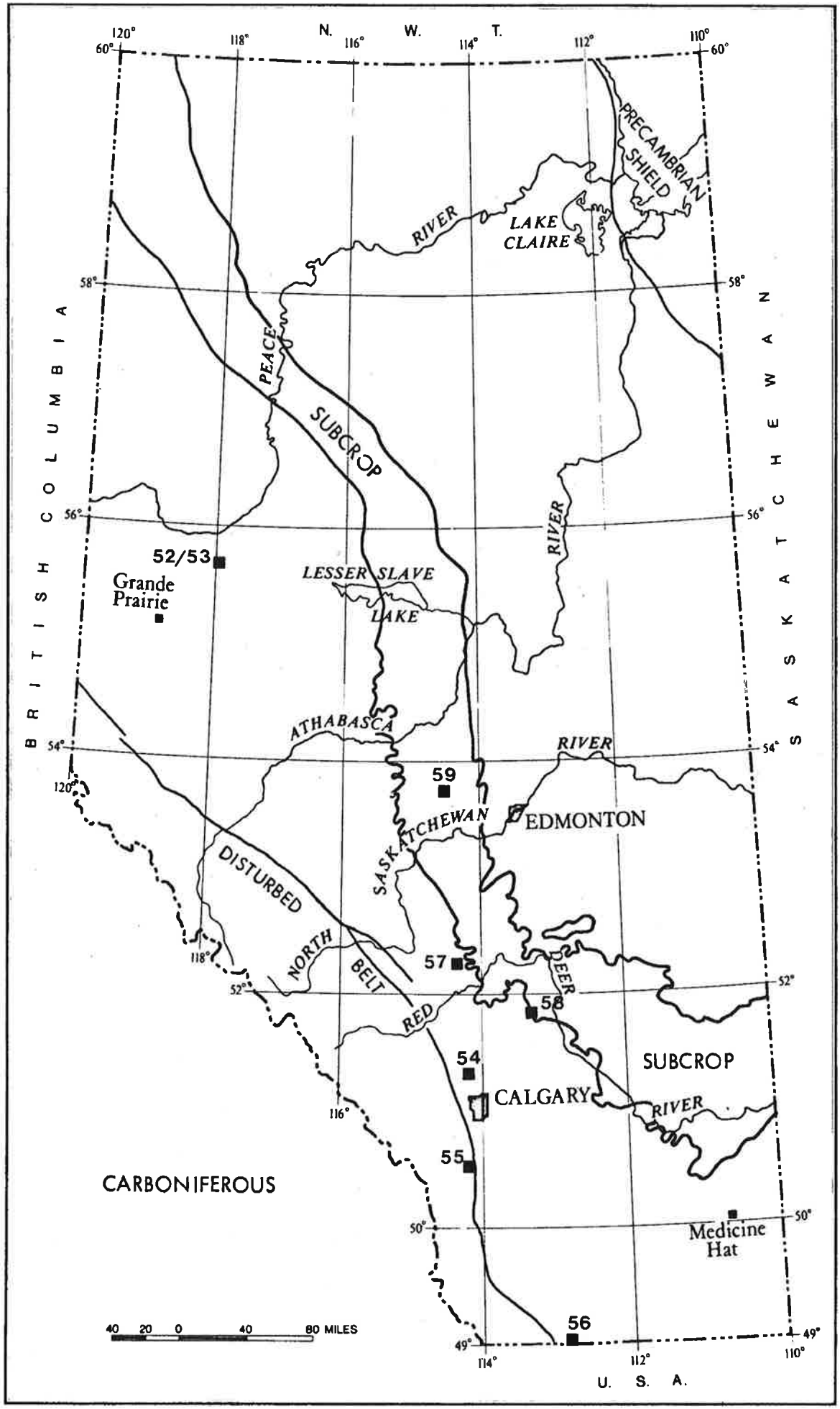
Method of production: Flowing Depth: 6942-6969 ft. (perforations)

Date sampled: 21st July 1970

Remarks:

S	(%)	0.667	(0.0443)				
V	(ppm)	0.185	(0.0046)				
Cl	(ppm)	10.29	(0.236)				
Na	(ppm)	0.119	(0.0026)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	53.0	(7.7)	Au	(ppb)		
Co	(ppb)	0.696	(0.261)	Sb	(ppb)		
I	(ppb)	14.21	(3.075)	Cr	(ppb)		
Mn	(ppb)	24.30	(0.582)	Rb	(ppb)		
Se	(ppb)	8.270	(2.122)	Sc	(ppb)	0.150	(0.020)
Hg	(ppb)	3.79	(1.16)	Eu	(ppb)		
Cs	(ppb)	1.391	(0.4304)	Ga	(ppb)		
Br	(ppb)	4.70	(0.336)				
As	(ppb)	2.443	(0.169)				

* Relative standard deviation (counting statistics) in parenthesis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PRECAMBRIAN SHIELD
LAKE CLAIRE
RIVER
PEACE
SUBCROP
52/53
Grande Prairie
LESSER SLAVE LAKE
ATHABASCA
RIVER
59
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH BELT
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BEER
RED
58
54
CALGARY
SUBCROP
RIVER
55
CARBONIFEROUS
Medicine Hat
56
40 20 0 40 80 MILES
U. S. A.

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 52
Field (pool): Eaglesham (Debolt D)
Stratigraphic unit: Carboniferous, Rundle Gp., Debolt Fm.
Well name: HB Union Eaglesham 6-14 MU 77-25
Location: Lsd 6 Sec 14 Twp 77 Rge 25 W 5 Mer
Source of sample: Flowline Pressure: 100 psig Temperature: 65 °F
Method of production: Pump Depth: 4900-4927 ft. (perforations)
Date sampled: 5th Aug. 1970
Remarks: Silt at bottom of original container

S	(%)	0.693	(0.0482)				
V	(ppm)	3.871	(0.0385)				
Cl	(ppm)	4.701	(0.1687)				
Na	(ppm)	3.07	(0.029)				
Fe	(ppm)	0.120	(0.041)				
Ni	(ppm)	5.18	(0.066)				
Zn	(ppb)	432.5	(7.83)	Au	(ppb)		
Co	(ppb)	0.811	(0.095)	Sb	(ppb)		
I	(ppb)	27.50	(3.247)	Cr	(ppb)		
Mn	(ppb)	53.26	(1.094)	Rb	(ppb)		
Se	(ppb)	13.4	(1.39)	Sc	(ppb)	0.013	(0.006)
Hg	(ppb)	1.9	(0.79)	Eu	(ppb)	0.11	(0.0373)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	76.7	(3.8)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 53

Field (pool): Eaglesham (Debolt C)

Stratigraphic unit: Carboniferous, Rundle Gp., Debolt Fm.

Well name: HB Union Eaglesham 6-14-MU-77-25

Location: Lsd 6 Sec 14 Twp 77 Rge 25 W 5 Mer

Source of sample: Casing Pressure: 100 psig Temperature: 60 °F

Method of production: Pump Depth: 4684-4721 ft. (perforations)

Date sampled: 22nd July 1970

Remarks: Well shut in at time of sampling; normally produces from tubing

S	(%)	0.382	(0.0752)		
V	(ppm)	1.893	(0.033)		
Cl	(ppm)	13.81	(0.723)		
Na	(ppm)	0.211	(0.004)		
Fe	(ppm)				
Ni	(ppm)	1.14	(0.039)		
Zn	(ppb)	123.0	(9.124)	Au	(ppb)
Co	(ppb)	3.46	(0.292)	Sb	(ppb)
I	(ppb)	174.2	(23.0)	Cr	(ppb)
Mn	(ppb)	5.40	(0.41)	Rb	(ppb)
Se	(ppb)	4.06	(2.02)	Sc	(ppb)
Hg	(ppb)			Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	50.2	(1.40)		
As	(ppb)	8.521	(0.374)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 54

Field (pool): Crossfield (Rundle C)

Stratigraphic unit: Carboniferous, Rundle Gp., Turner Valley Fm.

Well name: Triad et al Cross 8-11-27-2

Location: Lsd 8 Sec 11 Twp 27 Rge 2 W 5 Mer

Source of sample: Separator Pressure: 30 psig Temperature: 63 °F

Method of production: Flowing Depth: 8577-8591 ft. (perforations)

Date sampled: 23rd June 1970

Remarks:

S	(%)	0.653	(0.0516)				
V	(ppm)	0.068	(0.0029)				
Cl	(ppm)	5.432	(0.2089)				
Na	(ppm)	0.128	(0.0027)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)	29.0	(7.9)	Au	(ppb)		
Co	(ppb)	1.016	(0.254)	Sb	(ppb)		
I	(ppb)	8.735	(3.169)	Cr	(ppb)		
Mn	(ppb)	46.65	(0.8495)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.137	(0.023)
Hg	(ppb)			Eu	(ppb)	0.616	(0.155)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	3.38	(0.394)				
As	(ppb)	7.852	(0.329)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 55

Field (pool): Turner Valley (Rundle)

Stratigraphic unit: Carboniferous, Rundle Gp., Turner Valley Fm.

Well name: Royalite 39

Location: Lsd 3 Sec 29 Twp 18 Rge 2 W 5 Mer

Source of sample: Wellhead Pressure: 100 psig Temperature: 40 °F

Method of production: Pump Depth: 7600-8039 ft. (open hole)

Date sampled: 12th June 1969

Remarks:

S	(%)	0.377	(0.0233)		
V	(ppm)	0.028	(0.0022)		
Cl	(ppm)	5.292	(0.214)		
Na	(ppm)	0.065	(0.001)		
Fe	(ppm)				
Ni	(ppm)				
Zn	(ppb)			Au	(ppb)
Co	(ppb)			Sb	(ppb)
I	(ppb)			Cr	(ppb)
Mn	(ppb)	100.7	(1.88)	Rb	(ppb)
Se	(ppb)			Sc	(ppb)
Hg	(ppb)			Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	2.26	(0.210)		
As	(ppb)	36.3	(0.426)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 56

Field (pool): Del Bonita (Rundle)

Stratigraphic unit: Carboniferous, Rundle Gp.

Well name: National Cordasun Milk River 3

Location: Lsd 9 Sec24 Twp 1 Rge22 W 4 Mer

Source of sample: Tubing Pressure: 10 psig Temperature: cold F

Method of production: Pump Depth: 5143-5196 ft. (perforations)

Date sampled: 25th March 1970

Remarks: Formation water in original container

S	(%)	0.72	(0.0528)				
V	(ppm)	0.336	(0.0080)				
Cl	(ppm)	4.252	(0.2630)				
Na	(ppm)	0.160	(0.0027)				
Fe	(ppm)						
Ni	(ppm)	0.136	(0.0265)				
Zn	(ppb)	51.0	(10.6)	Au	(ppb)		
Co	(ppb)	0.886	(0.278)	Sb	(ppb)	3.262	(0.785)
I	(ppb)			Cr	(ppb)	13.14	(3.34)
Mn	(ppb)	50.79	(0.8219)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.542	(0.1411)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	7.94	(0.635)				
As	(ppb)	1218.0	(7.20)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 57

Field (pool): Sylvan Lake (Pekisko B)

Stratigraphic unit: Carboniferous, Rundle Gp., Pekisko Fm.

Well name: HB Sylvan Lake 4-16-38-3

Location: Lsd 4 Sec 16 Twp 38 Rge 3 W 5 Mer

Source of sample: Wellhead Pressure: 380 psig Temperature: 50 °F

Method of production: Flowing Depth: 7348-7354 ft. (perforations)

Date sampled: 24th June 1969

Remarks:

S	(%)	1.495	(0.127)				
V	(ppm)	17.50	(0.140)				
Cl	(ppm)	15.01	(1.11)				
Na	(ppm)	9.0	(0.180)				
Fe	(ppm)						
Ni	(ppm)	5.64	(0.108)				
Zn	(ppb)	1162.0	(25.8)	Au	(ppb)		
Co	(ppb)	4.484	(0.376)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	5.60	(0.455)	Rb	(ppb)	57.8	(17.3)
Se	(ppb)	67.4	(4.601)	Sc	(ppb)	0.203	(0.0248)
Hg	(ppb)	18.77	(2.76)	Eu	(ppb)	0.335	(0.159)
Cs	(ppb)	1.79	(0.5683)	Ca	(ppb)		
Br	(ppb)	7529.0	(110.8)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 58

Field (pool): Twining North (Rundle)

Stratigraphic unit: Carboniferous, Rundle Gp., Pekisko Fm.

Well name: Mic Mac et al Twin N 6-6-33-24

Location: Lsd 6 Sec 6 Twp 33 Rge 24 W 4 Mer

Source of sample: Wellhead Pressure: - psig Temperature: cold °F

Method of production: Pump Depth: 5424-5426 ft. (perforations)

Date sampled: 6th Nov. 1970

Remarks: Silt at bottom of original container

S	(%)	1.292	(0.1051)				
V	(ppm)	16.50	(0.1731)				
Cl	(ppm)	13.74	(0.6657)				
Na	(ppm)	4.39	(0.0742)				
Fe	(ppm)						
Ni	(ppm)	7.561	(0.1234)				
Zn	(ppb)	873.0	(22.6)	Au	(ppb)		
Co	(ppb)	8.683	(0.5311)	Sb	(ppb)		
I	(ppb)	100.9	(24.22)	Cr	(ppb)	19.08	(5.16)
Mn	(ppb)			Rb	(ppb)		
Se	(ppb)	57.5	(4.363)	Sc	(ppb)	0.231	(0.0255)
Hg	(ppb)	6.69	(1.2)	Eu	(ppb)	0.417	(0.182)
Cs	(ppb)	2.20	(0.610)	Ga	(ppb)		
Br	(ppb)	53.04	(2.73)				
As	(ppb)	5.56	(1.04)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 59

Field (pool): Glenevis (Banff)

Stratigraphic unit: Carboniferous, Banff Fm.

Well name: Texaco Glenevis 5-35-55-4

Location: lsd 5 Sec 35 Twp 55 Rge 4 W 5 Mer

Source of sample: test line from header Pressure: - psig Temperature: - °F

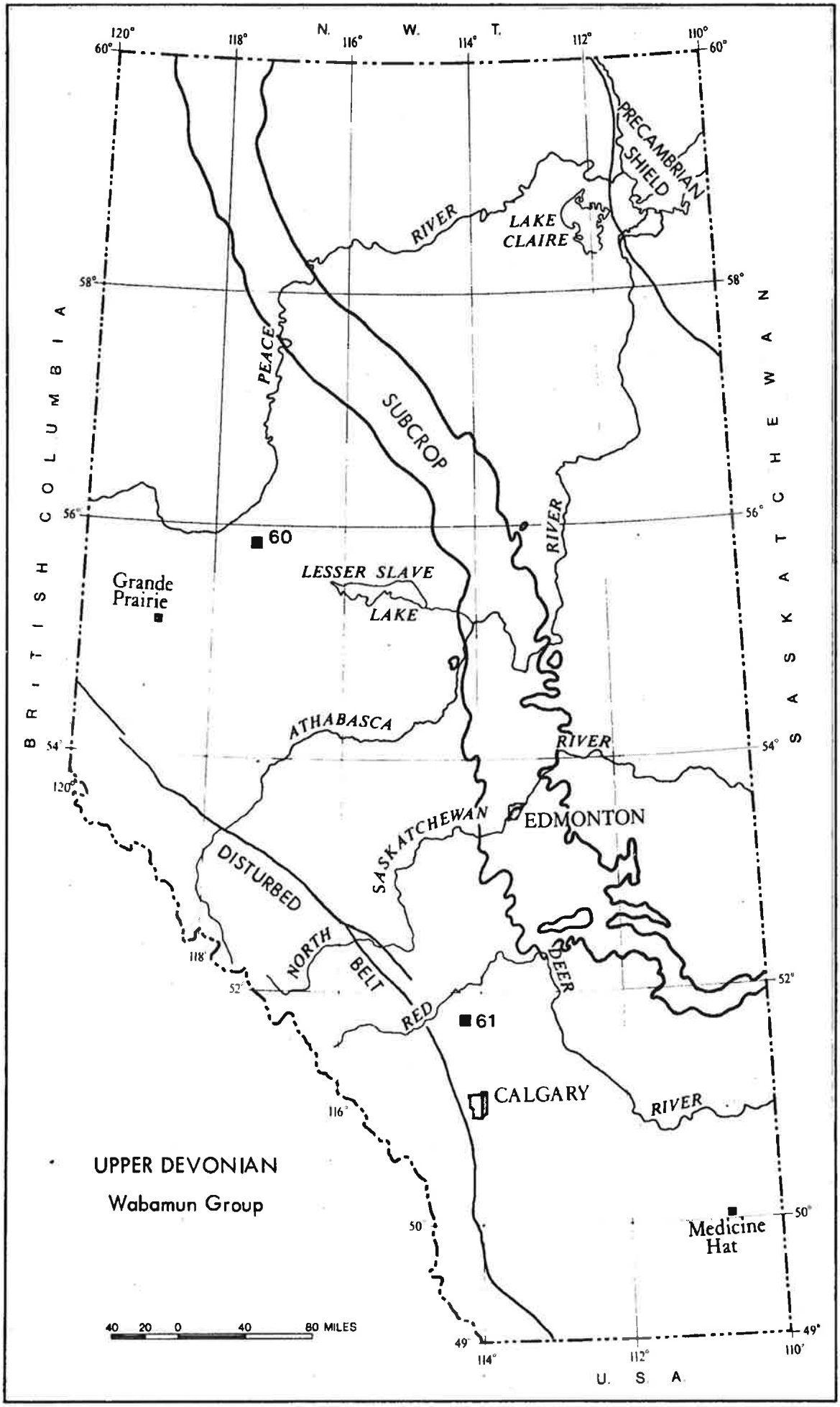
Method of production: Flowing Depth: 4354-4359 ft. (perforations)

Date sampled: 14th July 1969

Remarks: Formation water and emulsion at bottom of original container

S	(%)	3.355	(0.8108)				
V	(ppm)	138.8	(1.596)				
Cl	(ppm)	230.3	(3.422)				
Na	(ppm)						
Fe	(ppm)	2.19	(0.202)				
Ni	(ppm)	57.48	(0.870)				
Zn	(ppb)	563.4	(18.84)	Au	(ppb)		
Co	(ppb)	60.22	(1.05)	Sb	(ppb)	22.8	(1.29)
I	(ppb)			Cr	(ppb)	78.7	(7.23)
Mn	(ppb)			Rb	(ppb)	28.1	(8.82)
Se	(ppb)	252.3	(14.0)	Sc	(ppb)	0.128	(0.024)
Hg	(ppb)	7.74	(4.32)	Eu	(ppb)	0.493	(0.120)
Cs	(ppb)	9.03	(1.07)	Ga	(ppb)		
Br	(ppb)						
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis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SASKATCHEWAN
EDMONTON
DISTURBED
NORTH
BELT
DEER
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CALGARY
RIVER
Medicine Hat
BRITISH COLUMBIA
SASKATCHEWAN
U. S. A.
UPPER DEVONIAN
Wabamun Group
40 20 0 40 80 MILES

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 60

Field (pool): Normandville (D-1A)

Stratigraphic unit: U. Devonian, Wabamun Gp.

Well name: Col OK and Assoc Normand 8-16

Location: Lsd 8 Sec 16 Twp 79 Rge 22 W 5 Mer

Source of sample: Tubing Pressure: 20 psig Temperature: 55 °F

Method of production: Pump Depth: 5762-5802 ft (perforations)

Date sampled: 22nd July 1970

Remarks: Well shut in at time of sampling; water at bottom of original container

S	(%)	0.664	(0.0642)				
V	(ppm)	6.828	(0.0703)				
Cl	(ppm)	43.07	(3.001)				
Na	(ppm)	2.589	(0.0416)				
Fe	(ppm)						
Ni	(ppm)	10.94	(0.345)				
Zn	(ppb)	2433.0	(67.6)	Au	(ppb)		
Co	(ppb)	44.6	(3.35)	Sb	(ppb)		
I	(ppb)	398.7	(11.31)	Cr	(ppb)	15.07	(7.31)
Mn	(ppb)	36.22	(1.176)	Rb	(ppb)		
Se	(ppb)	16.33	(4.21)	Sc	(ppb)	0.178	(0.061)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	577.1	(8.18)				
As	(ppb)	23.67	(1.19)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 61

Field (pool): Olds (Wabamun A)

Stratigraphic unit: U. Devonian, Wabamun Gp.

Well name: Shell Cdn-Sup Olds 10-28W-32-2; Shell Cdn-Sup Olds 10-22W-32-2
Triad Shell Olds 10-8-33-2

Location: Lsd 10;10;Sec 28;22Twp 32;32Rge 2 W 5 Mer
10 8 33

Source of sample: Separator-meter run Pressure: 175 psig Temperature: 82 °F

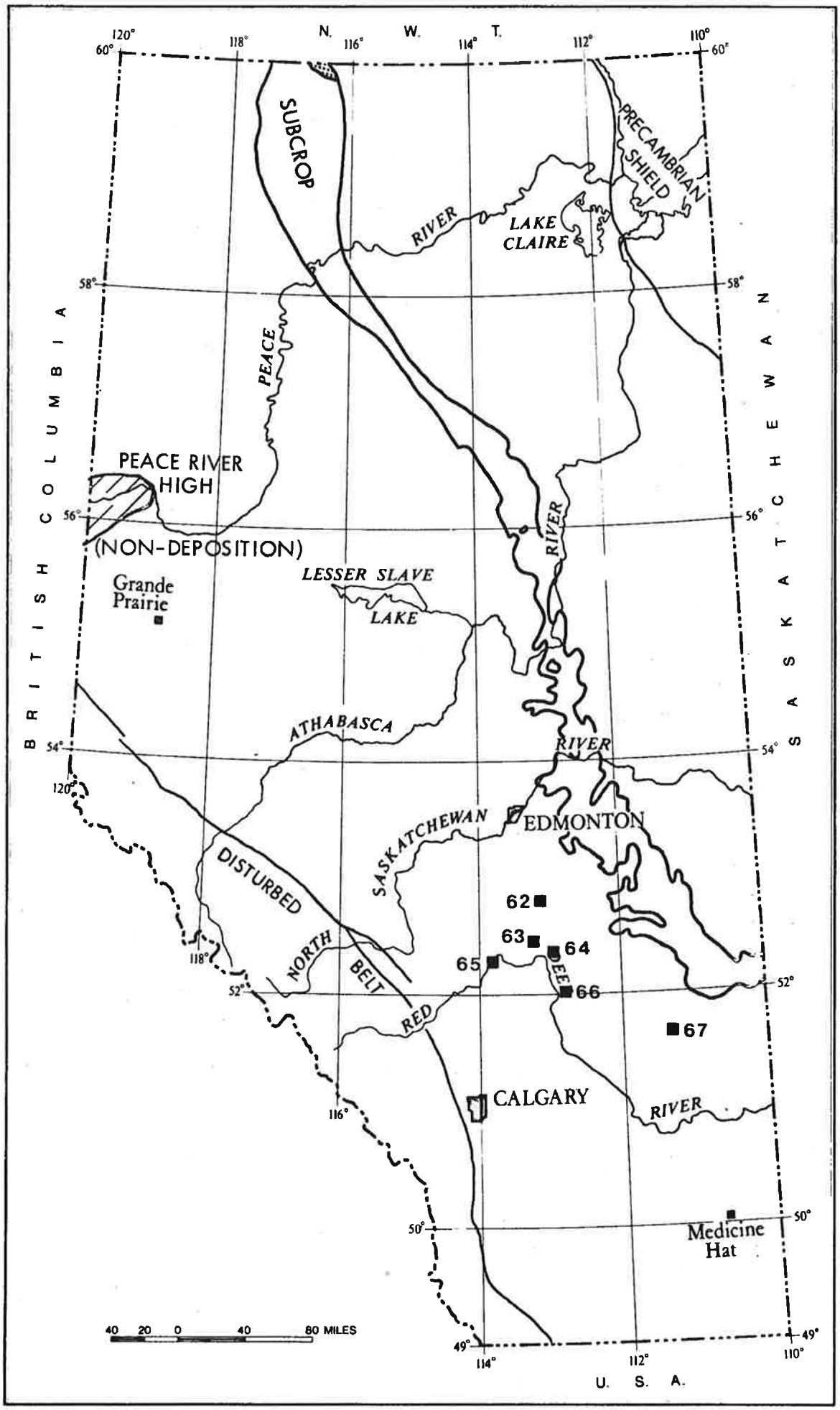
Method of production: Flowing Depth: 8953-8984; ft. (perforations)
8920-8952;
9028-9051

Date sampled: 8th April 1970

Remarks:

S	(%)	1.037	(0.0611)				
V	(ppm)	0.015	(0.0015)				
Cl	(ppm)	4.891	(0.1627)				
Na	(ppm)	0.156	(0.0019)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	0.516	(0.189)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	53.69	(0.9596)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.014	(0.0037)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	0.657	(0.294)	Ga	(ppb)		
Br	(ppb)	20.2	(0.585)				
As	(ppb)	10.28	(0.278)				

* Relative standard deviation (counting statistics) in parenthesis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PRECAMBRIAN SHIELD
RIVER
LAKE CLAIRE
PEACE
PEACE RIVER HIGH
(NON-DEPOSITION)
Grande Prairie
LESSER SLAVE LAKE
ATHABASCA RIVER
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH BELT
RED DEER
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63
64
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66
67
CALGARY
Medicine Hat
BRITISH COLUMBIA
SASKATCHEWAN
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U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 62
Field (pool): Malmo (D-2A)
Stratigraphic unit: U. Devonian, Winterburn Gp., Nisku Fm.
Well name: Scurry Malmo 5
Location: Lsd 9 Sec 15 Twp 44 Rge 22 W 4 Mer
Source of sample: Tubing Pressure: 20 psig Temperature: 35 °F
Method of production: Pump Depth: 5102-5121 ft. (perforations)
Date sampled: 22nd May 1970
Remarks:

S	(%)	0.783	(0.0691)		
V	(ppm)	1.09	(0.0245)		
Cl	(ppm)	15.0	(0.497)		
Na	(ppm)	0.80	(0.0063)		
Fe	(ppm)				
Ni	(ppm)	1.110	(0.0368)		
Zn	(ppb)	40.2	(6.33)	Au	(ppb)
Co	(ppb)	2.08	(0.230)	Sb	(ppb)
I	(ppb)			Cr	(ppb)
Mn	(ppb)	62.36	(0.874)	Rb	(ppb)
Se	(ppb)			Sc	(ppb)
Hg	(ppb)	5.920	(1.37)	Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	60.90	(1.473)		
As	(ppb)	21.71	(0.556)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 63

Field (pool): Alix (D-2)

Stratigraphic unit: U. Devonian, Winterburn Gp., Nisku Fm.

Well name: Ambass BA Alix 7-7-40-23

Location: Lsd 7 Sec 7 Twp 40 Rge 23 W 4 Mer

Source of sample: Wellhead Pressure: 390 psig Temperature: 45 °F

Method of production: Pump Depth: 5974-5981 ft. (perforations)

Date sampled: 4th Feb. 1970

Remarks:

S	(%)						
V	(ppm)						
Cl	(ppm)	693.2	(9.364)				
Na	(ppm)	57.76	(0.913)				
Fe	(ppm)	19.77	(0.421)				
Ni	(ppm)	0.531	(0.0474)				
Zn	(ppb)	5921.0	(77.5)	Au	(ppb)		
Co	(ppb)	45.04	(0.688)	Sb	(ppb)	34.8	(1.156)
I	(ppb)			Cr	(ppb)	205.8	(4.12)
Mn	(ppb)			Rb	(ppb)	60.0	(9.31)
Se	(ppb)			Sc	(ppb)	0.115	(0.0243)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	12.22	(1.633)	Ga	(ppb)		
Br	(ppb)	4.27	(0.165)				
As	(ppb)	1.081	(0.0542)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 64

Field (pool): Erskine (D-2)

Stratigraphic unit: U. Devonian, Winterburn Gp., Nisku Fm.

Well name: BA CPR Thom Erskine 8-13-39-21

Location: Lsd 8 Sec 13 Twp 39 Rge 21 W 4 Mer

Source of sample: Separator Pressure: 90 psig Temperature: 46 °F

Method of production: Pump Depth: 5172-5192 ft. (perforations)

Date sampled: 25th March, 1970

Remarks:

S	(%)						
V	(ppm)	46.03	(0.4539)				
Cl	(ppm)	1014.0	(19.67)				
Na	(ppm)	64.70	(1.460)				
Fe	(ppm)	1.00	(0.48)				
Ni	(ppm)						
Zn	(ppb)	515.6	(30.0)	Au	(ppb)		
Co	(ppb)	202.6	(12.3)	Sb	(ppb)		
I	(ppb)	7335.0	(285.4)	Cr	(ppb)	41.8	(8.25)
Mn	(ppb)	176.6	(11.30)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)	200.0	(11.2)	Eu	(ppb)	0.390	(0.100)
Cs	(ppb)	3.0	(0.83)	Ga	(ppb)		
Br	(ppb)	12470.0	(158.4)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 65

Field (pool): Joffre (D-2)

Stratigraphic unit: U. Devonian, Winterburn Gp., Nisku Fm.

Well name: Imp Joffre 4-36N-38-27

Location: Lsd 4 Sec 36 Twp 38 Rge 27 W 4 Mer

Source of sample: Separator Pressure: 74 psig Temperature: 40 °F

Method of production: Flowing Depth: 7077-7184 ft. (open hole)

Date sampled: 25th March 1970

Remarks:

S	(%)	0.354	(0.0216)				
V	(ppm)	0.030	(0.0019)				
Cl	(ppm)	13.34	(0.3865)				
Na	(ppm)	0.091	(0.0011)				
Fe	(ppm)	0.390	(0.123)				
Ni	(ppm)						
Zn	(ppb)	374.0	(15.4)	Au	(ppb)		
Co	(ppb)	4.312	(0.261)	Sb	(ppb)		
I	(ppb)	727.7	(14.75)	Cr	(ppb)	12.1	(3.0)
Mn	(ppb)	30.58	(3.382)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.067	(0.015)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)	2.17	(0.580)	Ga	(ppb)		
Br	(ppb)	16.8	(0.579)				
As	(ppb)	5.34	(0.236)				

* Relative standard deviation (counting statistics) in parentheses

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 66

Field (pool): Fenn-Big Valley (D-2A)

Stratigraphic unit: U. Devonian, Winterburn Gp., Nisku Fm.

Well name: CPR Connor 7

Location: Lsd 7 Sec 23 Twp 35 Rge 20 W 4 Mer

Source of sample: Wellhead - tubing Pressure: 260 psig Temperature: 30 °F

Method of production: Flowing Depth: 5322-5362 ft. (open hole)

Date sampled: 5th Feb. 1970

Remarks:

S	(%)	1.227	(0.0853)				
V	(ppm)	11.80	(0.1)				
Cl	(ppm)	55.9	(1.12)				
Na	(ppm)	0.632	(0.054)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)			Au	(ppb)		
Co	(ppb)			Sb	(ppb)		
I	(ppb)	424.7	(23.0)	Cr	(ppb)		
Mn	(ppb)	42.4	(1.0)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	165.0	(1.873)
Br	(ppb)	1541.0	(32.6)				
As	(ppb)	392.4	(5.19)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 67

Field (pool): Youngstown (Arcs)

Stratigraphic unit: U. Devonian, Peechee Fm., Ares Mbr.

Well name: Humber Charter Reist 14-25-31-10

Location: Lsd 14 Sec 25 Twp 31 Rge 10 W 4 Mer

Source of sample: Tubing Pressure: - psig Temperature: - °F

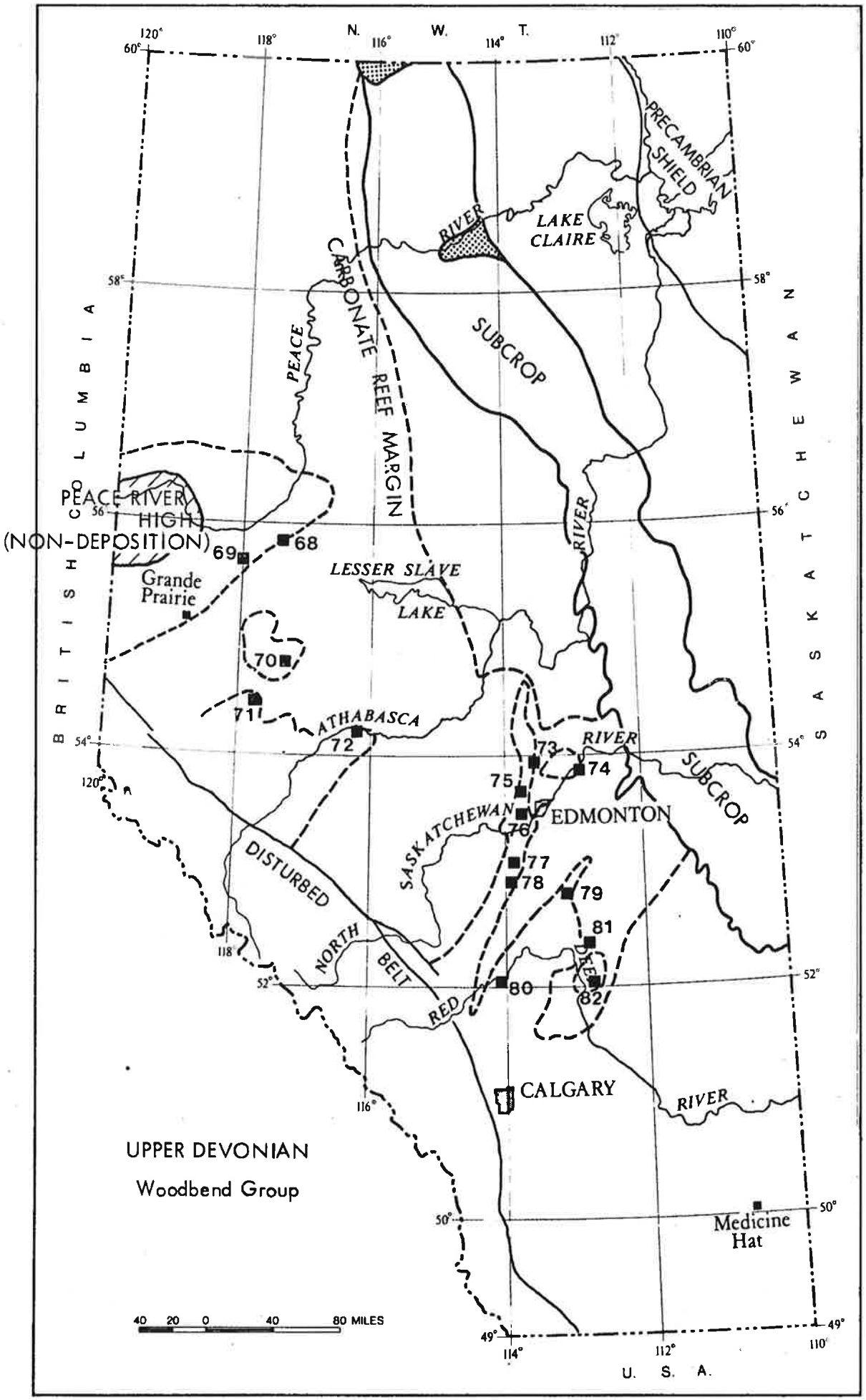
Method of production: Pump Depth: 3708-3720 ft. (open hole)

Date sampled: 23rd June 1970

Remarks: Vevsene squeeze job conducted satisfactorily in June, 1978, to increase oil production

S	(%)	1.318	(0.1056)				
V	(ppm)	41.27	(0.3747)				
Cl	(ppm)	6.580	(0.2451)				
Na	(ppm)	0.102	(0.0016)				
Fe	(ppm)						
Ni	(ppm)	18.03	(0.240)				
Zn	(ppb)	85.0	(3.8)	Au	(ppb)		
Co	(ppb)	15.1	(0.312)	Sb	(ppb)	0.462	(0.160)
I	(ppb)			Cr	(ppb)	29.63	(2.83)
Mn	(ppb)	60.45	(1.0510)	Rb	(ppb)		
Se	(ppb)	73.6	(0.270)	Sc	(ppb)	0.10	(0.0074)
Hg	(ppb)	6.89	(0.90)	Eu	(ppb)	0.158	(0.049)
Cs	(ppb)	0.641	(0.262)	Ga	(ppb)	21.2	(0.33)
Br	(ppb)	19.2	(0.700)				
As	(ppb)	25.8	(0.480)				

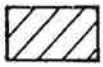
* Relative standard deviation (counting statistics) in parenthesis


N. W. T.
PRECAMBRIAN SHIELD
LAKE CLAIRE
RIVER
CARBONATE REEF MARGIN
SUBCROP
PEACE
PEACE RIVER HIGH
(NON-DEPOSITION)
BRITISH COLUMBIA
SASKATCHEWAN
RIVER
LESSER SLAVE LAKE
Grande Prairie
ATHABASCA
RIVER
SUBCROP
SASKATCHEWAN
EDMONTON
DISTURBED
NORTH BELT
RED DEER
RIVER
CALGARY
Medicine Hat
68
69
70
71
72
73
74
75
76
77
78
79
80
81
82
UPPER DEVONIAN
Woodbend Group
40 20 0 40 80 MILES
U. S. A.

LEGEND

Outcrop

Subcrop



Peace River High (Non-Deposition)

Boundary of Near-Shore Clastics .....

Carbonate Reef Margin .....

Sample Number ..... ■ 28

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 68

Field (pool): Normandville (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Col OK and Assoc Normand 5-15

Location: Lsd 5 Sec 15 Twp 79 Rge 22 W 5 Mer

Source of sample: Tubing Pressure: 400 psig Temperature: 50 °F

Method of production: Flowing Depth: 6673-6690 ft. (open hole)

Date sampled: 22nd July 1970

Remarks:

S	(%)	0.201	(0.029)				
V	(ppm)	1.542	(0.019)				
Cl	(ppm)	39.17	(0.721)				
Na	(ppm)	4.912	(0.0578)				
Fe	(ppm)						
Ni	(ppm)	2.86	(0.051)				
Zn	(ppb)	132.0	(5.5)	Au	(ppb)		
Co	(ppb)	36.5	(0.541)	Sb	(ppb)		
I	(ppb)	2467.0	(35.63)	Cr	(ppb)		
Mn	(ppb)	43.28	(0.983)	Ro	(ppb)		
Se	(ppb)	8.25	(0.062)	Sc	(ppb)	0.094	(0.0093)
Hg	(ppb)			Eu	(ppb)	0.247	(0.06)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	847.0	(21.2)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 69

Field (pool): Eaglesham (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: HB Union Eaglesham 10-14-77-25

Location: Lsd 10 Sec 14 Twp 77 Rge 25 W 5 Mer

Source of sample: Tubing Pressure: 400 psig Temperature: 60 °F

Method of production: Flowing Depth: 7552-7564; 7570-7575 ft. (perforations)

Date sampled: 22nd July 1970

Remarks:

S	(%)	0.216	(0.0466)				
V	(ppm)	1.174	(0.0232)				
Cl	(ppm)	6.093	(0.2158)				
Na	(ppm)	0.112	(0.0018)				
Fe	(ppm)						
Ni	(ppm)	1.510	(0.060)				
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	7.511	(0.584)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	49.33	(0.9963)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.044	(0.017)
Hg	(ppb)			Eu	(ppb)	0.424	(0.129)
Cs	(ppb)	1.751	(0.376)	Ga	(ppb)	1.694	(0.1121)
Br	(ppb)	39.40	(1.063)				
As	(ppb)	3.98	(0.270)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 71

Field (pool): Simonette (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Shell Simonette 12-6-63-25

Location: Lsd 12 Sec 6 Twp 63 Rge 25 W 5 Mer

Source of sample: Wellhead Pressure: 1000 psig Temperature: 58 °F

Method of production: Flowing Depth: 11,536-11,558 ft. (open hole)

Date sampled: 27th Aug. 1970

Remarks:

S	(%)	0.340	(0.0356)				
V	(ppm)	0.017	(0.0016)				
Cl	(ppm)	5.43	(0.1496)				
Na	(ppm)	0.183	(0.003)				
Fe	(ppm)	0.216	(0.052)				
Ni	(ppm)						
Zn	(ppb)	16.0	(2.8)	Au	(ppb)		
Co	(ppb)	4.09	(0.174)	Sb	(ppb)		
I	(ppb)	10.46	(2.9030)	Cr	(ppb)	13.6	(1.31)
Mn	(ppb)	47.70	(0.9142)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.094	(0.0077)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	15.33	(0.572)				
As	(ppb)	1.21	(0.1424)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 72

Field (pool): Windfall (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Fina Pan Am HB Wind 10-7-60-15

Location: Lsd 10 Sec 7 Twp 60 Rge 15 W 5 Mer

Source of sample: Separator Pressure: 1140 psig Temperature: 136 °F

Method of production: Flowing Depth: 8559-8580 ft. (perforations)

Date sampled: 18th Dec. 1970

Remarks:

S	(%)	0.593	(0.0480)				
V	(ppm)	0.014	(0.0017)				
Cl	(ppm)	6.267	(0.2060)				
Na	(ppm)	0.023	(0.0050)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	5.281	(0.247)	Sb	(ppb)		
I	(ppb)	51.98	(5.589)	Cr	(ppb)		
Mn	(ppb)	52.83	(1.003)	Rb	(ppb)	15.58	(3.20)
Se	(ppb)			Sc	(ppb)	0.081	(0.010)
Hg	(ppb)	3.0	(0.785)	Eu	(ppb)	0.255	(0.066)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	39.4	(1.107)				
As	(ppb)	7.844	(0.204)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 73

Field (pool): Fairydell-Bon Accord (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Imperial Fairydell 16-8L-57-24

Location: Lsd 16 Sec 8 Twp 57 Rge 24 W 4 Mer

Source of sample: Test treater Pressure: - psig Temperature: 140° F

Method of production: - Depth: not specified ft. Leduc Fm. 4005; T. D. 4038 . End casing 4020 May be open hole production

Date sampled: 4th Aug. 1970

Remarks:

S	(%)	1.244	(0.0631)				
V	(ppm)	6.553	(0.0579)				
Cl	(ppm)	38.74	(0.9333)				
Na	(ppm)	1.975	(0.0343)				
Fe	(ppm)	0.656	(0.0833)				
Ni	(ppm)	11.32	(0.177)				
Zn	(ppb)	329.3	(9.43)	Au	(ppb)	0.149	(0.0621)
Co	(ppb)	33.66	(0.527)	Sb	(ppb)	1.90	(0.272)
I	(ppb)	125.9	(9.151)	Cr	(ppb)	28.76	(3.20)
Mn	(ppb)	36.45	(1.465)	Rb	(ppb)		
Se	(ppb)	63.43	(4.70)	Sc	(ppb)	0.026	(0.010)
Hg	(ppb)	23.0	(4.45)	Eu	(ppb)	0.180	(0.062)
Cs	(ppb)			Ga	(ppb)	127.5	(1.565)
Br	(ppb)	322.5	(11.66)				
As	(ppb)	1219.0	(21.22)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 74

Field (pool): Redwater (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Imperial Simmons 70

Location: Lsd 14 Sec 19 Twp 56 Rge 20 W 4 Mer

Source of sample: Separator Pressure: 375 psig Temperature: 60 °F

Method of production: - Depth: 3086-3146 ft. (open hole)

Date sampled: 9th July 1969

Remarks:

S	(%)	0.474	(0.0516)				
V	(ppm)	6.365	(0.0862)				
Cl	(ppm)	28.61	(0.740)				
Na	(ppm)	0.053	(0.017)				
Fe	(ppm)	0.226	(0.095)				
Ni	(ppm)	14.6	(0.230)				
Zn	(ppb)	151.4	(7.82)	Au	(ppb)	0.204	(0.0243)
Co	(ppb)	60.3	(0.79)	Sb	(ppb)		
I	(ppb)	502.8	(15.06)	Cr	(ppb)	16.63	(2.92)
Mn	(ppb)	39.16	(1.479)	Rb	(ppb)		
Se	(ppb)	14.54	(2.75)	Sc	(ppb)		
Hg	(ppb)	3.13	(1.67)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	23.9	(0.422)
Br	(ppb)	460.0	16.22				
As	(ppb)	22.4	0.751				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 75

Field (pool): Morinville (D-3B)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Oakland Morinv 8-14-54-26

Location: Lsd 8 Sec 14 Twp 54 Rge 26 W 4 Mer

Source of sample: Separator Pressure: 20 psig Temperature: 20 °F

Method of production: Flowing Depth: 5206-5216 ft. (perforations)

Date sampled: 5th March 1970

Remarks:

S	(%)	0.237	(0.030)				
V	(ppm)	0.903	(0.0127)				
Cl	(ppm)	19.73	(0.65)				
Na	(ppm)	0.187	(0.0019)				
Fe	(ppm)						
Ni	(ppm)	6.74	(0.12)				
Zn	(ppb)	77.0	(8.4)	Au	(ppb)		
Co	(ppb)	15.4	(0.517)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)	11.32	(2.85)
Mn	(ppb)	49.2	(1.012)	Rb	(ppb)		
Se	(ppb)	14.23	(3.84)	Sc	(ppb)	0.096	(0.014)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	1.30	(0.123)
Br	(ppb)	38.8	(1.01)				
As	(ppb)	13.23	(0.343)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 76

Field (pool): Acheson (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Cal Std Welbourn 8-34

Location: Lsd 8 Sec 34 Twp 52 Rge 26 W 4 Mer

Source of sample: Header Pressure: - psig Temperature: - °F

Method of production: Flowing Depth: Not specified. ft. Leduc Fm. 5038; TD 5095; End casing 5016

Date sampled: 10 Feb. 1970 Production may be from open hole

Remarks:

S	(%)	0.3082	(0.037)				
V	(ppm)	0.981	(0.0118)				
Cl	(ppm)	12.86	(0.373)				
Na	(ppm)	0.336	(0.0041)				
Fe	(ppm)	0.312	(0.0754)				
Ni	(ppm)	4.16	(0.071)				
Zn	(ppb)	221.0	(7.2)	Au	(ppb)		
Co	(ppb)	14.9	(0.532)	Sb	(ppb)		
I	(ppb)	71.010	(6.02)	Cr	(ppb)		
Mn	(ppb)	55.4	(1.15)	Rb	(ppb)		
Se	(ppb)	16.0	(0.099)	Sc	(ppb)	0.10	(0.01)
Hg	(ppb)	1.34	(0.55)	Eu	(ppb)		
Cs	(ppb)	0.870	(0.268)	Ga	(ppb)	0.52	(0.14)
Br	(ppb)	137.3	(3.73)				
As	(ppb)	3.87	(0.273)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 70

Field (pool): Little Smoky (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Little Smoky 10-27

Location: Lsd 10 Sec 27 Twp 67 Rge 22 W 5 Mer

Source of sample: Wellhead- tubing Pressure: 200 psig Temperature: 50 °F

Method of production: Flowing Depth: 8707-8712 ft. (perforations)

Date sampled: 21st July 1970

Remarks:

S	(%)	0.463	(0.0378)				
V	(ppm)	0.099	(0.0034)				
Cl	(ppm)	12.10	(0.2801)				
Na	(ppm)	0.313	(0.040)				
Fe	(ppm)						
Ni	(ppm)	0.120	(0.0155)				
Zn	(ppb)	60.0	(4.9)	Au	(ppb)		
Co	(ppb)	4.93	(0.256)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	48.69	(1.044)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.115	(0.0107)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ce	(ppb)		
Br	(ppb)	31.64	(1.10)				
As	(ppb)	2.57	(0.245)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 77

Field (pool): Bonnie Glen (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Chevron Texaco B Glen 10-7-47-27

Location: Lsd 10 Sec 7 Twp 47 Rge 27 W 4 Mer

Source of sample: Flowline Pressure: 590 psig Temperature: - °F

Method of production: flowing Depth: 7060-7080 ft. (perforations)

Date sampled: 27th Jan. 1970

Remarks:

S	(%)	0.309	(0.0193)				
V	(ppm)	0.071	(0.0029)				
Cl	(ppm)	70.62	(0.5705)				
Na	(ppm)	0.0174	(0.0029)				
Fe	(ppm)						
Ni	(ppm)	0.10	(0.014)				
Zn	(ppb)	37.0	(3.5)	Au	(ppb)		
Co	(ppb)	8.25	(0.259)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	39.35	(0.0646)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.087	(0.0085)
Hg	(ppb)	291.1	(21.2)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	0.07	(0.02)
Br	(ppb)	21.6	(0.644)				
As	(ppb)	0.63	0.0511				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 78

Field (pool): Westerose (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: CPR Christensen 16

Location: Lsd 16 Sec 33 Twp 45 Rge 28 W 4 Mer

Source of sample: - Pressure: - psig Temperature: - °F

Method of production: Flowing Depth: Not given. ft. Leduc 6860; TD 7372 ft

Date sampled: 16th April 1970

Remarks:

S	(%)	0.311	(0.0250)				
V	(ppm)	0.178	(0.0047)				
Cl	(ppm)	6.100	(0.3681)				
Na	(ppm)	0.168	(0.0032)				
Fe	(ppm)	0.429	(0.118)				
Ni	(ppm)						
Zn	(ppb)	13.40	(3.00)	Au	(ppb)		
Co	(ppb)	1.57	(0.254)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	53.08	(0.8579)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.085	(0.014)
Hg	(ppb)	6.57	(1.34)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	0.551	(0.143)
Br	(ppb)	19.60	(1.25)				
As	(ppb)	3.843	(0.413)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 79

Field (pool): Malmo (D-3A)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Fina Pacific Malmo 5-11L-44-22

Location: Lsd 5 Sec 11 Twp 44 Rge 22 W 4 Mer

Source of sample: Treater Pressure: 10 psig Temperature: 110°F

Method of production: Pump Depth: 5233-5250 ft. (perforations)

Date sampled: 21st Aug. 1970

Remarks: Visco 1688 injected in ratio 1 pint to ~ 350 bbls fluid; treater temperature given as 150°F.

S	(%)	0.710	(0.0523)				
V	(ppm)	1.469	(0.0180)				
Cl	(ppm)	23.49	(0.4993)				
Na	(ppm)	0.130	(0.0024)				
Fe	(ppm)	4.43	(0.0723)				
Ni	(ppm)						
Zn	(ppb)	63.0	(5.10)	Au	(ppb)		
Co	(ppb)	25.75	(0.53)	Sb	(ppb)		
I	(ppb)	76.28	(4.0)	Cr	(ppb)		
Mn	(ppb)	50.15	(0.9319)	Rb	(ppb)		
Se	(ppb)	12.12	(0.123)	Sc	(ppb)	0.131	(0.0113)
Hg	(ppb)			Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	20.0	(0.434)
Br	(ppb)	317.8	(8.25)				
As	(ppb)	21.0	(0.577)				

* Relative standard deviation (counting statistics) in parentheses

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 80

Field (pool): Innisfail (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: HB WR C and E Innisfail 11-10-35-1

Location: Lsd 11 Sec 10 Twp 35 Rge 1 W 5 Mer

Source of sample: Wellhead-tubing Pressure: 660 psig Temperature: 68 °F

Method of production: Flowing Depth: 8489-8504 ft. (perforations)

Date sampled: 26th June 1970

Remarks:

S	(%)	0.443	(0.0551)				
V	(ppm)	0.017	(0.0023)				
Cl	(ppm)	3.854	(0.1591)				
Na	(ppm)	0.0434	(0.0062)				
Fe	(ppm)						
Ni	(ppm)						
Zn	(ppb)			Au	(ppb)		
Co	(ppb)	6.82	(0.30)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	58.67	(0.901)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.108	(0.0112)
Hg	(ppb)			Eu	(ppb)	0.20	(0.078)
Cs	(ppb)	0.90	(0.252)	Ga	(ppb)		
Br	(ppb)	8.02	(0.263)				
As	(ppb)	1.47	(0.071)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 81

Field (pool): Erskine (D-3)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: CPR Clark 6

Location: Lsd 6 Sec 7 Twp 39 Rge 20 W 4 Mer

Source of sample: Flow line Pressure: 21 psig Temperature: 30 °F

Method of production: Flowing Depth: 5379-5382 ft. (perforations)

Date sampled: 18th February 1970

Remarks:

S	(%)	2.144	(0.1096)				
V	(ppm)	5.699	(0.0542)				
Cl	(ppm)	27.17	(0.6127)				
Na	(ppm)	2.66	(0.039)				
Fe	(ppm)	1.581	(0.20)				
Ni	(ppm)	7.53	(0.240)				
Zn	(ppb)	114.3	(9.60)	Au	(ppb)		
Co	(ppb)	9.07	(0.804)	Sb	(ppb)	5.50	(0.793)
I	(ppb)	350.3	(12.04)	Cr	(ppb)	10.20	(0.578)
Mn	(ppb)	70.86	(1.441)	Rb	(ppb)		
Se	(ppb)	18.6	(5.35)	Sc	(ppb)	0.056	(0.026)
Hg	(ppb)			Eu	(ppb)	0.320	(0.130)
Cs	(ppb)	1.63	(0.564)	Ga	(ppb)		
Br	(ppb)	622.2	(14.20)				
As	(ppb)	40.0	(2.61)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 82

Field (pool): Fenn-Big Valley (D-3E)

Stratigraphic unit: U. Devonian, Woodbend Gp., Leduc Fm.

Well name: Imp HB Caprona 3-26L-36-20

Location: Lsd 3 Sec 26 Twp 36 Rge 20 W 4 Mer

Source of sample: Wellhead Pressure: 90 psig Temperature: 30°F

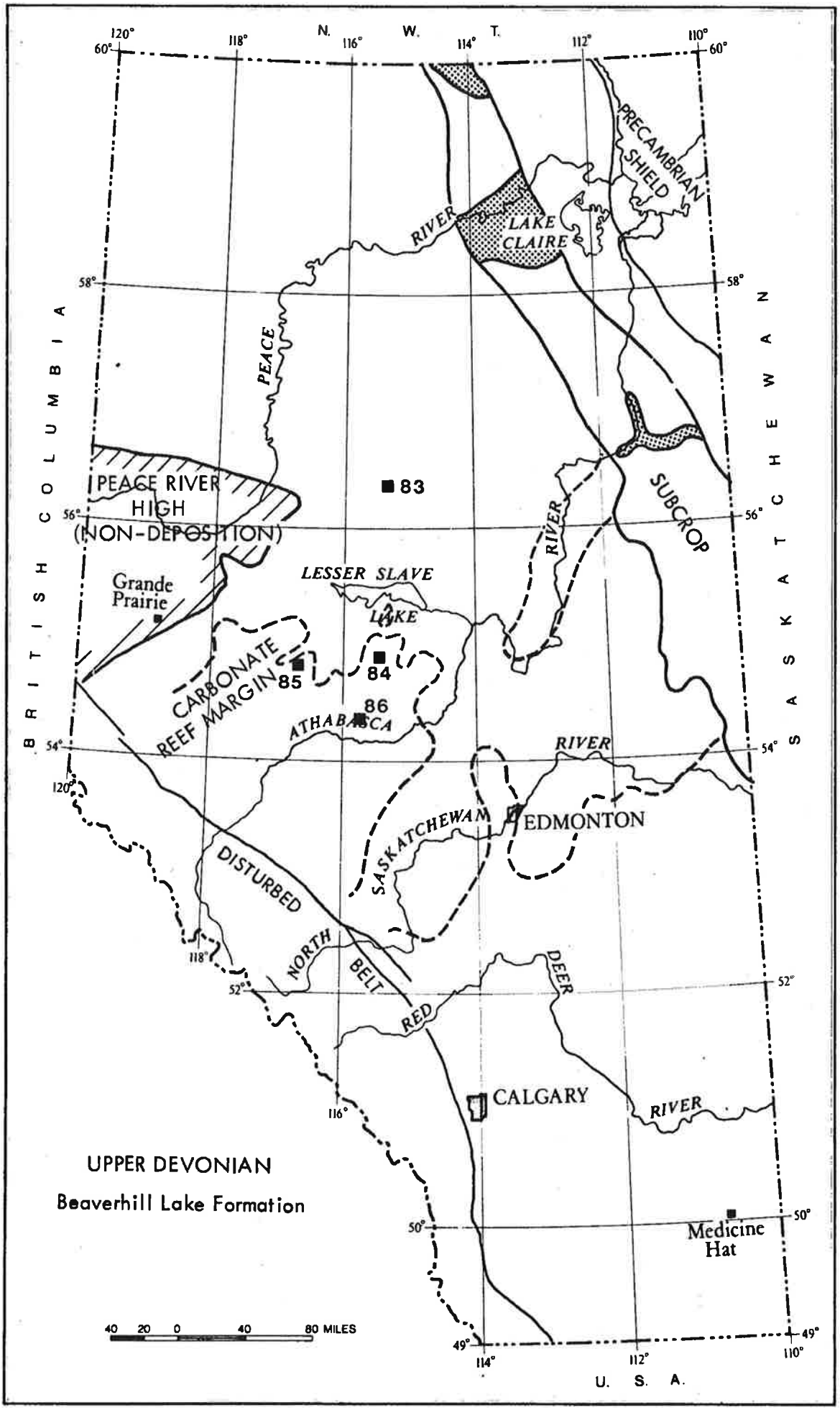
Method of production: Pump Depth: 5328-5329 ft. (perforations)

Date sampled: 5th Feb. 1970

Remarks: Very heavy oil emulsion which had to be thinned with benzene before centrifuging; analyzed sample contains less than 5% residual benzene.

S	(%)	2.991	(0.2492)				
V	(ppm)	52.67	(5926)				
Cl	(ppm)	250.0	(4.661)				
Na	(ppm)	0.354	(0.0058)				
Fe	(ppm)	0.557	(0.123)				
Ni	(ppm)	70.1	(1.01)				
Zn	(ppb)	940.1	(20.56)	Au	(ppb)		
Co	(ppb)	61.0	(0.752)	Sb	(ppb)	2.072	(0.233)
I	(ppb)	7396.0	(91.21)	Cr	(ppb)	83.5	(5.30)
Mn	(ppb)	91.92	(2.583)	Rb	(ppb)		
Se	(ppb)	56.1	(5.31)	Sc	(ppb)		
Hg	(ppb)	139.8	(12.73)	Eu	(ppb)	0.254	(0.082)
Cs	(ppb)	3.79	(0.591)	Ce	(ppb)	445.0	(11.9)
Br	(ppb)	8406.0	(289.0)				
As	(ppb)	672.9	(18.0)				

* Relative standard deviation (counting statistics) in parenthesis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PEACE
BRITISH COLUMBIA
SASKATCHEWAN
PEACE RIVER HIGH (NON-DEPOSITION)
83
SUBCROP
RIVER
Grande Prairie
LESSER SLAVE LAKE
CARBONATE REEF MARGIN
85
84
86
ATHABASCA
RIVER
SASKATCHEWAM
EDMONTON
DISTURBED
NORTH
BELT
RED
DEER
RIVER
CALGARY
Medicine Hat
UPPER DEVONIAN
Beaverhill Lake Formation
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 83

Field (pool): Loon (Slave Point A)

Stratigraphic unit: U. Devonian, Slave Point Fm.

Well name: Chevron Suptst Dome Loon 10-17-85-9

Location: Lsd 10 Sec 17 Twp 85 Rge 9 W 5 Mer

Source of sample: Wellhead Pressure: 50 psig Temperature: 55°F

Method of production: Pump Depth: 4567-4608 ft.(open hole)

Date sampled: 15th July 1970

Remarks:

S	(%)	0.2423	(0.0512)				
V	(ppm)	1.410	(0.023)				
Cl	(ppm)	10.2	(0.366)				
Na	(ppm)	0.644	(0.0054)				
Fe	(ppm)	0.574	(0.121)				
Ni	(ppm)	8.0	(0.13)				
Zn	(ppb)	1178.0	(32.0)	Au	(ppb)		
Co	(ppb)	26.3	(0.584)	Sb	(ppb)	4.61	(0.484)
I	(ppb)	36.7	(7.81)	Cr	(ppb)		
Mn	(ppb)	65.1	(1.183)	Rb	(ppb)		
Se	(ppb)	10.42	(2.54)	Sc	(ppb)	0.074	(0.014)
Hg	(ppb)	49.0	(6.0)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)	1.89	(0.244)
Br	(ppb)	69.6	(1.77).				
As	(ppb)	124.0	(1.65)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 84

Field (pool): Swan Hills (Beaverhill Lake A)

Stratigraphic unit: U. Devonian, Beaverhill Lake Fm.

Well name: Kewanee A-45 Swan H 12-32-68-10

Location: Lsd 12 Sec 32 Twp 68 Rge 10 W 5 Mer

Source of sample: Wellhead Pressure: - psig Temperature: - °F

Method of production: Pump Depth: 8469 -8489; ft. (perforations)
8500 -8512;
8535 -8545

Date sampled: 10th July 1969

Remarks: Emulsion and formation water at bottom of original sample container

S	(%)	0.156	(0.0259)				
V	(ppm)	0.037	(0.0027)				
Cl	(ppm)	3.789	(0.1849)				
Na	(ppm)	1.169	(0.0113)				
Fe	(ppm)	0.338	(0.118)				
Ni	(ppm)	0.494	(0.033)				
Zn	(ppb)	22.6	(3.49)	Au	(ppb)		
Co	(ppb)	1.21	(0.223)	Sb	(ppb)		
I	(ppb)	162.7	(8.8720)	Cr	(ppb)	6.05	(1.73)
Mn	(ppb)	43.94	(0.9052)	Rb	(ppb)		
Se	(ppb)	9.44	(1.96)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.335	(0.102)
Cs	(ppb)	1.71	(0.190)	Ga	(ppb)		
Br	(ppb)	31.66	(1.625)				
As	(ppb)	14.75	(0.664)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 85

Field (pool): Goose River (Beaverhill Lake A)

Stratigraphic unit: U. Devonian, Beaverhill Lake Fm.

Well name: BA Goose River 10-4-67-18

Location: Lsd 10 Sec 4 Twp 67 Rge 18 W 5 Mer

Source of sample: Wellhead - line just downstream of choke Pressure: - psig Temperature: - °F

Method of production: - Depth: 9278-9288 ft. (perforations)

Date sampled: 10th July 1969

Remarks:

S	(%)	0.126	(0.0297)				
V	(ppm)	0.014	(0.0021)				
Cl	(ppm)	4.664	(0.1642)				
Na	(ppm)	0.789	(0.010)				
Fe	(ppm)						
Ni	(ppm)	0.085	(0.010)				
Zn	(ppb)	148.0	(5.1)	Au	(ppb)		
Co	(ppb)	5.43	(0.20)	Sb	(ppb)		
I	(ppb)			Cr	(ppb)		
Mn	(ppb)	17.93	(0.6306)	Rb	(ppb)		
Se	(ppb)	4.0	(0.045)	Sc	(ppb)	0.091	(0.0072)
Hg	(ppb)			Eu	(ppb)	0.194	(0.050)
Cs	(ppb)	0.388	(0.156)	Ga	(ppb)		
Br	(ppb)	20.3	(0.887)				
As	(ppb)	1.45	(0.26)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 86

Field (pool): Carson Creek North (Beaverhill Lake A)

Stratigraphic unit: U. Devonian, Beaverhill Lake Fm.

Well name: Mobil PR Carson N 6-9MU-62-12

Location: Lsd 6 Sec 9 Twp 62 Rge 12 W 5 Mer

Source of sample: Wellhead Pressure: - psig Temperature: - °F

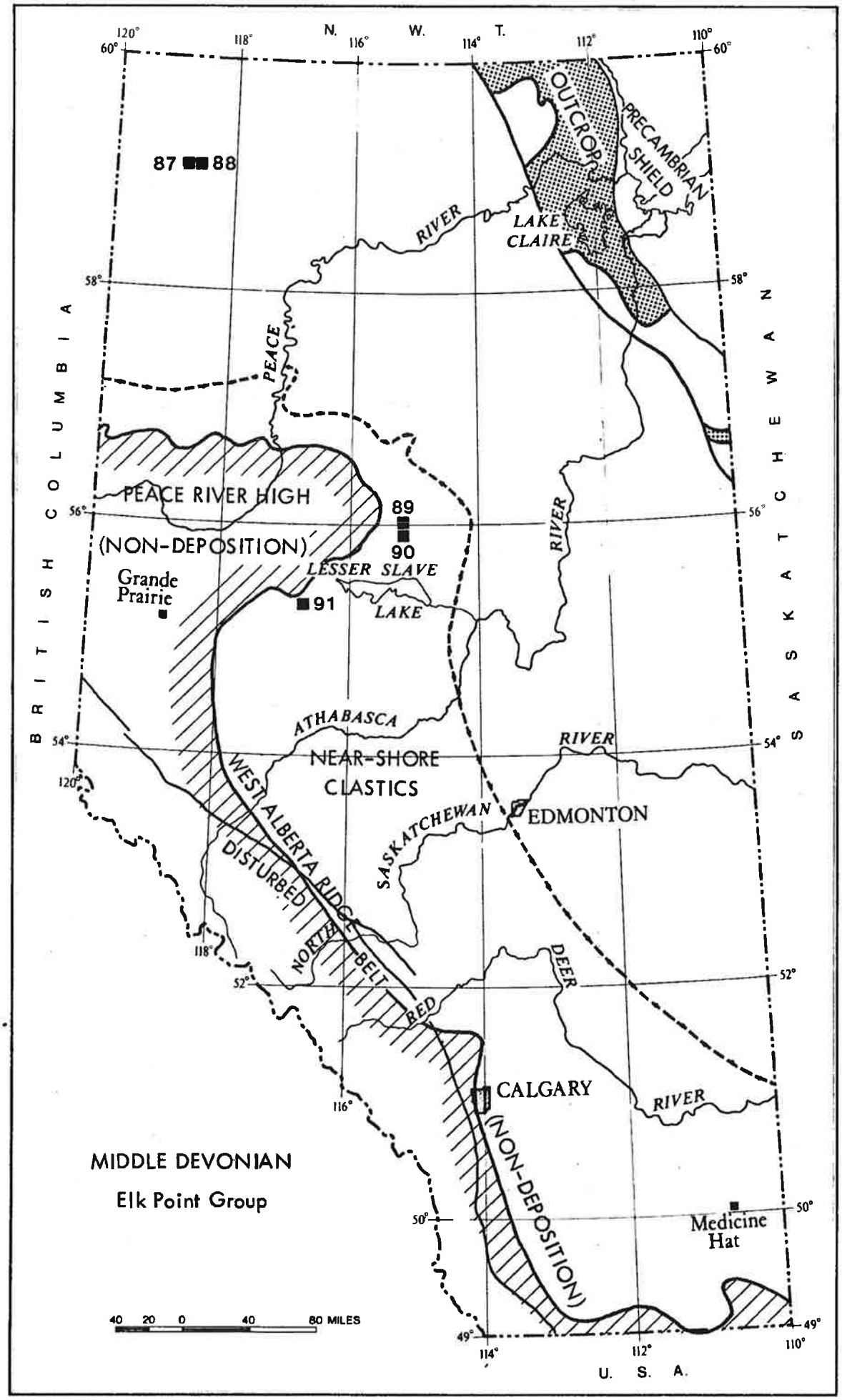
Method of production: Flowing Depth: 8529-8547; 8556-8582 ft. (perforations)

Date sampled: 13th Jan. 1971

Remarks:

S	(%)						
V	(ppm)	0.054	(0.0029)				
Cl	(ppm)	13.35	(0.4412)				
Na	(ppm)	0.735	(0.0089)				
Fe	(ppm)						
Ni	(ppm)	0.179	(0.0231)				
Zn	(ppb)	90.0	(8.6)	Au	(ppb)		
Co	(ppb)	1.28	(0.24)	Sb	(ppb)		
I	(ppb)	420.3	(11.13)	Cr	(ppb)		
Mn	(ppb)	22.73	(0.7542)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.111	(0.0141)
Hg	(ppb)	2.0	(0.55)	Eu	(ppb)		
Cs	(ppb)	19.45	(0.97)	Ga	(ppb)		
Br	(ppb)						
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis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PEACE RIVER HIGH
(NON-DEPOSITION)
Grande Prairie
LESSER SLAVE LAKE
ATHABASCA RIVER
NEAR-SHORE CLASTICS
WEST ALBERTA RIDGE
DISTURBED BELT
NORTH
SASKATCHEWAN
EDMONTON
DEER
RED
CALGARY
(NON-DEPOSITION)
Medicine Hat
BRITISH COLUMBIA
SASKATCHEWAN
U. S. A.
MIDDLE DEVONIAN
Elk Point Group
40 20 0 40 80 MILES

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RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 87

Field (pool): Zama (Keg River C)

Stratigraphic unit: M. Devonian, Elk Point Gp., Keg River Fm.

Well name: HB Zama North 3-11-116-5

Location: Lsd 3 Sec 11 Twp 116 Rge 5 W 6 Mer

Source of sample: Separator Pressure: 150 psig Temperature: 32 °F

Method of production: Pump Depth: 4751-4800 ft. (perforations)

Date sampled: 3rd March 1970

Remarks:

S	(%)	1.324	(0.0839)				
V	(ppm)	2.788	(0.0313)				
Cl	(ppm)	42.37	(0.9512)				
Na	(ppm)	3.347	(0.0461)				
Fe	(ppm)	0.842	(0.21)				
Ni	(ppm)	4.66	(0.150)				
Zn	(ppb)	68.23	(10.3)	Au	(ppb)		
Co	(ppb)	35.92	(2.46)	Sb	(ppb)		
I	(ppb)	71.72	(6.113)	Cr	(ppb)	14.53	(3.41)
Mn	(ppb)	27.08	(0.9115)	Pb	(ppb)		
Se	(ppb)			Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.30	(0.10)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	1407.0	(23.0)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 88

Field (pool): Zama (Muskeg B)

Stratigraphic unit: M. Devonian, Elk Point Gp., Muskeg Fm.

Well name: HB Zama North 8-17-116-4

Location: Lsd 8 Sec 17 Twp 116 Rge 4 W6 Mer

Source of sample: Wellhead Pressure: 100 psig Temperature: 35 °F

Method of production: Pump Depth: 4740-4806 ft. (perforations)

Date sampled: 3rd March 1970

Remarks:

S	(%)	1.506	(0.1120)				
V	(ppm)	9.456	(0.0911)				
Cl	(ppm)	43.70	(0.9778)				
Na	(ppm)	1.945	(0.0330)				
Fe	(ppm)	0.465	(0.104)				
Ni	(ppm)	17.2	(0.263)				
Zn	(ppb)	109.7	(7.83)	Au	(ppb)		
Co	(ppb)	23.2	(0.52)	Sb	(ppb)	3.291	(0.4304)
I	(ppb)	281.2	(12.94)	Cr	(ppb)	7.74	(3.40)
Mn	(ppb)	23.61	(0.8833)	Rb	(ppb)		
Se	(ppb)	15.8	(3.24)	Sc	(ppb)		
Hg	(ppb)			Eu	(ppb)	0.268	(0.095)
Cs	(ppb)			Ga	(ppb)	15.82	(0.555)
Br	(ppb)	728.0	(25.61)				
As	(ppb)	254.0	(4.94)				

* Relative standard deviation (counting statistics) in parenthesis

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 89

Field (pool): Nipisi (Keg River Sandstone A)

Stratigraphic unit: M. Devonian, Elk Point Gp., Keg River Ss.

Well name: Arco IOE Nipisi 12-5-81-8

Location: Lsd 12 Sec 5 Twp 81 Rge 8 W 5 Mer

Source of sample: Wellhead Pressure: 50 psig Temperature: 58 °F

Method of production: Pump Depth: 5760-5763 ft. (perforations)

Date sampled: 15th July 1970

Remarks:

S	(%)	0.286	(0.0323)				
V	(ppm)	1.11	(0.0172)				
Cl	(ppm)	17.97	(0.4871)				
Na	(ppm)	2.74	(0.032)				
Fe	(ppm)						
Ni	(ppm)	2.43	(0.050)				
Zn	(ppb)	697.0	(14.3)	Au	(ppb)		
Co	(ppb)	9.38	(0.314)	Sb	(ppb)		
I	(ppb)	567.1	(14.77)	Cr	(ppb)		
Mn	(ppb)	44.25	(0.8502)	Rb	(ppb)		
Se	(ppb)	13.1	(0.079)	Sc	(ppb)	0.066	(0.0089)
Hg	(ppb)	398.6	(29.34)	Eu	(ppb)		
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	642.6	(15.2)				
As	(ppb)						

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 90

Field (pool): Nipisi (Gilwood A)

Stratigraphic unit: M. Devonian, Watt Mountain Fm., Gilwood Ss. (Elk Point Gp.)

Well name: Pacific et al Nipisi 10-10-80-8

Location: Lsd 10 Sec10 Twp 80 Rge 8 W5 Mer

Source of sample: Separator Pressure: 40 psig Temperature: 56 °F

Method of production: - Depth: 5564-5574; 5580-5588 ft. (perforations)

Date sampled: 14th July 1970

Remarks:

S	(%)	0.187	(0.020)
V	(ppm)	0.190	(0.0111)
Cl	(ppm)	0.50	(0.063)
Na	(ppm)	1.206	(0.0169)
Fe	(ppm)		
Ni	(ppm)	0.70.	(0.07)

Zn	(ppb)	352.8	(14.4)	Au	(ppb)
Co	(ppb)	4.69	(0.546)	Sb	(ppb)
I	(ppb)			Cs	(ppb)
Mn	(ppb)			Rb	(ppb)
Se	(ppb)			Sc	(ppb)
Hg	(ppb)			Eu	(ppb)
Cs	(ppb)			Ga	(ppb)
Br	(ppb)	105.3	(3.96)		
As	(ppb)	12.3	(1.015)		

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 91

Field (pool): Gilwood (Gilwood A)

Stratigraphic unit: M. Devonian, Watt Mountain Fm., Gilwood Ss. (Elk Point Gp.)

Well name: Gr Pl Triad Musk Gilwood 1-9

Location: Lsd 1 Sec 9 Twp 73 Rge 18 W 5 Mer

Source of sample: Upstream of treater inlet Pressure: 30 psig Temperature: - °F

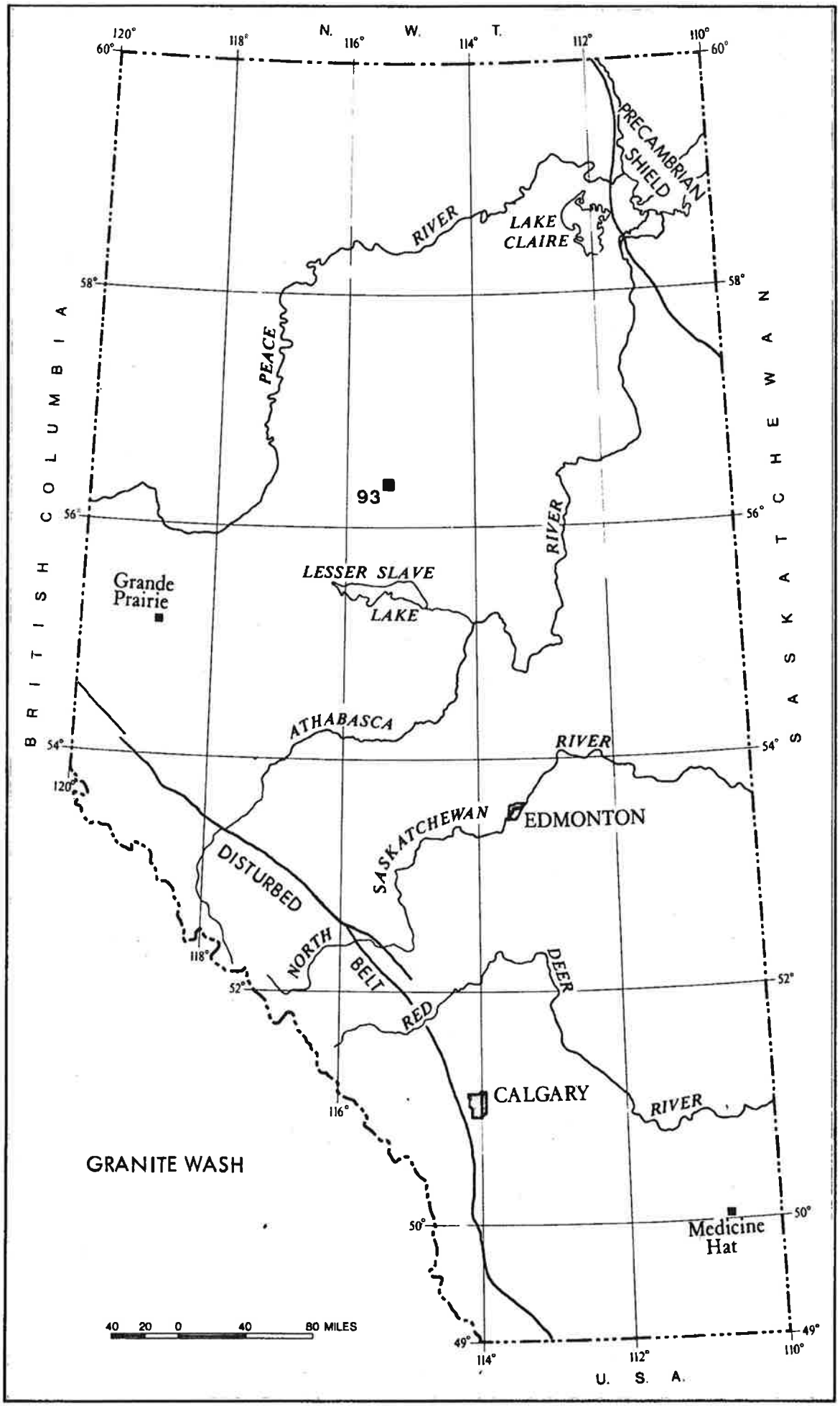
Method of production: - Depth: 8103-8121 ft. (perforations)

Date sampled: 10th July 1969

Remarks:

S	(%)	0.101					
V	(ppm)	0.490	(0.0113)				
Cl	(ppm)	11.070	(0.3097)				
Na	(ppm)	0.347	(3.26)				
Fe	(ppm)	0.876	(0.169)				
Ni	(ppm)	6.74	(0.12)				
Zn	(ppb)	730.0	(23.8)	Au	(ppb)		
Co	(ppb)	6.58	(0.340)	Sb	(ppb)	2.55	(0.469)
I	(ppb)	59.44	(4.749)	Cr	(ppb)	21.3	(2.44)
Mn	(ppb)	59.91	(1.170)	Rb	(ppb)		
Se	(ppb)			Sc	(ppb)	0.152	(0.019)
Hg	(ppb)	3.02	(0.362)	Eu	(ppb)	0.604	(0.16)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	265.0	(5.86)				
As	(ppb)	2.25	(0.383)				

* Relative standard deviation (counting statistics) in parenthesis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Grande Prairie
LESSER SLAVE LAKE
ATHABASCA RIVER
SASKATCHEWAN RIVER
EDMONTON
DISTURBED BELT
NORTH
RED DEER RIVER
CALGARY
GRANITE WASH
Medicine Hat
40 20 0 40 80 MILES
U. S. A.

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 93

Field (pool): Loon (Granite Wash A)

Stratigraphic unit: Granite Wash

Well name: Chevron Suptst Dome Loon S 4-9-85-9

Location: Lsd 4 Sec 9 Twp 85 Rge 9 W 5 Mer

Source of sample: Separator Pressure: 70 psig Temperature: 60 °F

Method of production: Flowing Depth: 5068-5069 ft. (perforations)

Date sampled: 15th July 1970

Remarks:

S	(%)	0.203	(0.0285)				
V	(ppm)	0.587	(0.0126)				
Cl	(ppm)	12.19	(0.7810)				
Na	(ppm)	0.889	(0.0084)				
Fe	(ppm)						
Ni	(ppm)	0.900	(0.021)				
Zn	(ppb)	1126.0	(14.41)	Au	(ppb)		
Co	(ppb)	3.57	(0.127)	Sb	(ppb)	0.190	(0.095)
I	(ppb)			Cr	(ppb)	3.63	(0.940)
Mn	(ppb)	77.31	(1.183)	Rb	(ppb)		
Se	(ppb)	11.6	(0.871)	Sc	(ppb)		
Hg	(ppb)	150.1	(8.37)	Eu	(ppb)	0.091	(0.0322)
Cs	(ppb)			Ga	(ppb)		
Br	(ppb)	237.0	(6.85)				
As	(ppb)	22.0	(0.585)				

* Relative standard deviation (counting statistics) in parenthesis

Alberta
RESEARCH COUNCIL

TRACE ELEMENTS IN ALBERTA CRUDE OILS -- DATA SHEET

Sample number: 94
Field (pool): Athabasca oil sand deposit
Stratigraphic unit: L. Cretaceous, Mannville Gp., McMurray Fm.
Well name: -
Location: Lsd - Sec - Twp - Rge - W - Mer
Source of sample: - Pressure: - psig Temperature: - °F
Method of production: - Depth: ft.
Date sampled: -
Remarks:

S	(%)	3.881	(0.4426)				
V	(ppm)	176.5	(2.3011)				
Cl	(ppm)	7.961	(1.1630)				
Na	(ppm)	20.78	(0.343)				
Fe	(ppm)	254.2	(4.83)				
Ni	(ppm)	71.88	(1.07)				
Zn	(ppb)			Au	(ppb)	1.316	(0.216)
Co	(ppb)	1998.0	(14.0)	Sb	(ppb)	27.7	(1.65)
I	(ppb)			Cr	(ppb)	1682.0	(12.51)
Mn	(ppb)	3853.0	(35.30)	Rb	(ppb)	720.2	(44.1)
Se	(ppb)	517.1	(22.4)	Sc	(ppb)	190.7	(0.60)
Hg	(ppb)			Eu	(ppb)	23.2	(0.77)
Cs	(ppb)	68.53	(6.41)	Ga	(ppb)	267.0	(7.79)
Br	(ppb)	103.6	(12.81)				
As	(ppb)	320.9	(10.24)				

* Relative standard deviation (counting statistics) in parenthesis

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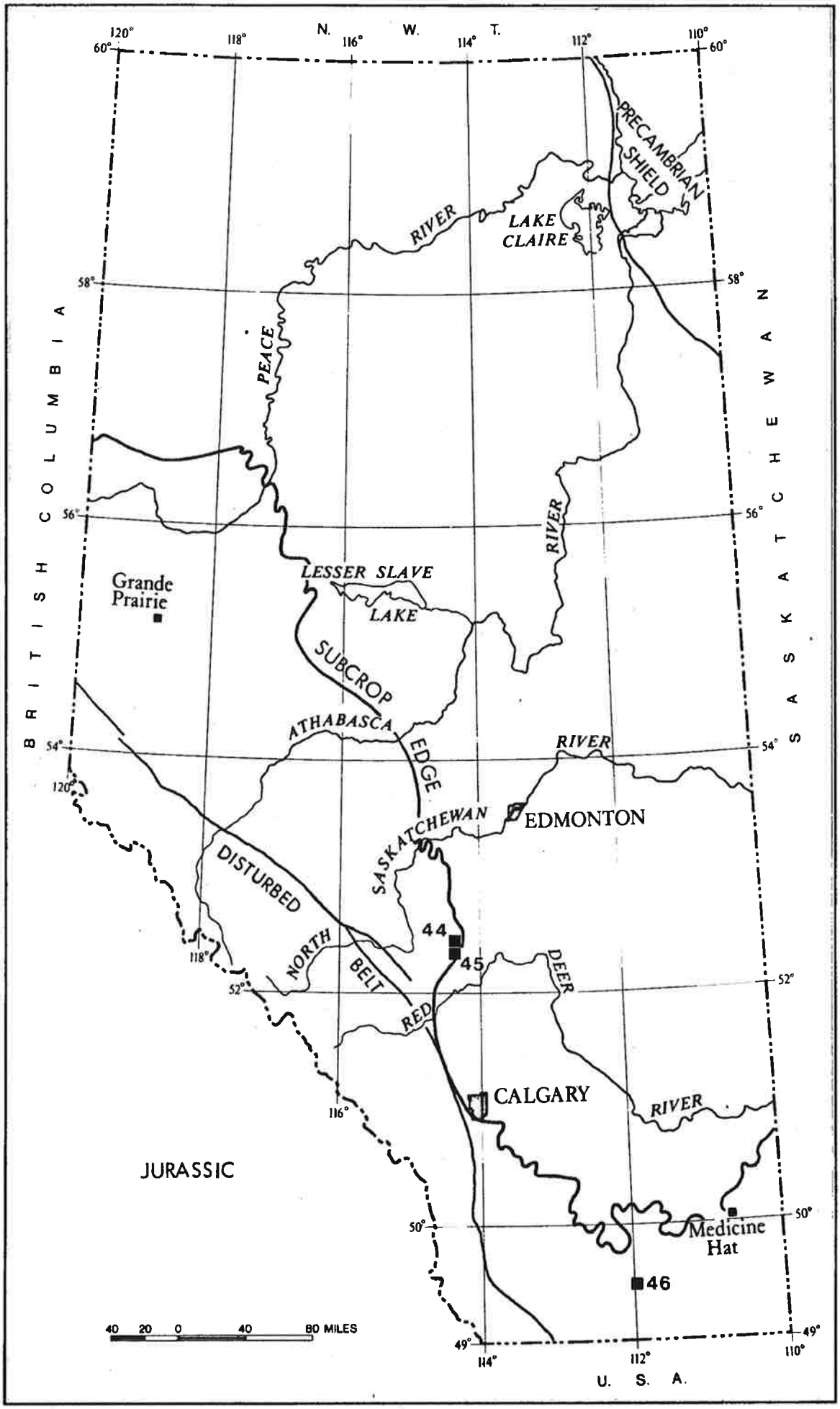
RESEARCH COUNCIL

ADDITIONAL DATA - Sample No. 94

Oil sand sample from syncrude Canada Ltd. Extracted in Soxhlet extractor with benzene, centrifuged, and evaporated down using mild heating (< 80° C).

Ramsbottom carbon residue: 12.57%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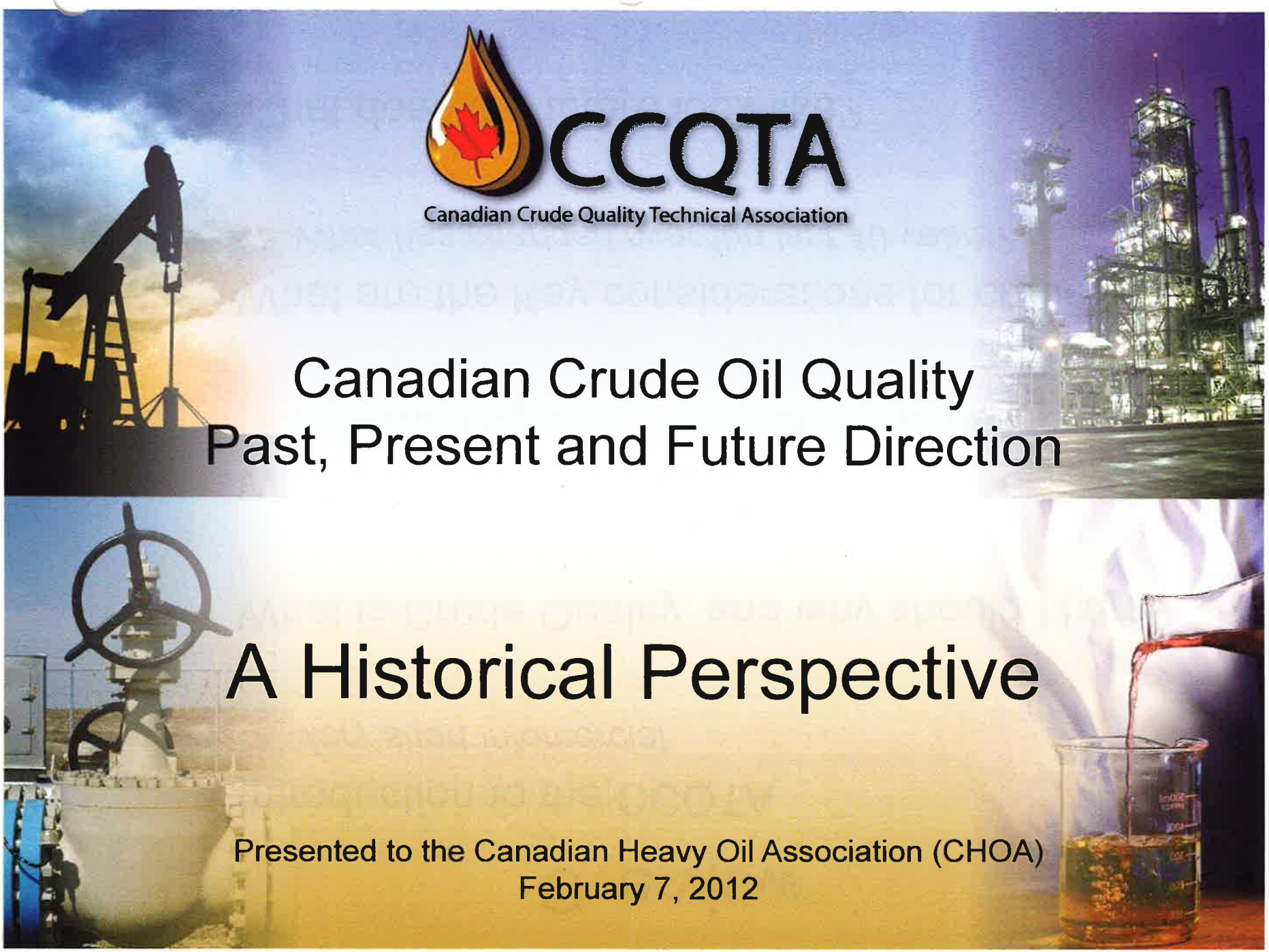

CCQTA
Canadian Crude Quality Technical Association
Canadian Crude Oil Quality
Past, Present and Future Direction
A Historical Perspective
Presented to the Canadian Heavy Oil Association (CHOA)
February 7, 2012

Overview

- Introduction to the CCQTA
  - *Very short infomercial*
- What is Crude Quality, and why should I care?
- Where do I sit in the value chain
  - *Producer/Upgrader, Transportation or Refinery*
- What are the Key considerations for crude quality
  - *What has changed over the last 40 years?*
- What does the future look like?

CCQTA

- Canadian Crude Quality Technical Association
- Founded in 1996, the CCQTA is a registered not-for-profit Association formed to:
  - *Facilitate the resolution of common crude oil quality issues through collaborative research*
  - *Develop and run "industry research projects"*
  - *Improve industry knowledge and awareness of crude oil quality*
- 59 member companies, 7 active, 3 pending projects
  - →TAN, Condensate Quality, Emulsion Characterization, Heavy Oil Compatibility, H2S Measurement in Crude, Phosphorus, Oilsands Bitumen Processability
  - →H2S PVT, Bitumen Dewatering, Shrinkage Calculation
- For more information, visit: **www.ccqta.com**

Crude Oil Quality

- Broad Measure:
  - *Quality is:*
    - →the degree to which a product (or service)
      - – *meets the expectations (and needs) of the end user*
- The end user of crude oil may be:
  - *Production facility*
  - *Pipeline/transportation system*
  - *Refinery/Upgrader customer*
- Crude oil quality starts with the oil in the reservoir, and changes with each handling step along the way to "finished products"

What Does Quality Mean?

- Depends on where you are..
  - *For a SAGD producer*
    - →Get crude in the pipeline and out to my customers
      - *Meeting pipeline specifications and declared grade quality*
  - *For a pipeline company*
    - → I have rules that you must meet to allow this in the pipeline
      - *Related to pumping, transport and pipeline integrity*
    - → Make every effort to deliver material "at expected quality"
  - *For a refinery customer*
    - → I need to know what molecules will be delivered
    - → How can I convert these into saleable products?
    - → I need to be in compliance with my operating permits

Where Are You?

- Consider a SAGD production facility
  - →Need to satisfy pipeline specifications
  - →Many grades marketed with similar "specs"
- *Key mainline pipeline specifications* *(for Heavy Crude)*
  - → API Gravity (density) @ 15C
    - *940 kg/m3 (19 API)*
  - → Viscosity (at the reference pipe temperature)
    - *Max 350 cSt* ***...dilbits hit this constraint before density!***
  - → Sediment and water (BS&W)
    - *Max 0.5 vl%*
  - →Maximum Reid Vapour Pressure (RVP)
    - *From light ends (ie Butane and lighter) in crude*
  - → No organic chlorides (corrosion concern)
  - → No olefins (fouling and product specification concern)
  - → No "deleterious components"
    - *physical or chemical characteristics that may render crude oil "not readily transportable"*

Quality Measures - Cushing

Industry continues to expand Quality testing requirements for even legacy benchmark crudes like West Texas Intermediate (WTI)

…linked to physical delivery linked to NYMEX and CME (Chicago Mercantile Exchange)

Parameter	Test Method	Comment
API gravity	ASTM D287	Density
Sulphur	ASTM D4249	Contamination
Vapour Pressure	ASTM D5191	Butane in crude
Viscosity	ASTM D445	Pipeline pumping
Pour Point	ASTM D97	Wax

- New Recommended additional quality parameters

Parameter	Test Method	Comment
Acid Number	ASTM D664	TAN number
Distillation	ASTM D7169	Hydrocarbon
Metals	ASTM D5708	Ni, V and Fe
MCR	ASTM 4350	Microcarbon residue

Refining Crude Oil

- Need more than sulphur and gravity to determine the "acceptability and valuation" of crude oil in a refinery.
- The crude oil's hydrocarbon footprint and contaminants determine the value of crudes
- Refinery has computer models (LP) representing the refinery hardware capabilities and constraints
  - *Used to determine the crude diet and relative value of crude oils*
    - →Recognizes price/quality of crude oil
    - →Looking for an "optimal" crude slate to meet the refinery production requirements
- The most valuable crude may NOT be the "cheapest" crude?

Refining Crude Oil

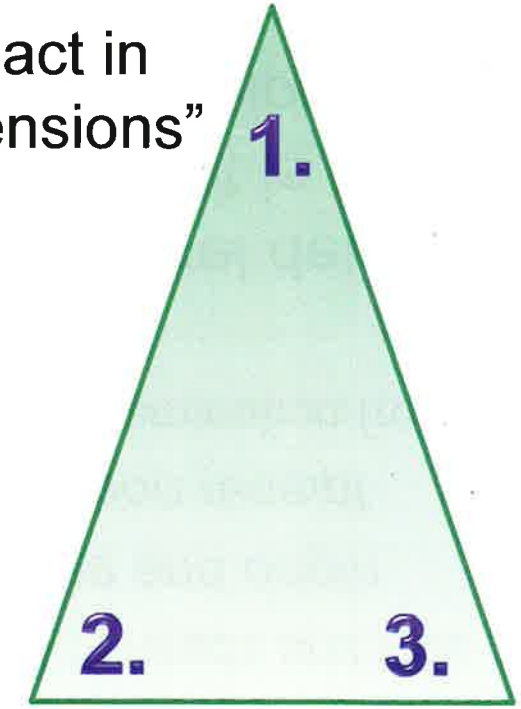
- Crude assays are the "fingerprints" of the oil
  - *Think "DNA"*
- Traditionally, crude sold on as-is basis represented with a "typical" assay
  - *No specific warrants on quality but batch production data is generally known and measured for at least API gravity, sulphur content, and BS&W*
- A good crude assay is:
  - *Expensive and potentially complex or problematic*
  - *Necessary for comprehensive crude oil evaluation*
    - →Provides details on the hydrocarbon (and contaminant ?) make-up used for performance predictions
- Logistics are important as a produced barrel and a delivered barrel can be different

What to Measure - Hierarchy

Property	Must Have	Better	Best
API Gravity (density)	✓		
Sulphur	✓		
TAN	✓		
MCR whole crude	✓		
MCR residue	✓		
Light ends analysis	✓		
Distillation	✓		
Metals – NI, V, FE	✓		
Salt		✓	
Sediment		✓	
RVP, Pour Point, Nitrogen, viscosity		✓	
Arsenic and Mercury			✓
Se, Cu, Na, Mg			✓
Asphaltenes and Mercaptan Sulphur			✓

Crude Quality in a Refinery

Quality impact in "three dimensions"



Presence

Just being there can be a problem
- Toxins
- Banned material

Mercury

"No surprises"

Quantity

The amount of material exceeds tolerable levels

Sulphur, Benzene

Variability

The variability of a material in crude may exceed plant process capabilities

Salt, Sediment

Hydrocarbon Portion

- What can effect hydrocarbon change?
  - *Reservoir performance over time*
  - *Pipeline batching*
    - →Mixing of heads and tails
  - *Diluent type and origin*
  - *Handling upon receipt*
    - →Heel contamination from tankage
- Ideally, the barrel delivered "to" the pipe will look the same when it is received by the customer at the "end" of the pipeline
  - *Distance and handling can alter this outcome*

Contamination

- Non-hydrocarbon contamination
  - *Salt, sediment and metals (in the oil)*
  - *Oil treatment chemicals*
  - *Well stimulation chemicals*
  - *Pipeline additives*
- Specific environmental considerations
  - *Mercury, selenium, arsenic*
  - *Others:*

    →Copper, titanium, manganese, sodium, silicon
- Surprises are never good

  →Mercury, phosphorous, silicon
- Can impact Prime Refinery Concerns
  - Corrosion, Water Treatment, Catalyst Poisons, Safety

Historical Perspective
Crude Oil Quality

What was crude quality like in the 60's & 70's??

Historical Overview

- Produced crude
  - *Light sweet crude oil was the norm*
  - *Blending was less prevalent (production quality=sales quality)*
  - *BS&W and sulphur were the key qualities of interest*
- Refineries produced gasoline, diesel, jet, fuel oil and asphalt
  - *Simpler operation*
    - → most plants were not running at capacity
  - *Most product streams were virgin (straight run)*
  - *Frequent plant shutdowns for cleaning/maintenance*
  - *Desalting for corrosion control*
    - → always been a key contaminant focus

Historical Quality Parameters

- BS&W (Bottoms Sediment and Water)
  - *used as for volumetric accounting of non hydrocarbon portion*
    - →Reported on a % basis
    - →Used in pipeline industry worldwide
- API gravity
  - *was a useful tool for establishing value to a refiner based on a estimate of yields of various products…for conventional crudes, gravity was a proxy for yield value*
- Sulphur content
  - *used to estimate the sulphur levels of products*

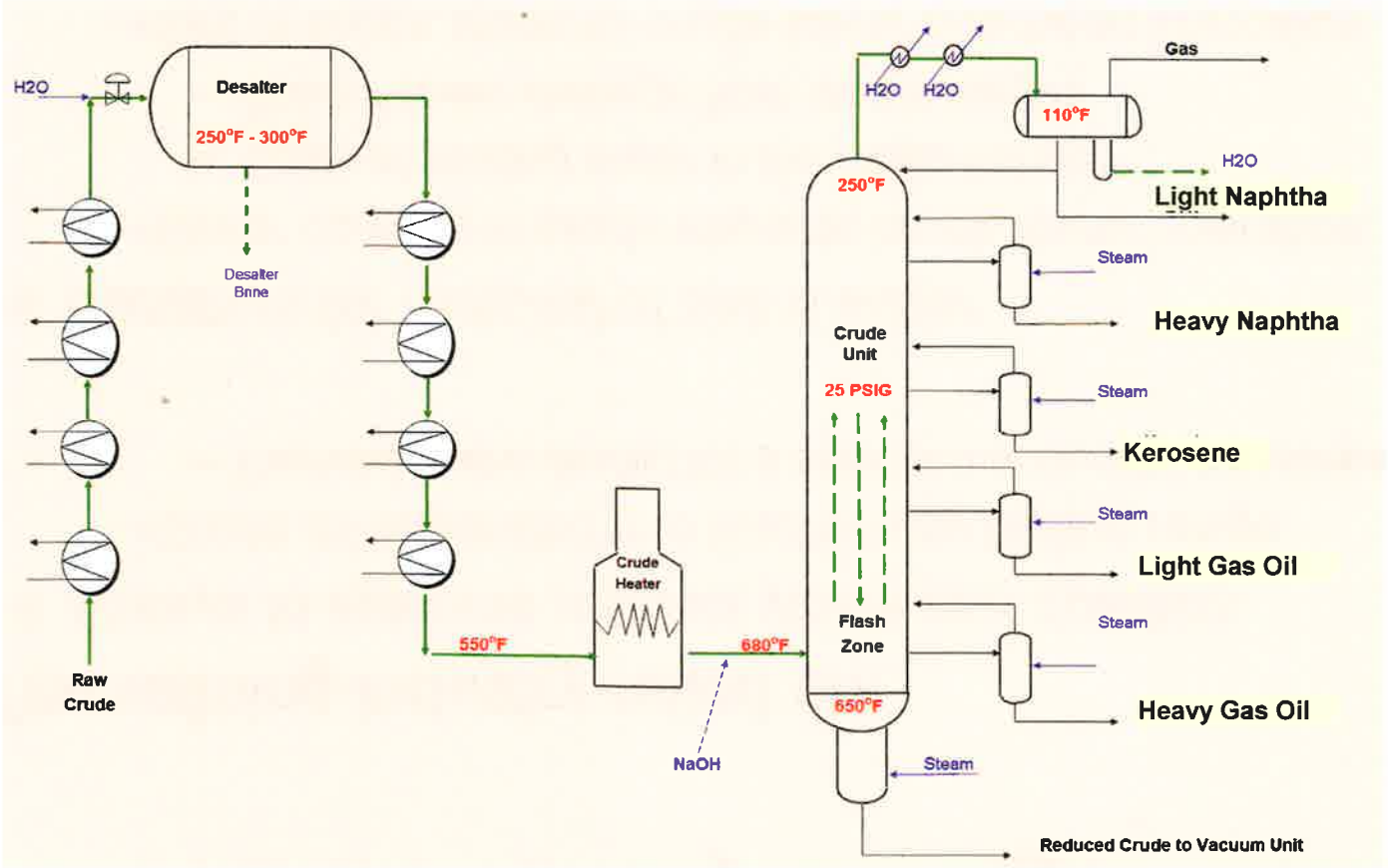
Life was much simpler then…..

Historical Quality Management

- The refining industry relied on:
  - *Assays to estimate product yields and qualities.*
    - →Crude oil separated into fractions by boiling range
      - *Derived matrix tested for a selection of quality measures*
  - *Desalters for removal of salt & water*
    - →Water content in crude impacts crude tower operation
      - *Costs of heating water in the crude furnace*
      - *Fear of tower damage from steam surges*
    - →Salt in crude leads to crude tower overhead corrosion
      - *Salt usually found in produced water within crude*

Simplified Crude Unit Operation

Crude Unit is the first processing step in the refinery



Current WCSB State

- Light oil reserves depleted
  - *New heavy oil production dominating*
- New chemicals/processes developed to enhance production & recovery
- Oil sand production using steam methods
  - *Cyclical steam stimulation (CSS)*
  - *Steam Assisted Gravity Drainage (SAGD)*
- Synthetic Crude Oil (SCO) and bitumen blends (Dilbit and Synbit) driving marketplace change
  - *Bitumen derived thus blending is an important producer marketing metric*
- Environmental controls continue
  - *Air, water, land*

Market Changes

- Crude oil demand increases
  - *Price volatility with high crude oil prices*
- Heavy oil being produced economically
  - *Wide adoption of SAGD technology*
  - *Low cost natural gas*
    - → lower quality/heavier crude oil in the market
- Increased quality risk
  - *Introduction of non-traditional components into the crude pool (diluents)*
- More crude choices for refineries and upgraders
  - *Greater variability in crude supply requires more work to optimize crude selection*
    - → needing data and improved selection methods

Pipeline Changes

- More pipelines built to handle production
  - *greater mix of products being transported*
- Interface mix not insignificant
  - *increases the potential for product quality impact*
- More concerns/issues associated with tank heels
  - *More grades, same tanks increases complexity*
- Increased volumes of trucked-in production
  - *Including oil from waste oil treatment facilities*

More complexity in the delivery systems impacts ultimate quality of delivered crude oil.....

Refinery Changes

- Fewer Refineries
  - *Profitable remaining refineries debottlenecking for future*
    - → focussed on running at capacity with new crudes
- Cdn feedstock gets heavier and superceding foreign heavy depletions
  - *More conversion/upgrading units needed to process more "bottom of the barrel"*
    - →thermal cracking (coking) & catalytic upgrading of heavier fraction
  - *Yield and product quality implications*
    - →Processing heavier, more contaminated feedstocks

Refinery Changes

- Run Lengths increasing to improve profitability
  - *No longer shutdown every year to clean/maintain*
  - *Target 4 to 6 year run lengths*
  - *Driving more need for managing incoming crude quality understanding*
- Environmental Pressure
  - *Low sulphur fuel production*
  - *Tighter specifications on air emissions*
    - →Stack emissions (SOX and NOX), GHG's
  - *Implication for waste disposal and water treatment*
    - →By-products and effluent
  - *Carbon footprint and carbon intensity*
    - →Well to wheels measures

Refineries have to work harder to stay in business

Finished Product Changes

- Driven by fuel efficient engine requirements
  - *Tighter performance specifications on*
    - → gasoline, diesel, jet fuel, etc.
  - *Less tolerance for contaminants*
- Low Sulphur Products everywhere…..
  - *Low sulphur gasoline, ULSD, Low sulphur heavy fuel oil in the works*
  - *Requires additional hydrotreating processing to reduce sulphur content*
    - →Additional cost and complexity

Oil sands derived feedstocks have unique processing challenges due to the nature of the chemistry of highly condensed aromatics and high sulphur content.

Production vs Refining

- **Production Direction**
  - → *Heavier feed*
  - → *More entrained contaminants*
  - → *More additives used in production*
  - → *More complex product mix in pipelines*
  - → *Greater risk for introduction of changing diluents*

- **Refinery Direction**
  - → *Higher throughputs*
  - → *Longer run lengths*
  - → *More complex/sensitive units*
  - → *Tighter product specifications*
  - → *Seeking more predictability*
  - → *More environmental pressure*

Feedstock Quality vs Refinery Quality capabilities are being pushed in different directions

Present Day Crude Oil Quality

What is being done to manage Crude Quality?

Industry Crude Quality Initiatives

Refinery Based

- Improved documentation/sharing of plant incidents
  - *What went wrong and why*
    - →Learning from experience; share through conferences
- Increased/improved monitoring of feedstock quality; for pipeline batches and cargos
  - *Procedures, testing methods providing key metrics*
- Better understanding of contaminant quality issues
  - *What they are and how to deal with them successfully*
- Active participation in industry based quality projects
  - *Targeted collaborative research for deal with industry wide problems*

Producer Based

- Canadian Association of Petroleum Producers
  - *CAPP/Enbridge incentive tolling agreement*
    - →Recognition of value in crude quality management best practices by pipeline
- Crude quality information widely available
  - *Continuous testing and reporting of key quality metrics for Western Canadian Sedimentary Basin crudes funded by Producers for entire Industry*
    - →www.crudemonitor.ca
- Introduction of new specifications for more product classes
  - *sweet/sour/heavy crudes, high/low TAN crudes, synthetics/resid free*
- Tightening of diluent stream specifications
  - *Redwater condensate quality (CRW)*
    - →Enbridge managed common diluent with detailed specifications

Pipeline Based

- Stricter guidelines for new/intermittent product addition to the pipeline system
- More/tighter monitoring of key product specifications
  - *Gravity (density), viscosity, olefins*
- Adoption of improved on-line monitoring technologies
  - *Real time information on contaminant levels*
- Interface management protocols/batch sequencing
  - *Procedures to minimize pipeline interface material*
- Managing tank heels/cleaning procedures
  - *Minimizing contamination/mixing risk*

Service Industry Based

- More additive supplier involvement in multi sector quality discussions
  - *Phosphorus, fluorocarbons, etc.*
- More information exchange between the service sector and other oil industry sectors
- Continuous improvements and regulation within the waste oil treatment industry

Improved Analytical Services

- Continuous improvements in instrument capabilities
  - *Better machines with lower detection limits of specific contaminants*
  - *Development of new on-line instrumentation technologies*
    - →NMR, X-Ray
- Laboratory services
  - *Bring analytical capabilities closer to the "action"*
    - →Production facilities, pipeline, and refinery
  - *Consistency in laboratory practices*
    - →Test method standardization and laboratory certification

Industry Associations

- Collaborative research/testing efforts for a technical focus
  - *Pooled resources*
    - →Results oriented
- Canadian Crude Quality Technical Association (CCQTA)
  - *Canadian producer and North American Refinery based*
  - *formed 1996*
- Crude Oil Quality Association (COQA)
  - *US Refinery centric issues*
  - *formed in RCOQ in 1994*

Formal Alignment Between CCQTA and COQA Started in 2008

Crude Oil Quality

Knowledge Breeds
Understanding

Future Crude Quality Initiatives

Industry Initiatives

- Better understanding of the technical drivers that underpin successful crude oil production/processing
- Communication/education
  - *Providing a factual basis for crude quality in context*
    - →for environmental challenges
- Dispelling "myths"
  - *What, exactly, is "dirty oil"?*
- Application to Production, Transportation and Refining sectors

Producer Based

- Improved understanding of the implication of production/treating/handling methods on product desirability
  - *Producer → pipeline → refinery customer relationship*
  - *Better data for crude assessment/valuation work*
    - → good data improves decision quality
- Do we need different key crude quality specifications?
  - *More than API gravity, sulphur and BS&W?*
  - *Alignment and EQ of grades into fewer commodities?*
  - *Greater awareness of banned/restricted materials*
- Controllable contaminants
  - *Clean/dry bitumen…..?*

Pipeline Based

- Greater efforts to control product quality
  - *Larger batch sizes to reduce interfacial contamination (adds scheduling complexity)*
  - *Tighter product specifications (best practices to enhance crude netback)*
  - *More product segregation (quality enhancemnet supports incremental cost?)*
- Quality monitoring of streams
  - *More product quality testing of incoming streams*
  - *Use of on line instrumentation, real-time information*
  - *Continue Batch tracking metrics*

Refining Industry

- Improved understanding/documentation of crude quality impacts on refinery operation
- Better monitoring of feedstock quality
  - *Ensure LP "Assays" are valid*
  - *Use of on-line instrumentation to track crude properties and contaminants*
  - *Incorporation of real time data into process control*
- Valuation of crude quality
  - *Provide incentives for the production and delivery of consistent quality crudes*
  - *Prediction/Modeling of seasonal heavy crude blending*
- Environmental compliance considerations
  - *Trace contaminants testing ongoing*

Building Understanding

- Improved discussion with industry sector members of crude quality impact of products, applications and processes
- More disclosure of information on product/application chemistries to industry
- Greater role in the monitoring and management of crude quality in all sectors
- Links to International views and markets
- Role of key associations to foster best practices
  - *Canadian Association of Petroleum Producers*

Getting the Facts Right

- Improved/standardized testing methods
  - *Analytical service providers*
- Greater role in product quality testing for all sectors
- Continuous improvements in analytical testing
  - *Faster turnover time*
  - *Lower detection limits*
  - *Development of more at-line/on-line instruments*
- *"With good crude quality information, better decisions can be made"*

Conclusions

- Increased awareness of the implications of crude quality does benefit all industry sectors
  - *Producer, Upgrader, Pipeline, Refiner*
- "I need the right information to make good decisions…."
  - *Know the molecules and avoid "surprises"*
  - *A good crude assay is a good starting point*
  - *Better understanding of the role of contaminants*
  - *Anticipating future quality changes based on new crude oil sources and production techniques.*
- Industry collaboration solving common technical problems has wide reaching benefits

CCQTA/COQA Joint Meeting

Will have normal Joint Agenda…

- Tue Jun19 CCQTA focus
- Wed Jun20 COQA format
- Thu Jun21 Golf Outing

…80 rooms planned

…online booking open shortly

…seeking sponsors ☺

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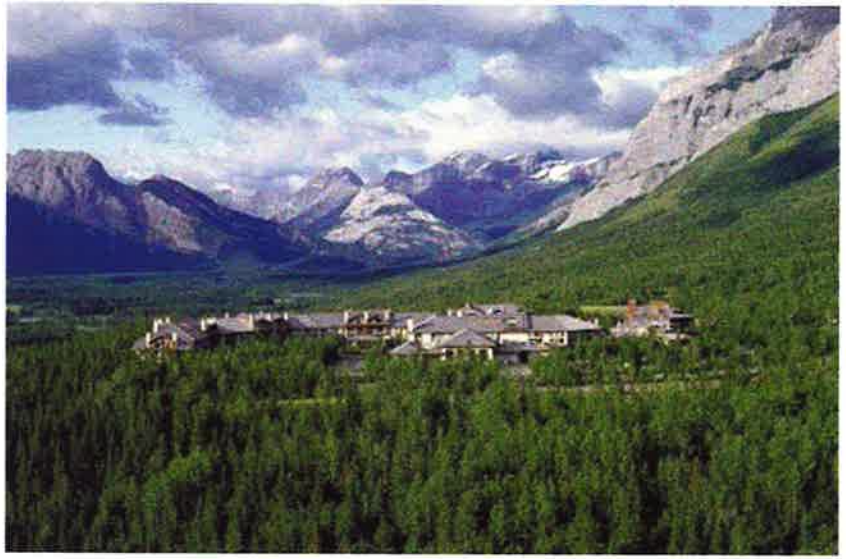

Date: Scheduled for week of June 18th 2012

Location: Kananaskis Resort – located 90 km/50mi west of Calgary

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…End ☺

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EVALUATION OF CRUDE OIL QUALITY

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Abstract

Fourteen type crude oils originated from USA, Mexico, Africa, Middle East, Russia, Canada, Colombia, Ecuador, and Venezuela having density and sulfur in the range API = 12.1 ÷ 40.8; S = 0.4 ÷ 3.3% and total acid number varying in the range TAN = 0.1 ÷ 3.72 mg KOH/g oil have been investigated. The studied crude oils have been classified into four groups: I group – light, low sulfur one (30 - 40^0 API; S ≤ 0.5 % mass); II group – light, sulfur one (30-40^{0}API; S= 0, 5 - 1.5 % mass); III group – heavy, high sulfur one (15-30^{0}API; S=1.5 ÷ 3.1% mass); IV group – extra-heavy, high sulfur one (15^{0}API, S ≥3 % mass). It has been established that extra-heavy crude oils (IV group) are characterized by light fraction low content, diesel fractions low cetane index, vacuum gas oil fractions low K-factor and vacuum residue fractions high Conradson carbon content. It also has been found on the base of crude oil averaged prices for June 2009 (Brent crude oil price = 69 US $/ barrel) that the difference of the Ist and IVth group crude oil prices was about 9 US $/ barrel. This difference amounts up to 22 US $/ barrel, as the crude oil price rises up to 140 US $/ barrel. The high acid crude oil price (such having TAN > 0.5 mg KOH/g oil) may be approximately 9 US $/ barrel lower than one determined on the base of density and sulfur content for the corresponding group.

Key words: opportunity crudes, crude value, high acid crudes, crude oil quality

1. Introduction

Crude cost is the single most important determinant for the profitability of an oil company. With crude costs accounting for around 80% of the refinery expenditures, processing cheaper crudes can have a very positive impact on refinery margins [2].

For refineries that have freedom in crude choice, the selection of an optimum crude package is of vital importance. This requires intense teamwork between the trader and the supply/ manufacturing economist and typically linear-program (LP) models of individual refineries are routinely used to determine the relative use values among crudes [3-14]. To achieve optimal crude selection and processing decisions, a refiner must have exact information refer to crude oil quality [15-35]. This includes: crude oil TBP-curve as main data for correct operation of refinery crude oil atmospheric-vacuum distillation plants [36]; the characteristics of crude oil fraction:

Naphtha fraction:
- density, naphthenes and arene content, octane number, sulfur and metal as lead and arsenic content, affecting isomerization and reforming plants operation;

Kerosene fraction:
- density, sulfur, pour point, freezing point, arene content (for aviation fuel); cetane index, low temperature properties (CFPP), pour point and diesel and fuel oil blending viscosity;

Diesel fraction:
- density, sulfur, arene content, cetane index, pour point, low temperature properties (CFPP), diesel and fuel oil blending viscosity;

Heavy gas oil:

- UOP "K" factor or hydrogen content, nitrogen and Conradson carbon quality characteristics of this fraction as feed for Fluid Catalytic Cracking and other conversion processes;

Atmospheric residue and vacuum gas oils:

- density, pour point, sulfur, viscosity, metals for fuel oil blending;
-UOP "K" factor or content of hydrogen, nitrogen and Conradson carbon as suitability criteria for conversion processing (Fluid Catalytic Cracking and Hydrocracking)

Vacuum residue:

- density, pour point, sulfur, viscosity, metals for fuel oil blending; UOP "K" factor or hydrogen, nitrogen and Conradson carbon content as suitability criteria for their processing by the conversion processes (Catalytic cracking and Hydrocracking).

In practice, data is also needed for additional cuts in order to generate property profiles as a basis for recutting to actual refinery straight-run products. For example: the increase demand of diesel in Europe drives the European refiners to cut naphtha fractions and in this way to increase diesel yield [37]. This information may be obtained by use of standard laboratory test methods normally API or ASTM. One extensive laboratory analysis of crude oil may cost over 20 000 US $ and generally it takes two to four weeks [36]. In practice it is extraordinary difficult and very expensive to carry out full laboratory analysis of every cargo crude oil received at the refinery. This has resulted in development of a number computing methods that permit prediction of petroleum fractions properties by routine laboratory analyses [38-45]. These methods require information about petroleum fractions distillation characteristics, density, sulfur content, viscosity and refraction. This information may be obtained only after crude oil TBP distillation, analysis which duration is not less than 24 hours [46]. Another important information of crude oil quality is organic acid content since organic acid presence at high concentration may generate number of problems at equipment operation and especially at crude oil atmospheric-vacuum distillation plants via their acid high corrosion activity [47-53]. The crude oil total acid number is an indicator of the organic acids content in the crude oil. It is well known that problem crude oils are those which total acid number is over 0.5 mg KOH/g oil [53]. But not always low acid number of crude oil feed means that it is not possible corrosion problem to occur due to the presence of naphthenic acids in definite petroleum fractions [49]. Such cases exist when definite fraction has very low sulfur content and presence of organic acids. Sulfur presence result to formation of protective layer of ferric sulfide that renders difficult organic acid attack of the metal and by that manner the equipment accelerated corrosion is reduced. Because of lower prices of high acid crude oil feeds (total acid number over 0.5 mg KOH/g crude oil) their processing is one opportunity to increase crude oil processing profit after use of adequate programs for minimizing the risk of accelerated corrosion and subsequent equipment untimely, unplanned damage [1].

There are basically four types of crude available to refiners around the world. They are light-sweet (30-40^0 API, <0,5 wt% S), light-sour (30-40^0 API, 0.5-1.5 wt% S), heavy-sour (15-30^0 API, 1.5-3.1 wt% S) and extra-heavy (<15^0 API and >3 wt% S). High acid crude oils (HACs) represent the fastest-growing segment of global oil production. California, Brasil, North Sea, Russia, China, India and West Africa are known to supply HAC_S. Over half of the world's oil supply is heavy and sour. Hence, synthetic petroleum feeds derived from bitumen sands are considered as high acid ones. Table 1 represents comprehensive assay of fourteen crudes which belong to the four basic type petroleum feeds. The crude comprehensive assay of all investigated crude oils was obtained in the Lukoil Neftochim Research Laboratory. Price of the crudes was obtained from the Energy Information Agency (EIA) and this price was related to June 2009 [54]. These data show that for June month 2009 the average prices of four type basic petroleum feeds are as follow:

I group – light low sulfur ones (30-40^0 API; S ≤ 0.5 % mass) = 68.5 US $ / barrel
II group – light sulfur ones (30-40^0 API ; S=0,5 1.5 % mass) = 67.5 US $ / barrel
III group – heavy, high sulfur (15-30^0 API S=1.5 ÷ 3.1% mass) = 66.8 US $ / barrel
IV group – extra-heavy high sulfur (15^{0}API, S ≥3 % mass)= 60.0 US $ / barrel

Herein, it is seen that the price of light, low sulfur crude oil from Louisiana nevertheless of its low sulfur content and low density (High API) according to which it

should be applied to first group crude oils, is equal to that of the lowest quality crude oils of fourth group. This may be explained by the high acid number of the crude oil that in combination with low sulfur content means high corrosion reactivity and so unfavorable feed for processing [49]. The price of EMERAUDE (origin – Congo) and TIA JUANA PESADO (origin Venezuela) crude oils is not included in Table 1 as they are not available on the market due to their extra high acid number. The crude oil light fraction (distilled up to 343°C) content decreases in the following order: I group > II group > III group > IV group. Vacuum residue content decreases in reverse sequence. The average characteristics of the fractions derived from crude oil groups included in Table 1 show (Table 2) that IV group crude oil naphtha has the highest octane number and diesel fractions of the same crude oils have the lowest cetane number that corresponds to the conclusions drawn up by other authors [14].

Table 2 Average characteristics of the fractions derived from crude oils presented in Table 1

Typical properties	RON			Cetane index			K- factor	Conradson carbon
	Fractions			Fractions			Fraction	fraction
Crude oil group	IBP-70	70-100	100-190	190-235	235-280	280-343	343-565	> 565
I	70.7	61.0	44.4	46.0	46.2	53.3	12.08	14.2
II	71.1	58.3	42.0	45.0	45.0	50.2	11.84	21.8
III	66.8	50.9	32.8	50.0	49.8	51.2	11.78	21.9
IV	75.5	64	53.6	37.0	37.4	41.4	11.56	23.1

Vacuum gas oil fraction from group IV has the lowest K-factor and due to this at conversion processes from them will be produced the lowest yields of valuable products like naphtha, diesel fraction and C_3 and C_4 alkenes. The IV group crude oil vacuum residues have the highest Conradson carbon and so they are unfavorable feeds for catalytic conversion processes. They are suitable for processes like cocking at which as by product is produced low valuable coke. All of these characteristics show that from IV group crude oils may be produced the lowest yields of high valuable transport fuels. Similarly, they require higher costs for processing due to required high degree of upgrading at hydrotreating processes and higher consumption of high prices hydrogen. The difference in prices between group I and group IV crude oils has been 8.5 US $ / barrel on June 2009, but as a whole this difference depends on the crude oil price. The plot of Brent type crude oil price change for period 1997 – 2009 is shown on Figure 1. It may be seen from it that difference of prices between high quality crude oils (low density and low sulfur content) and low quality crude oils (high density and high sulfur content) increases along with the increase of crude oil price. For example at crude oil price of order 30 US $ / barrel the difference between Brent и Maya crude oils is about 30 US $ / barrel and at 140 US $ / barrel it is already 20 US $ / barrel. Hence, as higher is the crude oil price so more advantageous is to process low quality crude oils.

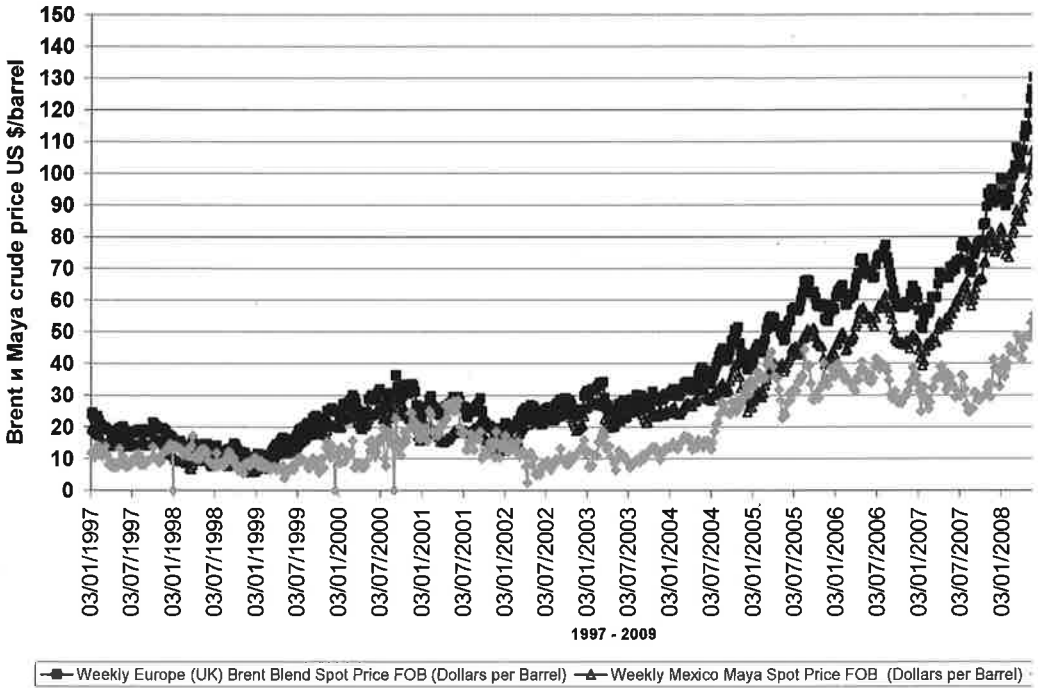

Figure 1 Crude Brent price and difference in prices of crudes Brent and Maya in the period 1997 - 2209 (Source: Energy Information Administration; http://tonto.eia.doe.gov/dnav/pet/pet_pri_refoth_dcu_nus_m.htm

Conclusions

The following conclusions may be drawn up as a result of the carried out investigation:

1. The high quality crude oils (low density and low sulfur content) are characterized by light fractions high content, diesel fractions high cetane index, vacuum gas oils high K-factor and vacuum residue fractions low Conradson carbon content.
2. Low quality crude oils (high density and high sulfur content) are characterized by light fractions low content, diesel fractions low cetane index, vacuum gas oils low K-factor and vacuum residue fractions high Conradson carbon content.
3. The difference between the high quality and low quality crude oils increases with the crude oils price growth.
4. At high crude oil prices the most profitable will be refineries having available heavy petroleum residues conversion plants that allow them to process low quality crude oils to high valuable transport fuels.
5. The high acid number and low sulfur content of crude oil results to its price decrease by about 9 US $/ barrel at crude oil price about 69 US $/ barrel.
6. It is proved the conclusion drawn up by other authors that high acid crude oil diesel fractions have low cetane index.

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Table 1 Comprehensive assay of crude oils under study

Crude oil feed	LLS	WTI	MAD	BR	CAB	WTS	CL	ORIEN	REB	ISTH	AL	AM	KUW	AH	MAYA	LLOYD	EMER	TIA
Density, ° API	36.1	40.8	40.5	38.3	31.7	34.1	29.3	29.2	31.8	33.3	33.4	28.5	31.4	27.4	22.2	20.7	23.6	12.1
Density, g/cm^3	0.844	0.821	0.823	0.833	0.867	0.854	0.880	0.881	0.867	0.859	0.858	0.884	0.869	0.890	0.921	0.930	0.912	0.985
Sulfur, % mass	0.45	0.34	0.78	0.40	0.17	1.64	0.51	0.88	1.53	1.49	1.77	2.85	2.52	2.80	3.30	3.15	0.6	2.7
Pour point, ° C	-37	-29	-24	-42	18	-46	-1	-7	-12	-42	-54	-23	-15	-45	-36	-32.22	-38.9	-1.1
Viscosity at 37.8 °C, mm^2/s	4.3	3.9	2.7	3.9	19.0	4.6	15.1	13.6	8.3	5.9	8.4	17.5	9.8	27.6	102	101	47.3	3700
V, ppm	1.2	1.6	1.00	6.0	2.40	6.4	15.0	83.5	46.7	49.1	13.5	20.0	30.0	57.3	314.0	105	5	284
Ni, ppm	7.1	1.6	0.7	1.0	15.9	3.7	34.6	39.2	14.7	9.4	3.3	12.0	8.0	16.4	52.0	52.7	50	38.5
Conradson carbon, % mass	1.1	1.1	1.4	2.1	3.6	3.3	4.3	6.3	3.9	4.3	3.6	5.9	5.3	6.8	12.0	9.18	7.57	11.2
Acid number, mg KOH(g	0.58	0.10	0.2	0.10		0.11	0.17	0.39	0.56	0.06	0.00	0.06	0.15	0.10	0.28	0.78	3.72	3.61
IBP -70° C % mass of crude oil	3.2	5.1	5.3	6.0	2.3	4.7	0.8	3.3	4.0	3.7	3.9	3.5	3.8	4.1	2.4	3.9	1.4	0.15
Density, g/cm^3	0.657	0.651	0.658	0.660	0.647	0.645	0.652	0.654	0.656	0.648	0.649	0.654	0.652	0.653	0.682	0.648	0.659	0.664
Sulfur, % mass	0.018	0.011	0.003	0.000	0.023	0.079	0.025	0.012	0.005	0.017	0.012	0.008	0.030	0.002	0.010	0.022	0.155	0.074
RON	73.3	69.8	64.4	71.8	74.4	69.1	72.3	74.7	70.9	68.3	66.4	64.3	69.1	67.5	69.5	70.5	74.3	87.7
70-100°C % mass of crude oil	3.5	5.2	4.7	5.4	3.0	4.8	2.2	2.7	3.3	4.1	3.4	2.9	3.4	2.4	2.5	2.6	2.4	0.07
Density, g/cm^3	0.715	0.712	0.717	0.719	0.708	0.717	0.706	0.713	0.703	0.701	0.703	0.704	0.696	0.705	0.715	0.714	0.722	0.732
Sulfur, % mass	0.022	0.027	0.004	0.001	0.032	0.143	0.040	0.020	0.013	0.039	0.025	0.025	0.024	0.013	0.024	0.046	0.183	0.140
Naphthenes,% v/v	23.0	37.6	18.5	28.6	30.5	27.1	25.9	47.1	25.2	19.6	14.7	14.6	14.6	16.7	21.7	34.1	47.1	52
Arenes, % v/v	7.5	4.3	6.4	9.6	2.1	9.0	0.6	2.9	3.7	8.6	6.5	6.6	4.4	4.4	28.1	8.7	1.6	2.8
RON	66.6	62.8	52.3	62.5	60.7	59.3	59.6	66.7	50.9	54.9	51.9	51.5	53.3	47.0	57.2	57.6	66.5	75.2
100-190°C % mass of crude oil	16.9	21.6	19.9	17.3	10.5	16.4	11.7	12.7	13.0	15.5	14.6	11.4	12.3	10.3	9.3	8.5	9.3	1.2
Density, g/cm^3	0.772	0.766	0.763	0.774	0.765	0.774	0.757	0.775	0.764	0.734	0.757	0.765	0.753	0.754	0.759	0.779	0.766	0.804
Sulfur, % mass	0.031	0.044	0.011	0.002	0.065	0.351	0.030	0.031	0.053	0.054	0.047	0.111	0.080	0.108	0.198	0.191	0.290	0.214
Naphthenes,% v/v	36.0	38.5	19.8	35.0	45.3	38.4	28.4	45.1	37.1	20.5	18.3	19.2	22.9	21.7	27.9	38.2	56.3	72.3
Arenes,% v/v	13.4	13.2	18.1	17.5	7.7	18.0	10.5	9.6	11.7	13.6	15.8	16.4	13.9	12.4	29.2	16.5	6.0	11.1
RON	49.8	50.6	33.4	43.2	44.8	43.1	36.5	46.8	39.2	44.3	33.3	38.9	35.7	23.5	34.9	55.2	54.8	69.6
190-235°C .% mass of crude oil	10.3	9.0	9.6	6.8	5.9	8.3	7.9	7.4	7.4	8.1	8.1	7.3	6.6	6.4	5.3	4.2	5.0	2.6
Density	0.809	0.811	0.803	0.812	0.807	0.815	0.802	0.825	0.812	0.805	0.800	0.802	0.797	0.800	0.810	0.836	0.834	0.854
Sulfur, % mass	0.074	0.088	0.202	0.012	0.082	0.646	0.073	0.191	0.211	0.254	0.252	0.421	0.436	0.428	0.327	0.426	0.304	0.458
Arenes,% v/v	15.8	13.7	21.6	22.0	13.2	21.8	15.8	19.9	21.8	21.3	22.2	22.1	19.5	19.7	19.0	21.7	11.2	18.1

Table 1 Comprehensive assay of crude oils under study, contd.

Crude oil feed	LLS	WTI	MAD	BR	CAB	WTS	CL	ORIEN	REB	ISTH	AL	AM	KUW	AH	MAYA	LLOYD	EMER	TIA
Aniline point, °C	140	143	140.1	139	149.0	134	151	138	136	139	142	141	143	145	132	125.2	131.7	126.1
Smoke point, mm	22	24	22	20	23	20	21	22	22	26	23	22	26	24	15	20.6	20.2	16.5
Cetane index	46	45	49	44	47	43	49	40	45	48	50	49	51	50	46	36.1	36.7	31.2
Freezing point, °C	-59	-42	-43	-64	-24	-53	-52	-53	-57	-51	-46	-43	-41	-47	-52	-68.5	-70.5	-75.2
Pour point, °C	-67	-54	-59	-54	-52	-61	-58	-60	-67	-65	-44	-48	-49	-41	-58	-100.6	-77.8	-85.2
Viscosity at 37.8°C mm^2/s	1.48	1.49	1	1.44	2	1.48	1.67	1.55	1.52	1.41	1.39	1.43	1.41	1.40	1.47	1.6	1.6	1.9
Viscosity at 98.9°C mm^2/s	0.69	0.74	0.79	0.79	0.76	0.70	0.83	0.74	0.72	0.70	0.69	0.71	0.69	0.70	0.74	0.8	0.8	0.9
235-280°C % mass of crude oil	10.8	8.8	9.4	9.1	6.3	7.9	8.6	9.0	7.7	7.7	8.2	7.5	6.9	6.7	5.8	5.1	5.4	3.8
Density, g/cm^3	0.835	0.831	0.825	0.832	0.827	0.840	0.828	0.848	0.837	0.832	0.826	0.830	0.826	0.830	0.841	0.866	0.870	0.882
Sulfur, % mass	0.20	0.17	0.46	0.07	0.11	0.90	0.18	0.39	0.61	0.59	0.74	0.97	1.16	0.90	1.06	0.61	0.32	0.81
Arenes,% v/v	17.9	13.7	21.6	22.9	16.7	25.2	17.5	29.0	27.8	21.3	21.4	21.6	22.1	23.8	21.6	22.6	11.4	24.4
Aniline point, °C	153	158	153.3	151	160.3	143	159	147	142	145	152	151	151	154	138	128.1	136.8	132
Smoke point, mm	17	21	20	17	21	16	17	16	18	21	20	18	22	21	10	17.5	13.1	10.4
Cetane index	46.2	45.1	49	44.3	47	43.4	49.2	39.9	44.9	47.8	49.6	49.0	50.9	49.7	45.8	36.1	36.7	31.2
Freezing point, °C	-14	-9	-9.2	-33	17.3	-17	-13	-12	-18	-32	-1	-3	-8	-4	-26	-36.4	-40.5	-46.1
Pour point, °C	-27	-12	-16	-16	-11	-28	-16	-16	-28	-24	-5	-4	-16	-11	-18	-61.7	-51.2	-56.1
Viscosity at 37.8°C mm^2/s	2.9	2.5	2	2.4	3	2.5	2.9	2.7	2.6	2.3	2.4	2.4	2.4	2.4	2.6	2.9	3.5	3.5
Viscosity at 98.9°C,mm^2/s	1.1	1.1	1.1	1.1	1.2	1.0	1.2	1.1	1.1	1.1	1.1	1.0	1.0	1.0	1.1	1.2	1.3	1.3
280-343°C % mass of crude oil	16.0	11.3	12.6	11.4	10.9	10.4	12.44	12.8	11.2	11.4	11.1	10.6	10.5	9.36	8.78	8.8	7.5	8.14
Density, g/cm^3	0.857	0.850	0.849	0.858	0.849	0.868	0.850	0.869	0.864	0.862	0.856	0.863	0.858	0.861	0.876	0.896	0.899	0.912
Sulfur, % mass	0.361	0.274	0.774	0.327	0.136	1.232	0.326	0.654	1.086	1.161	1.431	1.991	1.930	1.674	2.018	0.890	0.350	1.411
Aniline point, °C	164	174	169	165	174	153	169	160	149	153	162	160	160	162	146	134.1	142.8	138.8
Cetane index	52.0	54.0	54	51.5	55	48.7	54.0	48.2	49.7	50.4	52.3	50.1	51.5	50.7	46.4	41.2	40.4	37.5
Pour point, °C	10	25	30.4	23	32.0	12	27	27	29	18	31	35	18	24	22	-23.0	-27.0	-32.5
Viscosity at 37.8°C mm^2/s	5.7	5.5	5	5.0	5	5.2	5.9	5.7	5.5	4.9	5.0	4.8	5.2	5.0	5.5	6.4	8.1	10.1
Viscosity at 98.9°C,mm^2/s	1.8	2.0	1.6	1.7	2.0	1.7	1.9	1.8	1.8	1.8	1.8	1.6	1.7	1.7	1.9	2.1	2.1	2.4
343-565°C % mass of crude oil	31.1	26.6	28.1	29.3	38.3	31.1	31.7	26.6	31.2	30.5	32.0	31.4	32.0	29.9	24.4	25.7	26.7	41.9
Density, g/cm^3	0.908	0.896	0.893	0.902	0.900	0.922	0.904	0.920	0.923	0.922	0.920	0.927	0.922	0.929	0.932	0.944	0.937	0.967
Sulfur, % mass	0.792	0.444	1.448	0.603	0.160	2.042	0.557	1.026	1.809	1.924	2.441	3.288	3.268	2.931	3.023	2.9	0	2.3
K-factor	11.91	12.14	12.16	12.05	12.14	11.82	11.98	11.81	11.80	11.80	11.85	11.73	11.82	11.74	11.66	11.5	11.68	11.3

Table 1 Comprehensive assay of crude oils under study, contd.

Aniline point, °C	192	202	202	193	211	179	192	185	168	170	178	176	180	175	163	161.9	175	159.1
Crude oil feed	LLS	WTI	MAD	BR	CAB	WTS	CL	ORIEN	REB	ISTH	AL	AM	KUW	AH	MAYA	LLOYD	EMER	TIA
Ni, ppm	0.1	0.1	0		0	0.1		0.4	0.2	0.1	0.0	0.0	0.1	0.0	0.6	0.0	0	ND
V, ppm	0.0	0.0	0.1		0.1	0.1		0.2	0.2	0.8	0.1	0.1	0.4	0.1	0.1	0.1	0	ND
Conradson carbon, % mass	0.3	0.4	0.5	0.1	0.8	0.7	0.4	0.6		0.4	0.2	0.3	0.9	0.3	0.1	0.5	0	1.2
Atmospheric residue,.% of crude oil	38.1	37.2	37.2	42.1	60.1	45.7	56.3	51.2	52.2	48.5	49.9	56.4	54.5	59.6	66.0	66.6	68.1	84.0
Viscosity at 98.9°C,mm²/s	11.2	16.7	10.0	15.7	38.5	28.4	38.7	44.4	30.9	30.6	24.7	46.3	49.2	116.5	617.0		188	510
Ni, ppm	16.4	4.4	1.9	2.4	26.7	10.1	61.5	72.3	29.6	19.4	6.7	21.9	14.8	27.7	77.0	75.6	85.2	45.8
V, ppm	3.0	4.4	2.8	14.2	4.1	17.4	26.7	161.1	92.9	102.3	27.2	35.5	55.2	96.7	462.7	141.9	7.6	338.0
Conradson carbon, % mass	3.0	3.9	4.0	4.9	6.1	7.2	9.6	11.6	7.5	9.0	7.8	10.0	10.6	13.3	15.8	15.5	9.0	14.3
Asphaltenes, % mass	0.6	0.3	0.1	0.9	1.3	1.6	1.5	5.0	1.7	1.9	2.0	4.4	2.5	9.7	8.5	4.8	1.4	3.8
Insoluble in n-Pentane, % mass	1.7	0.9	1.1	0.4	3.3	4.1	3.8	12.9	4.4	5.0	3.1	5.8	6.6	9.5	22.1	12.5	3.6	9.9
Vacuum residue, % mass of crude oil	7.0	10.6	9.1	12.9	21.8	14.6	24.6	24.6	21.0	18.0	17.9	25.0	22.5	29.7	41.5	40.9	41.38	42.1
Viscosity at 98.9°C,mm²/s	768	382	934	400	1210	7440	5364	160000	1290	5300	1313	6582	5059	31112	306862		2260	99000
Ni, ppm	89.2	15.3	8	7.8	73	31.4	140.6	150.0	73.4	52.0	18.6	49.3	35.7	55.7	122.0	123	140	91.4
V, ppm	16.6	15.2	11.0	46.6	11.0	54.1	61.0	335.0	230.6	274.0	75.6	79.9	133.0	194.2	735.0	231	12.4	674.0
Conradson carbon, %mass	16.2	12.6	12.4	15.8	14.1	22.6	21.4	22.8	18.5	23.6	16.5	23.1	24.4	23.7	25.2	25.2	16.5	25.6
Asphaltenes, % mass	4.1	1.3	0.8	2.1	4.3	5.8	6.8	17.3	5.0	5.2	5.2	11.6	5.6	19.6	15.8	9.3	2.7	9.0
Insoluble in n-Pentane, % mass	9.1	2.9	4.7	1.4	9.5	12.8	15.0	38.1	11.1	11.5	9.4	14.4	16.1	17.4	34.7	20.4	5.9	19.8
			6.5															
Price, US $ June 2009	60,10	66.2	71.0	68.5	68.4	66.2	70.74	63.48	68.6	68.3	67.3	66.2	67.8	66.0	62.8	57.15		

Note: Names of the investigated crude oils are as follow:

LLS= LOUISIANA LIGHT SWEET (origin:USA); WTI = WEST TEXAS INTERMEDIATE (origin:USA);
MAD = MURBAN (origin:Abu Dhabi, UAE); BR = BRENT BLEND (origin:Nord sea); CAB = CABINDA (origin:Angola);
WTS = WEST TEXAS SOUR (origin:USA); CL = CANO LIMON (origin:Colombia); ORIEN = ORIENTE (origin:Ecuador);
REB = Russian Export Blend (origin:Russia); ISTH = ISTHMUS (origin:Mexico); AL = ARAB LIGHT (origin:Saudi Arabia);
AM = ARAB MEDIUM (origin:Saudi Arabia);KUW = KUWAIT EXPORT (origin:Kuwait); AH = ARAB HEAVY (origin:Saudi Arabia);
MAYA = MAYA (origin:Mexico); LLOYD = LLOYDMINSTER BLENDED CRUDE (origin:Canada, Alberta);
EMER = EMERAUDE (origin:Congo, Brazzaville); TIA = TIA JUANA PESADO (origin:Venezuela)

U.S. Department
of Transportation

Federal Railroad
Administration

GENERAL HEALTH EFFECTS OF TRANSPORTATION NOISE

DTS-34-RR297-LR2
FRA/RDV-03/01

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The Federal Railroad Administration (FRA) sponsored preparation of this report to support a rulemaking process about the use of locomotive horns and the accompanying environmental impact statement. This document reviews select current and past research on the effect of transportation noise on physiological and psychological health for both people and wildlife.

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1. INTRODUCTION

The Federal Railroad Administration (FRA) has sponsored preparation of this report to support a rulemaking process about the use of locomotive horns and the accompanying environmental impact statement. This document reviews select current and past research on the effect of transportation noise on the physiological and psychological health for both people and wildlife. This review includes research from national and international sources.

1.1 Background

Noise is often defined as any unpleasant or unwanted sound. The earliest record of a government taking action to reduce transportation noise dates back to the Roman Empire just before Julius Caesar. Complaints of the noise caused by the wheels of chariots led to the passage of a law which banned the use of chariots at night.

Two thousand years later - while chariots are no longer a noise concern in the US, we can still relate to too much noise in our communities. In addition to interference with speech, sleep, and thought, noise can lead to annoyance and indirect effects on human health and well-being.

Protection of the public against the possible harmful effects of noise are the goals of organizations including the U.S. Environmental Protection Agency (EPA), the World Health Organization (WHO), the Institute of Noise Control Engineering (INCE), the International Committee on Biological Effects of Noise (ICBEN), and the Occupational Safety and Health Administration (OSHA). These organizations, working with government agencies, as well as other noise- and health-interest researchers, continually monitor transportation and technology trends in order to better understand and mitigate the effects of its by-product, noise, on all living beings.

1.2 Scope and Objectives

The objective of this document is to provide a brief summary of the health effects of environmental noise on people and wildlife. The scope of this document includes all environmental noise with a primary focus on transportation noise, such as aircraft, highway, and rail noise. This document includes a review of select current and past national and international research.

2. TERMINOLOGY

This section presents pertinent terminology used throughout the document. Note: Definitions are generally consistent with those of the American National Standards Institute, Taber's Cyclopedic Medical Dictionary, and Dorland's Illustrated Medical Dictionary.

A-WEIGHTING - A frequency weighting network used to account for changes in human auditory sensitivity as a function of frequency.

ACTIGRAPH - Wrist-watch-sized devices that record human movement and can provide an indication of sleep.

ANNOYANCE - Any bothersome or irritating occurrence.

ANXIETY - A feeling of apprehension, uncertainty, and fear without apparent stimulus. It is associated with physiological changes (tachycardia, sweating, tremor, etc.) - the source of which is often nonspecific or unknown to the individual.

ARRHYTHMIA - Any irregularity in the rhythm of the heart's beating.

AUDIOMETRY - Measurement/testing of the hearing, including aspects other than hearing sensitivity.

AUDITORY THRESHOLD - Minimum audible perceived sound.

CARDIOVASCULAR - Pertaining to the heart and blood vessels.

DAY-NIGHT AVERAGE SOUND LEVEL (abbreviation DNL, denoted by the symbol L_{dn}) - Twenty-four hour average sound level for a given day, after addition of 10 decibels to levels from midnight to 0700 hours and from 2200 hours to midnight. L_{dn} is computed as follows:

$$L_{dn} = L_{AE} + 10*\log_{10}(N_{day} + 10*N_{night}) - 49.4 \quad \text{(dB)}$$

where:

L_{AE} = Sound exposure level in dB (see definition on Page 5);
N_{day} = Number of vehicle pass-bys between 0700 and 2200 hours, local time;
N_{night} = Number of vehicle pass-bys between 2200 and 0700 hours, local time; and
49.4 = A normalization constant which spreads the acoustic energy associated with highway vehicle pass-bys over a 24-hour period, i.e., $10*\log_{10}(86{,}400 \text{ seconds per day}) = 49.4$ dB.

DECIBEL (unit dB) - Unit of level when the base of the logarithm is the tenth root of ten, and the quantities are proportional to power.

EPINEPHRINE - A hormone secreted by the adrenal medulla (inner or central portion of an organ) in response to stimulation of the sympathetic nervous system.

EQUIVALENT SOUND LEVEL (abbreviation TEQ, denoted by the symbol L_{AeqT}) - Ten times the logarithm to the base ten of the ratio of time-mean-squared instantaneous A-weighted sound pressure, during a stated time interval T, to the square of the standard reference sound pressure. L_{AeqT} is related to L_{AE} by the following equation:

$$L_{AeqT} = L_{AE} - 10*\log_{10}(t_2-t_1) \quad \text{(dB)}$$

where L_{AE} = Sound exposure level in dB (see definition on Page 5).

HEARING IMPAIRMENT - A decreased ability to perceive sounds as compared with what the individual or examiner would regard as normal. The result is an increase in the threshold of hearing.

HEARING THRESHOLD - For a given listener and specified signal, the minimum: (a) sound pressure level; or (b) force level that is capable of evoking an auditory sensation in a specified function of trials.

HERTZ - (abbreviation Hz) Unit of frequency, the number of times a phenomenon repeats itself in a unit of time.

L_{AE} (see Sound Exposure Level)

L_{dn} (see Day-Night Average Sound Level)

L_{Aeq} (see Equivalent Sound Level)

NOISE - Any unwanted sound.

NOISE INDUCED TEMPORARY THRESHOLD SHIFT - Temporary hearing impairment occurring as a result of noise exposure, often phrased temporary threshold shift.

NOISE INDUCED PERMANENT THRESHOLD SHIFT - Permanent hearing impairment occurring as a result of noise exposure, often phrased permanent threshold shift.

NOREPINEPHRINE - A hormone produced by the adrenal medulla similar in chemical and pharmacological properties to epinephrine, but chiefly a vasoconstrictor with little effect on cardiac output.

PARACUSIS - Any abnormality or disorder of the sense of hearing.

PEAK SOUND PRESSURE LEVEL - Level of the peak sound pressure with stated frequency weighting, within a stated time interval.

PATHOLOGICAL - Any condition that is a deviation from the normal.

PHYSIOLOGICAL - Of the branch of biology dealing with the functions and vital processes of living organisms or their parts and organs.

PRESBYACUSIA, PRESBYCUSIS - Hearing deterioration occurring after middle age.

PSYCHOPHYSIOLOGICAL - Characteristic of or promoting normal, or healthy functioning of the mind or mental processes.

RECRUITMENT - (Loudness) Abnormal loudness perception.

REVERBERATION - Sound that persists in an enclosed space, as a result of repeated reflection or scattering, after the source has stopped.

REVERBERATION TIME - Of an enclosure, for a stated frequency or frequency band, time that would be required for the level of time-mean-square sound pressure in the enclosure to decrease by 60 dB, after the sound source has stopped.

SOUND EXPOSURE LEVEL (abbreviation SEL, denoted by the symbol L_{AE}) - Over a stated time interval, T (where $T=t_2-t_1$), ten times the base-10 logarithm of the ratio of a given time integral of squared instantaneous A-weighted sound pressure, and the product of the reference sound pressure of 20 micropascals, the threshold of human hearing, and the reference duration of 1 sec. The time interval, T, must be long enough to include a majority of the sound source's acoustic energy. As a minimum, this interval should encompass the 10 dB down points (see Figure 1). In addition, L_{AE} is related to L_{AeqT} by the following equation:

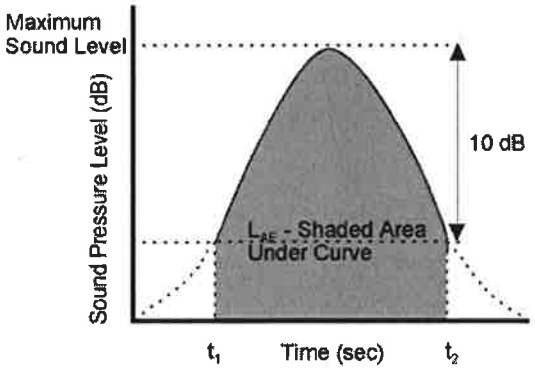


Figure 1. Graphical representation of L_{AE}.

$$L_{AE} = L_{AeqT} + 10*\log_{10}(t_2-t_1) \quad \text{(dB)}$$

where L_{AeqT} = Equivalent sound level in dB (see definition above).

SOUND PRESSURE LEVEL (abbreviation SPL) - Ten times the base-10 logarithm of the ratio of the time-mean-square pressure of a sound, in a stated frequency band, to the square of the reference sound pressure in gases of 20 micropascals.

STRESS - The sum of the biological reactions to any adverse stimulus, physical, mental, or emotional, internal or external, that tends to disturb the organism's state of stability.

THRESHOLD OF HEARING - (see Hearing Threshold)

TINNITUS - A sound of ringing or whistling in the ears, excluding hallucinations of voices. Otological condition in which sound is perceived by a person without an external auditory stimulation.

VASOCONSTRICTION - Tightening or compressing of the blood vessels.

3. GENERAL EFFECTS

The effects of environmental noise on people are generally undesirable. These effects include psychological effects (see Section 4), such as annoyance, and physiological effects (see Section 5), such as hearing impairment and sleep disturbance. In 1972, the Noise Control Act was established to address the concerns of noise as a growing danger to the health and welfare of the Nation's population, particularly in urban areas. In response to the Noise Control Act, the Environmental Protection Agency (EPA) published, " Information of Levels of Environmental Noise Requisite to Protect Public Health and Welfare with an Adequate Margin of Safety" (EPA 1974). The objective of the EPA's "Levels Document" was to identify levels of environmental noise required to protect the public from adverse health and welfare effects (Beranek 1992).

Table 1 summarizes the yearly average equivalent sound level and/or day-night average sound level (L_{eq} or L_{dn}, respectively) recommended as protective of the public health and welfare. Twenty-four-hour exposure levels of 70 decibels or less are identified as necessary to prevent measurable hearing loss over a lifetime. Levels of 55 decibels outdoors and 45 decibels indoors are identified as requisite for preventing activity interference and annoyance. It should be noted that the levels presented in the table below are not to be considered standards, criteria, or regulatory goals. The levels also include a margin of safety and should be viewed as levels below which there is no reason to suspect that the general population will be at risk from any of the identified effects of noise (EPA 1974, FICAN 1992, WHO 2000).

Table 1. Yearly average equivalent sound levels in decibels identified as requisite to protect the public health & welfare with an adequate margin of safety (EPA 1974).*

		Indoor			Outdoor		
	Measure	Activity Interference	Hearing Loss Consideration	To Protect Against Both Effects (b)	Activity Interference	Hearing Loss Consideration	To Protect Against Both Effects (b)
Residential with Outside Space and Farm Residences	Ldn	45			55		55
	Leq(24)		70	45		70	
Residential with No Outside Space	Ldn	45		45			
	Leq(24)		70				
Commercial	Leq(24)	(a)	70	70(c)	(a)	70	70(c)
Inside Transportation	Leq(24)	(a)	70	(a)			
Industrial	Leq(24)(d)	(a)	70	70(c)	(a)	70	70(c)
Hospitals	Ldn	45		45	55		55
	Leq(24)		70			70	
Educational	Ldn	45		45	55		55
	Leq(24)		70			70	
Recreational Areas	Leq(24)	(a)	70	70(c)	(a)	70	70(c)

	Measure	Indoor			Outdoor		
		Activity Interference	Hearing Loss Consideration	To Protect Against Both Effects (b)	Activity Interference	Hearing Loss Consideration	To Protect Against Both Effects (b)
Farm Land and General Unpopulated Land	Leq(24)			(a)	70	70(c)	

Note: * The exposure period which results in hearing loss at the identified level is a period of forty years.

(a) Since different types of activities appear to be associated with different levels, identification of a maximum level for activity interference may be difficult except in those circumstances where speech communication is a critical activity.

(b) Based on lowest level.

(c) Based only on hearing loss.

(d) An Leq(8) of 70 dB may be identified in these situations so long as the exposure over the remaining sixteen hours is low enough to result in a negligible contribution to the 24-hour average, i.e., no greater than an Leq(24) of 60 dB.

In addition to noise exposure guidelines, the EPA and other agencies have established noise exposure limits (see Table 2), above which feasible administrative or engineering controls must be utilized. The permissible equivalent A-weighted sound level for an eight-hour exposure period ranges from 75 dBA for the EPA to 90 dBA for the Occupational Safety and Health Administration (OSHA 1998).

Table 2. Permissible Equivalent Sound Exposure Levels (Beranek 1992).

Duration of Exposure (Hours)	OSHA	U.S. Army	U.S. Air Force	EPA
8	90	85	84	75 *
4	95	89	88	
2	100	93	92	-
1	105	97	96	-
0.5	110	101	100	-
0.25 or less	115 **	105	104	-

Note: * The threshold for detectable noise-induced permanent threshold shift (NIPTS) at 4,000 Hz; exposures exceeding 75 dB may cause NIPTS exceeding 5 dB in 100 percent of the population after cumulative noise exposure of 10 years.

** Ceiling on exposure level and duration.

Long-term exposure to high noise levels results in interference with conversation and other normal human activities, such as sleeping, working, and recreation. To determine if long-term exposure to any specific type of environmental, primarily transportation, noise is a risk factor for cardiovascular or other disorders, would require highly sophisticated epidemiologic field studies. In addition, these field studies would need to place greater emphasis on ensuring appropriate controls to account for other known risk factors such as age, sex, smoking, caffeine, body weight, diet, and hereditary proclivity (HCN 1994, Thompson 1996). The 1982 EPA publication, Guidelines for Noise Impact Analysis, stated that research implicates noise as one of several factors producing stress-related health effects such as heart disease, high-blood

pressure, stroke, ulcers, and other digestive disorders. However, the relationship between noise and these effects has not been fully quantified with most studies on these effects having only limited data available and no definitive conclusions (EPA 1982, Babisch 1993, HCN 1994, IEH 1997, Thompson 1981, Thompson 1996).

The estimated of the number of people exposed to environmental noise at various levels as published in a 1981 EPA document is shown in Table 3 (EPA 1981). As noise levels are directly related to population density (EPA 1974), the general trend is that noise in communities is increasing (Suter 1991). Although in 2000, the Federal Aviation Administration estimated that there will be about 500,000 Americans exposed to significant levels of aircraft noise - down substantially from the 4.7 million estimated in 1981 (FAA 2000). This decrease in population exposure can be attributed to the advent of quieter aircraft.

Table 3. Summary of U.S. population exposed to various day-night average sound levels (L_{dn}) from noise sources in the community (EPA 1981).

L_{dn} (dB)	Estimated Number (in Millions) of People in Each Noise Category					
	Roadway Traffic	Aircraft	Construction	Rail	Industrial	Total
> 80	0.1	0.1	-------	-------	-------	0.2
> 75	1.1	0.3	0.1	-------	-------	1.5
> 70	5.7	1.3	0.6	0.8	-------	8.1
> 65	19.3	4.7	2.1	2.5	0.3	27.8
> 60	46.6	11.5	7.7	3.5	1.9	63.6
> 55	96.8	24.3	27.5	6.0	6.9	92.4

Note: L_{dn} values are yearly averages, outdoors. In addition, there is some overlap among populations exposed to multiple noise sources.

4. PSYCHOLOGICAL EFFECTS

This section contains a discussion of the psychological effects of environmental noise. Psychological effects include annoyance, speech interference, and interference with recreational activities.

4.1 Annoyance

Annoyance with environmental transportation noise is the general adverse reaction of people resulting from speech interference and disturbance to comfort, peace of mind, and sleep. Earlier EPA publications and related reports (EPA 1974, EPA 1973, EPA 1978, Galloway 1970, von Gierke 1977) on transportation noise and human annoyance were later superceded by the 1978 Schultz synthesis (Schultz 1978). This study examined 11 major social surveys on annoyance due to transportation noise. It resulted in the so-called "Schultz curve" which related noise level to annoyance. Since its publication, the Schultz curve has been used nationally and internationally as the nominal response curve for characterizing the average community response to transportation noise.

More recent U.S. Air Force (USAF) research re-examined the Schultz curve as part of a long-term study on the effects of aircraft noise on humans (Finegold 1990, Finegold 1993a, Finegold 1993b, Finegold 1994). In this work, the Percent Highly Annoyed (%HA) relationship, the accepted measure for assessing long-term community noise exposure, was also re-examined. Based on the Schultz data and further refined by the USAF work, the following relationship has become the more widely used model for predicting community annoyance response to all types of transportation noise:

$$\%HA = \frac{100}{1 + e^{(11.13 - 0.14 L_{dn})}}$$

where: %HA is the Percent Highly Annoyed and
L_{dn} is the Day-Night Average Sound Level.

Figure 2 illustrates the Percent Highly Annoyed relationship for the 3 primary types of transportation noise: aircraft, highway/road traffic, and railway. Although the figure implies that people are more sensitive to aircraft noise as compared to noise from highway and rail, more data are required to substantiate this observation. The highway and rail data used in the figure are relatively sparse.

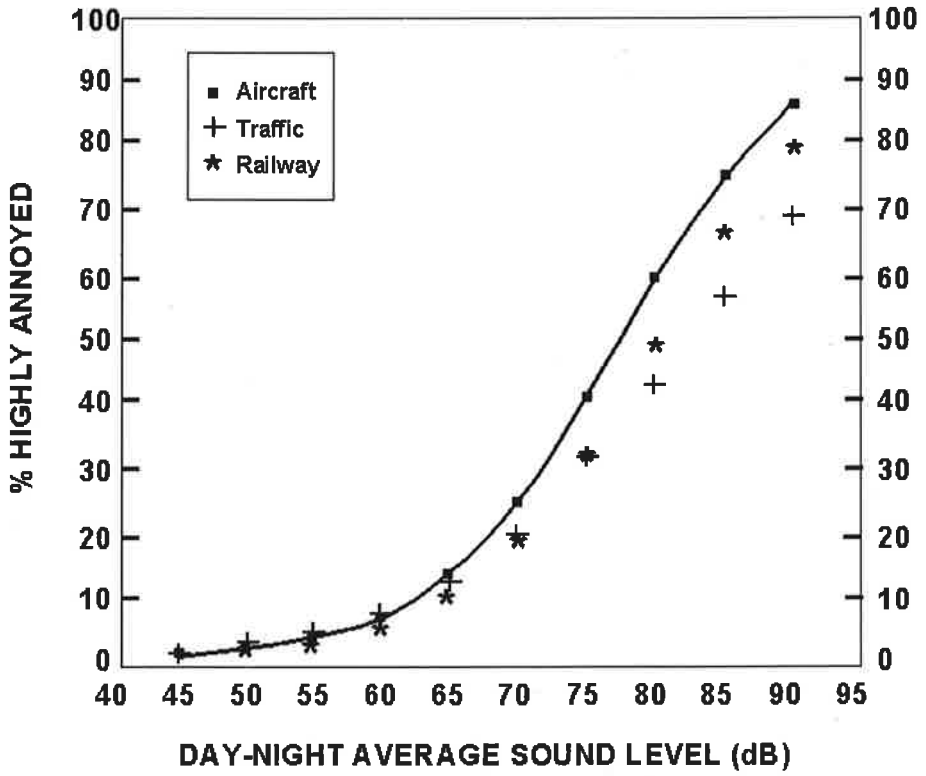


Figure 2. %HA versus L_{dn} from aircraft, road traffic, and railway noise (Finegold 1994).

Further research is required to more fully assess other factors which contribute to a person's level of annoyance in association with transportation noise. These factors may include frequency content, duration, and accompanying rattle/vibration. It is well documented that annoyance increases with sound-induced rattle/vibration (WHO 2000, HCN 1994, Stansfeld 1997). In addition, community attitudes towards noise sources can be an important influence on the degree of annoyance. Annoyance may be reduced when the noise maker maintains a good community relationship and convinces the community that everything that can be done is being done to reduce the noise (FAA 2001).

Finally, it should be mentioned that although annoyance is used to predict community response to noise, complaints are not as useful a measure of community impact (FICAN 1992). This is because annoyance can exist without complaints and, conversely, complaints may occur without high percentages of annoyance. As such, complaints cannot reliably be used to predict community response (FICAN 1992).

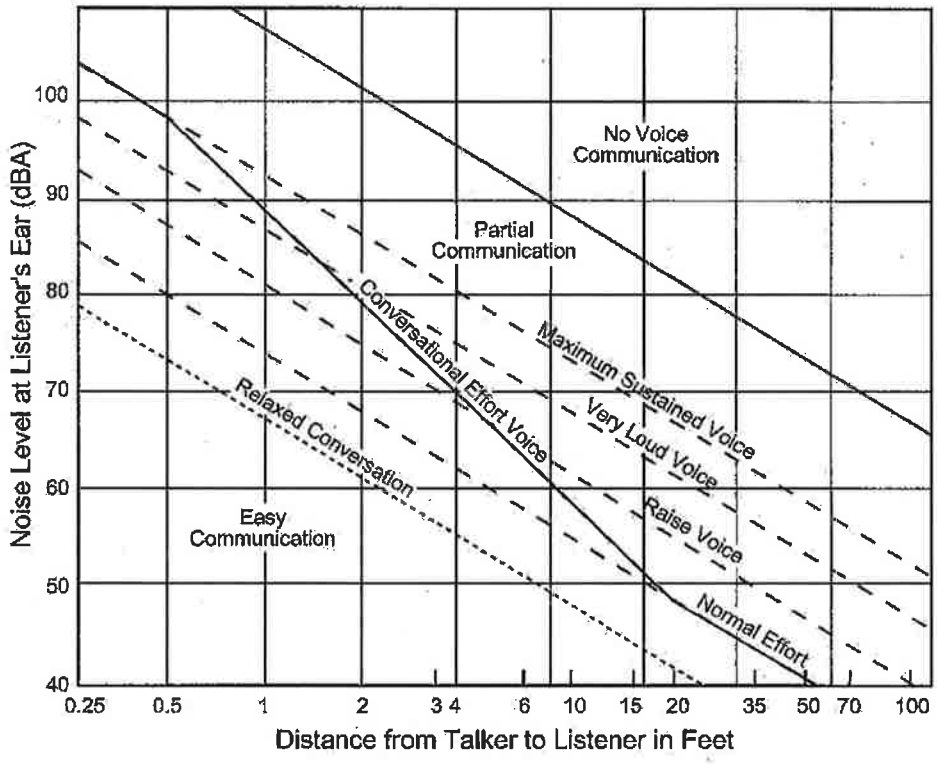
4.2 Speech Interference

Speech interference occurs when speech is masked by other sounds occurring simultaneously. Because most of the acoustic energy associated with speech is in the frequency range of 250-5000 Hz, speech intelligibility is often adversely affected by noise (Harris 1991, WHO 2000). The effects of noise are compounded by other factors also influencing speech intelligibility, including speech pronunciation,

distance between speaker and listener, hearing acuity, level of attention, reverberation characteristics of the listening environment, and sound characteristics of the interfering noise. As the sound pressure level of an interfering noise increases, the speaker compensates by also increasing the volume of his voice. This places additional strain on the listener as well. Noise may mask not only speech but can be of even greater concern when it masks other acoustical signals important for daily life, such as door bells, telephones, alarm clocks, fire alarms, music, and other warning signals (Edworthy 1996).

The American National Standards Institute (ANSI) Standard S3.5, "Methods for Calculation of the Speech Intelligibility Index," includes a Speech Interference Level (SIL) metric which relates speech interference caused by relatively constant noise sources (ANSI 1997). However, because this standard is based on steady-state noise, its applicability to evaluation of nonsteady-state noise events, such as aircraft and railroad grade-crossing horn noise, is limited. Use of the SIL metric would require careful judgement regarding the variability of level, duration, direction, and frequency spectrum of the noise source (FICAN 1992).

Figure 3 shows the voice effort required at differing distances and sound levels and can be used as a very general guide to the effects of noise and speech interference.



Source: U.S. EPA 1973

Figure 3. Noise level effects on speech interference (EPA 1973).

4.3 Effects of Noise on Performance

In both laboratory and workforce environments, noise adversely affects cognitive task performance (WHO 2000). The level of noise necessary to produce adverse effects does depend greatly on the type of task, however. For example, transportation noise may be more disruptive during reading activities as compared to recreational activities. In addition to type of activity, the frequency content and temporal characteristics may also play a part in producing adverse effects. High frequency sound is typically more disruptive than low-frequency; and intermittent sounds are generally more disruptive than continuous sounds of equivalent acoustic energy (Suter 1991).

Other studies suggest that noise slows rehearsal in memory, acting as a distraction stimulus, thus, increasing the need for focusing (Mohindra 1983). People whose performance strategies are already limited for other reasons (e.g., through high anxiety), and who are faced with multiple tasks may be more vulnerable to the masking and distracting effects of nosie (Stansfeld 1997).

Of considerable research interest are the effects of transportation noise on children. Noise has been shown to affect children's cognitive and motivational parameters (Bronzaft 1975, Bronzaft 1981, Cohen 1980, Evans 2001, Evans 1998, Evans 1995, Evans 1993, Haines 1998, Hygge 1998, Kohut 2001, Stansfeld 1997). Among the cognitive effects, reading, attention, problem solving, and memory are most affected by noise. Most of the studies reviewed have found the uncontrollability of noise, rather than the intensity of the noise, appears to be the most critical variable.

4.4 Interference with Recreational Activities

Noise affects people at home and at work, but also during leisure. It is difficult, however, to estimate the magnitude of these effects, especially when considering other noise sources present during leisure activities. High noise exposure sources/situations which people expose themselves to voluntarily, include music (e.g., concerts, clubs, and headphones), loud games, and sports (e.g., hunting, stadiums, and motor sports), and toys (e.g., cap pistols, rattles, and arcades). The trend in noise levels for these kinds of activities has been upward (Axelsson 1996, Suter 1991).

For those leisure activities where people purposely avoid noise or expect quiet, including libraries, churches, and remote areas of parks, the intrusion of transportation noise is of greater concern. Several studies document the development of relationships between noise exposure (or change in noise exposure) and human response. These relationships are being used as tools to assist the National Park Service in reducing the impacts of aircraft overflight sound levels on visitors to the National Parks (NPS 1995, Anderson 1993, Fleming 1998). Impacts caused by aircraft overflights in the National Parks are of two types: impacts on natural quiet and impacts on visitor enjoyment. The findings of a study performed in Bryce Canyon National Park indicate that approximately one-quarter of the survey respondents expressed annoyance as a result of aircraft overflight noise. It should be noted, however, that respondents also reported a number of other factors besides aircraft noise as annoyances, e.g., crowds/other people, trail conditions, weather, lack of restroom facilities (Fleming 1998).

5. PHYSIOLOGICAL EFFECTS

This section discusses the physiological effects of environmental noise. Physiological effects include hearing impairment, sleep disturbance, and startle and defense reactions leading to potential increase of blood pressure.

5.1 Hearing Impairment/Loss

A report by the Federal Interagency Committee on Aviation Noise documents some of the direct, indirect, short-term, and long-term health and welfare effects of airport noise (FICAN 1992). Hearing impairment is typically defined as an increase in the threshold of hearing. Changes in hearing threshold level, the lowest level a sound signal of a specific frequency can be detected by the ear, of less than 5 dB are not generally considered noticeable. A noise-induced permanent threshold shift of 5 dB or more is considered significant by the EPA (EPA 1974). Hearing impairment due to noise is typically detected beginning at the frequencies 4000 Hertz (Hz) or above. The impairment may also be accompanied by tinnitus (ringing in the ears).

In 1995, at the World Health Assembly, it was estimated that there are 120 million people with disabling hearing difficulties worldwide (Smith 1998). Men and women are equally at risk of noise-induced hearing impairment (ISO 1990, WHO 2000). Prolonged exposure to high levels of noise exposure can cause hearing impairment, though most cases were found to be related to occupational noise exposure. Guidance for determining noise-induced hearing impairment due to occupational noise exposure can be found in the International Organization for Standardization's (ISO), "Acoustics - Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment" (ISO 1996).

A review of studies by the World Health Organization shows hearing impairment/loss for cases outside of occupational noise exposure. Deterioration of the hearing capability is more often shown to be caused by diseases, head trauma, hereditary links, and normal aging. Environmental noise exposures due to noisy activities, especially leisure-time activities of children and young adults, have not been as scrutinized or regulated (WHO 2000).

5.2 Sleep Disturbance.

The National Sleep Foundation reports that the most common environmental elements affecting sleep are noise, sleep surface, temperature or climate, and altitude with age and gender playing a part in determining the level of sleep disturbance caused by these factors (NSF). It is estimated that only 10 to 20 percent of the reported cases of sleep disturbance are for reasons relating to transportation noise (Reyner & Horne 1995). While most experimental research studies have been performed in controlled environments, some field studies of sleep disturbance conducted within people's homes have been performed (ANSI 2000). The majority of transportation-noise, sleep-disturbance research has focused on aircraft noise (Basner 2001) - few are conducted for highway or railway noise (Griefahn 1996, Griefahn

1998). Most studies monitor awakening by a verbal response, a button push, and/or various physiological signals, including polysomnographic measurements (EEG, EOG, EMG), the electrocardiogram, finger pulse amplitude, respiration, and actigraphy. In addition, urine samples were examined for stress indicators, including cortisol, epinephrine, and norepinephrine.

Of greater concern are the possible secondary effects, i.e., after effects, of sleep disturbance. These effects may include reduced perceived sleep quality, increased fatigue, depressed mood or well being, and decreased performance (Carter 1996, Ohrstrom 1993, Passchier-Vermeer 1993, Pearsons 1995, Pearsons 1998).

Based on the literature reviewed in a USAF-sponsored study of sleep disturbance (Pearsons 1989), no specific long-term health effects have been clearly linked with sleep disturbance (FICAN 1992). It has also been observed that habituation typically occurs over time when similar sound exposures occur across nights (Griefahn 1977, LeVere 1975, NSF, Ohrstrom 1988, Stansfeld 1997, TRB 1997). Comparison of results of laboratory and field studies shows that habituation to night-time noises occurs to a large extent with respect to awakening, but not, or to a much lesser extent, with respect to changes in sleep stage and heart rate (HCN 1994). Regardless however, sleep disturbance is indisputably undesirable and, thus, still considered a noise impact.

Scientists agree that more studies are needed to confirm or disaffirm what relatively little is known about sleep (NSF). The USAF review also included the need for additional data to satisfy the following areas of concern:

- large discrepancies between laboratory and field studies;
- highly variable and incomplete data bases;
- lack of appropriate field studies;
- the studies' methodologies; and
- lack of consideration of non-acoustic effects.

In addition, it has been found that people do intervene to a certain extent in their own living and working environment, e.g., by moving to more quiet surroundings or by changing their job. This may result in a selection in which people who are "noise-proof" will remain in noise situations and those who are not will leave the situation (HCN 1994).

The findings of several field studies over the past twenty-five years have been compiled and analyzed using meta-analytic techniques to derive a prediction model of noise-induced awakenings for transportation noise sources. The prediction model is based on a curvilinear power-function fit through the data points defining the percent of study participants awakened at given indoor single-event noise levels, or sound exposure levels (L_{AE}), of aircraft (Finegold 2001). This relationship is shown in Figure 3 below and in the following equation:

$$\%\,Awakening = 0.58 + (4.30 \times 10^{-8}) \times L_{AE}^{4.11}$$

In considering Figure 4, it should be noted that the time interval between two noise events also has been found to have an important influence on the probability of obtaining a response (WHO 2000, Griefahn 1977, Ohrstrom 1990).

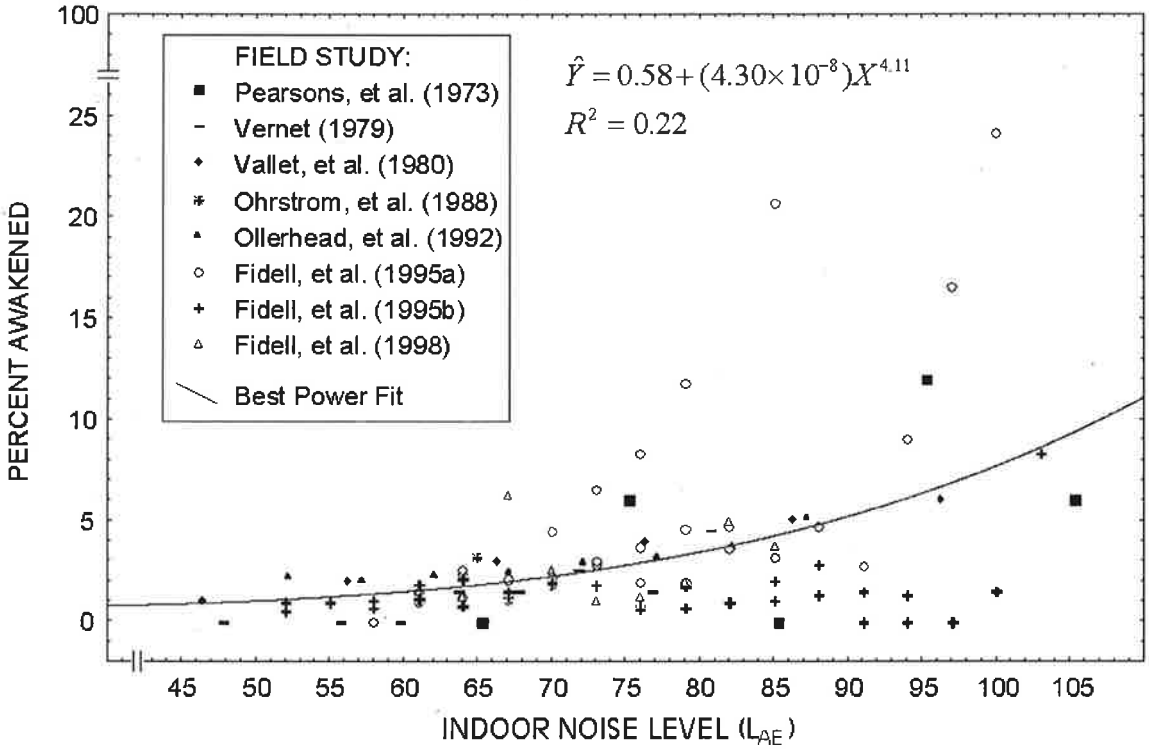


Figure 4. Sleep disturbance as a function of single-event noise exposure levels (Finegold 2001).

5.3 Muscular Effects of Noise

Muscular reflex responses to noise involves obvious movements (startle response) of voluntary muscle groups and less obvious electrical activity found in muscle tension. The magnitude of a startle response is related to the magnitude of the noise stimulus and rate of onset (May 1971, Thackray 1973). Most research concerning human reaction to impulsive noise comes from studies of the effects of gunfire with some limited occupational noise exposure data from industrial situations. While a reduction in response, or habituation, is typical with continuation or repetition of the stimulus, the sudden muscular response due to noise could potentially interfere with critical motor tasks at critical moments. In addition, short and long term muscular tension has many negative cardiovascular implications (Harris 1991, Stansfeld 1997).

A hazard exists when impulsive noises exceed a peak sound pressure level of 140 dB for more than 5 continuous milliseconds (ms). Higher maximum levels may be tolerable for durations of less than 5 ms. Peak sound pressure levels in excess of 165 dB even for minuscule durations, are likely to cause acute cochlear damage (WHO 2000).

5.4 Cardiovascular Effects of Noise

Epidemiological and laboratory studies involving workers exposed to occupational noise and general populations (including children) living in noisy areas around airports, industries, and noisy streets, indicate that noise may have both temporary and permanent impacts on physiological functions of people (WHO 2000).

Research has correlated exposure to environmental noise with physiological changes in blood pressure, sleep, digestion and other stress-related disorders (Parrot 1992, Passchier-Vermeer 1993, Petiot 1992). Low frequency noise (0 to 20 Hz) can be directly absorbed through the surface of the body and can excite sense organs other than the ears causing disturbances to the nervous system, equilibrium, and sight (Newman 1985). These changes relate chronic noise exposure and some elevated neuroendocrine and cardiovascular measures. Certain acute noise exposures can activate the autonomic and hormonal systems. Laboratory experiments and field research have shown that if noise exposure is temporary, the physiological system usually returns to normal after an appropriate recovery period (WHO 2000).

Prolonged exposure, however, may cause more serious cardiovascular and hormonal responses, including increases in heart rate and peripheral vascular resistance, changes in blood pressure, blood viscosity, and blood lipids, and shifts in electrolyte balance. Susceptible individuals may develop permanent effects, such as coronary heart disease (Bronzaft 1997, Cohen 1980, Evans 1995, Ising 1997, Schwarze 1988).

The overall conclusion is that cardiovascular effects are associated with long-term exposure to daily equivalent sound levels greater than 65 dB. These exposures have been implicated in the development and exacerbation of a variety of health problems, ranging from hypertension to psychosis. However, most of these findings have been criticized by the research community because the associations are usually weak, only the average risk is considered, and sensitive subgroups have not been sufficiently characterized (WHO 2000, Suter 1991, Thompson 1981, Thompson 1996). More research into the long-term effects of noise is required before unequivocal guideline values can be established.

5.5 Mental Health Effects of Noise

Noise, particularly transportation noise, is not believed to be a direct cause of mental illness, but it is assumed that it accelerates and intensifies the development of latent mental disorder (WHO 2000). Studies on the adverse effects of transportation noise on mental health include symptoms such as anxiety, emotional stress, nervousness, nausea, headaches, instability, argumentativeness, changes in mood, increase in social conflicts, as well as general psychiatric disorders, including neurosis, psychosis, and hysteria.

There is also only very limited evidence that relatively more admissions to psychiatric hospitals might occur from areas with very noisy conditions, primarily due to high levels of aircraft noise, than from more quiet surroundings (HCN 1994). Most studies linking transportation noise, primarily aircraft noise, to mental disorders are inconclusive and have been criticized for methodology and response bias (Halpern 1995, Schwarze 1988, Thompson 1996, WHO 2000). In many cases, it should also be noted that noise-sensitive individuals are likely to be sensitive to other non-transportation-related sounds in their environment as well (Anderson 1971, Broadbent 1979, Ohrstrom 1988, Thomas, 1982, Weinstein, 1978).

6. EFFECTS OF NOISE ON WILDLIFE

There has been extensive research into the effects of environmental noise, particularly transportation noise, on wildlife, especially since the establishment of the Endangered Species Act in 1973. There are over 200 published and unpublished reports which include many species that vary in scope from long-term research studies to government investigations to environmental agency citings (Gladwin 1987, NPS 1994). This section overviews the research on the effects of transportation noise on wildlife.

The majority of transportation-noise research, primarily tied to aircraft noise, has shown that certain noise intrusions can cause some effects on wildlife - however, less than originally expected. In general, some research indicates that extensive or repeated loud noise intrusions interfere with communication and breeding. By far, two of the largest, most comprehensive reviews of current and past research are contained in:

- The National Parks Service's Report to Congress (NPS 1994)
- The National Ecology Research Center's report for the U.S. Department of the Interior (Gladwin 1987)

Noise impacts on wildlife cover a wide range of effects. The following categories of impacts are adapted from work performed by Oak Ridge National Laboratory, the U.S. Army Construction Engineering Research Laboratory, and the U.S. Environmental Protection Agency (Efroymson 2000, NPS 1994). They represent a practical synopsis for judging potential impacts on wildlife due to transportation noise:

- Negligible Impacts
  - No species of concern are present and no or minor impacts on any species are expected.
  - Minor impacts that do occur have no secondary (long-term or population effects).

- Low/Minor Impacts
  - Non-breeding animals of concern are present in low numbers.
  - Habitat is not critical for survival and not limited to the noise intrusion area.
  - Occasional fright responses are expected, but without interference with feeding, reproduction, or other activities necessary for survival.
  - No serious concerns are expressed by State or Federal fish and wildlife officials.

- Moderate Impacts
  - Breeding animals of concern are present, and/or animals are present during particularly vulnerable life-stages, such as migration or winter.
  - Mortality or interference with activities necessary to survival is expected on an occasional basis.
  - Mortality or interference are not expected to threaten the continued existence of the species.
  - State and Federal officials express some concern.

- High/Major Impacts
  - Breeding individuals are present in relatively high numbers, and/or animals are present during particularly vulnerable life-stages.
  - Habitat (subjected to noise intrusions) has a history of use by the species during critical periods, and this habitat is somewhat limited to the noise impacted area; animals cannot go elsewhere to avoid impacts (animals can rarely relocate except temporarily).
  - Mortality or other effects (injury, physiological stress, effects on reproduction and young raising) are expected on a regular basis.
  - These effects could threaten the continued survival of the species.
  - State and Federal wildlife officials express serious concern.

While extensive research has been conducted on the effects of noise on wildlife - differences between study species and methodologies make a clear, quantitative definition of noise impact on wildlife difficult. Most studies pertain to specific species and result in primarily broad generalizations and observations of the behavioral responses (Arner 1999, Lelis 1999, Levy 1999, Parks 2000, Presley 2000, Reijnen 1995, Robertson 2000). With regard to transportation-related noise, most of these studies have been based on aircraft noise (DND 1998, Wells 2000). Responses of wildlife to aircraft noise vary from almost no reaction to virtually no tolerance of the sound with the question of the adaptability of wildlife to certain sounds remaining largely unanswered (Newman 1985). Further research is necessary to better correlate actual noise levels, critical frequency ranges, types of noise sources, and other variables to the behavioral responses of wildlife.

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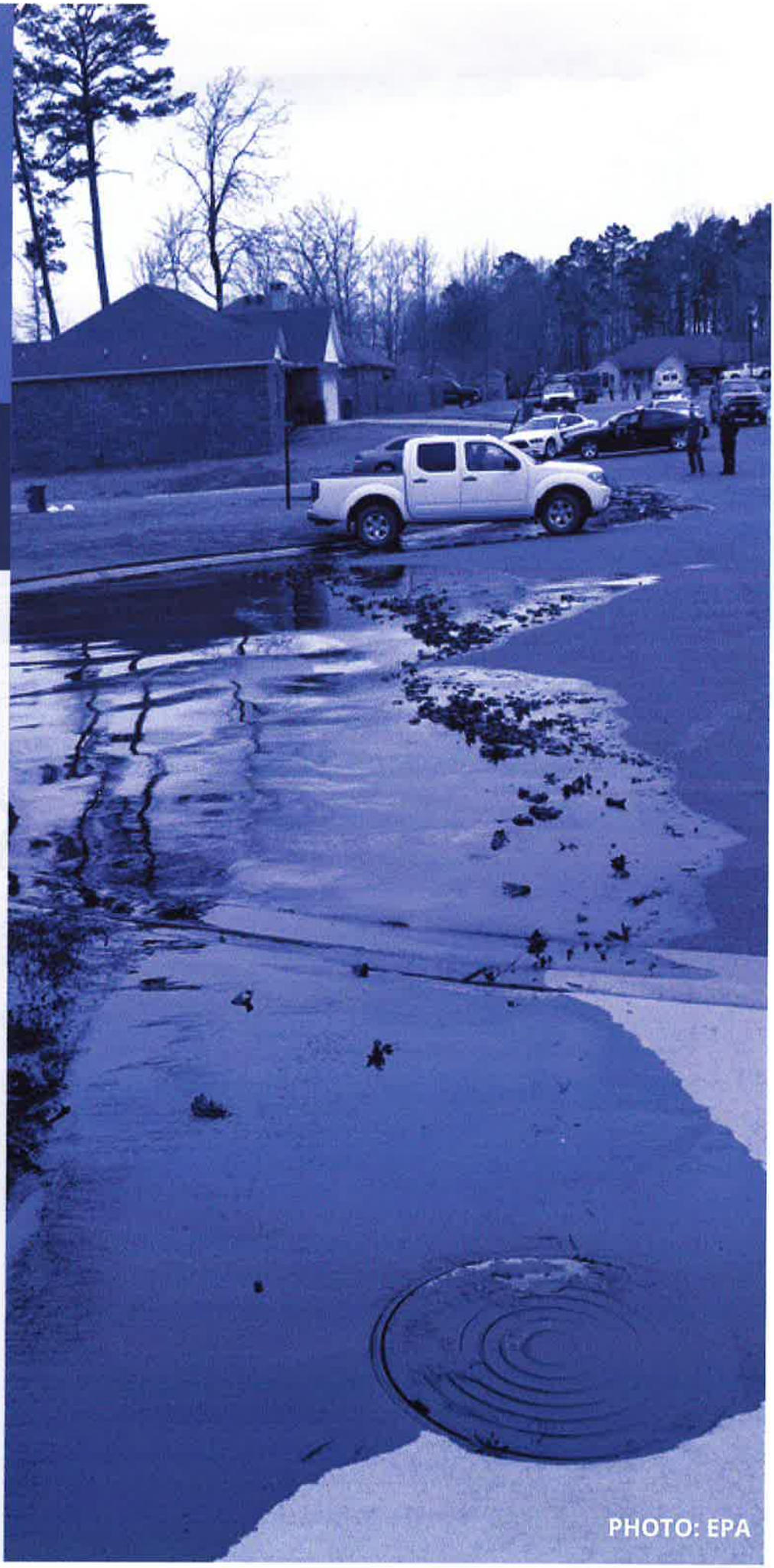
POISONS IN THE PIPELINE

TESTS FIND TOXIC STEW IN OIL SPILL

ENVIRONMENTAL WORKING GROUP

JUNE 2013



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The mission of the Environmental Working Group (EWG) is to use the power of public information to protect public health and the environment. EWG is a 501(c)(3) non-profit organization, founded in 1993 by Ken Cook and Richard Wiles.

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POISONS IN THE PIPELINE

Tests Find Toxic Stew in Oil Spill

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AS THE NATION DEBATES WHETHER THE PROPOSED KEYSTONE XL PIPELINE SHOULD BE SHELVED OR MOVE FORWARD, A KEY QUESTION REMAINS: WHAT KINDS OF TOXIC CHEMICALS WILL THE PIPELINE BE TRANSPORTING THROUGH THE AMERICAN HEARTLAND? GIVEN THE SIGNIFICANT POTENTIAL FOR LEAKS AND SPILLS, AND THE FACT THAT THE PIPELINE WILL TRAVERSE CRITICAL DRINKING WATER SOURCES, THE PUBLIC HAS A RIGHT TO KNOW.

In an attempt to answer this question, clean energy advocate and philanthropist Tom Steyer sought the help of a community resident to collect a sample of spilled tar sands crude oil from the March 29 ExxonMobil pipeline rupture in Mayflower, Arkansas. The sample, taken on April 11, was provided to Environmental Working Group roughly two months later to test for the presence of toxic chemicals.

EWG commissioned an independent laboratory analysis, which found that – despite the likelihood of significant off-gassing of volatile chemicals prior to the sample's delivery – the spilled tar sands oil contained seven highly toxic compounds, including lead, benzene and others that can cause cancer and developmental problems. EWG's findings raise new questions about the potential health and safety risks of the proposed Keystone XL pipeline, which would carry 830,000 barrels of tar sands oil daily from Canada across the United States. *(see Table 1 on page 4)*

The seven contaminants found in EWG's testing may be only the beginning, however. Crude oil is known to contain many volatile and semi-volatile chemicals that rapidly disperse into air. Community air sampling conducted directly after the Mayflower spill confirmed the presence of more than 25 toxic chemicals, including the hazardous air pollutants benzene, toluene, ethylbenzene, xylene, hexane and cumene (GCM 2013).

"I couldn't breathe. My throat and nose and eyes were burning really bad.... I could smell that horrible smell. I got really scared."

– Mayflower, Ark. resident

Eight students were sent home from school after the spill because they were vomiting and complaining of headaches (McAllister 2013). Other Mayflower residents complained of nausea, headaches, breathing problems, respiratory problems and

TABLE I

TAR SANDS OIL SPILLED IN MAYFLOWER, ARK. CONTAINED 7 TOXIC CHEMICALS

Chemical name	Potential health effects
Benzene	Known human carcinogen associated with leukemia; exposure during pregnancy may cause birth defects; can also have toxic effects on blood and bone marrow, the nervous system and immune system. (IARC 1982; 1987; 2012; ATSDR 2007a; Lupo 2011)
Toluene	Toxic to the nervous system and kidneys; exposure to high levels during pregnancy may lead to birth defects and other health problems. (ATSDR 2000)
Ethylbenzene	Listed as a carcinogen in California's Proposition 65 inventory of toxic chemicals; animal studies suggest that it may also be toxic to the nervous system, cause developmental harm and damage hearing and the kidneys. (ATSDR 2010; OEHHA 2013)
1,2,4-Trimethylbenzene	A volatile chemical that can irritate the skin and respiratory system; exposure to high levels may cause adverse central nervous system effects such as drowsiness and headache; animal experiments show that it can have toxic effects on development. (EPA 1994, Saillenfait 2005)
Xylenes	Toxic to the nervous system; can cause respiratory irritation; some human studies indicate that exposure may also affect the kidneys and liver. (ATSDR 2007b)
Chromium	A common form (called chromium-6 or hexavalent chromium) is a known human carcinogen and has been shown to cause birth defects and developmental defects in animals. (ATSDR 2012)
Lead	Highly toxic to the developing nervous system; can cause serious and permanent damage to the unborn, including effects on cognition and growth; can affect almost every organ and system of the body and has no known safe level. (ATSDR 2007c)

other symptoms after breathing the fumes; these complaints persisted for days and even weeks after the spill (McAllister 2013, GCM 2013, Peeples 2013). One community resident who lives just outside the evacuation zone described waking up in the middle of the night a couple of days after the spill: "I couldn't breathe. My throat and nose and eyes were burning really bad... I could smell that horrible smell. I got really scared" (Peeples 2013).

The citizen-collected sample provided to EWG was not gathered in a manner that could adequately capture the volatile and semi-volatile chemicals that likely off-gassed from the spilled oil into the surrounding air. Rather than being placed in a special, hermetically sealed container of the kind typically used to transport volatile-containing samples to a lab, the Mayflower oil sample was placed into a plastic food container with a piece of plastic wrap over the top. It was later transferred to an ordinary screw-top jar, which was then given to EWG. In addition, the sample was kept at room temperature rather than refrigerated. Such handling is known to increase the volatilization of chemicals and can make them break down or dissipate faster. Furthermore, EWG received

the sample more than two months after the spill took place, providing significant opportunity for the crude oil to off-gas.

For these reasons, EWG's tests focused on the *presence* of toxic chemicals, not their levels.

Despite the fact that EWG's test results likely represent significant underestimates of the concentrations of volatile and semi-volatile chemicals, the level of benzene found (4.5 parts per million) is still cause for significant concern. This volatile compound readily dissolves in water, and the estimated 3,500-to-5,000 barrels of oil spilled in Arkansas contained enough benzene to contaminate 132-to-188 million gallons of drinking water at levels above the 5 parts per billion limit allowed under federal drinking water standards (PHMSA 2013). Long-term exposure to benzene above this limit increases the risk of cancer and blood problems (EPA 2013a).

TABLE 2

TWO MONTHS AFTER THE MAYFLOWER DISASTER, SPILLED TAR SANDS OIL STILL CONTAINED DETECTABLE LEVELS OF MANY TOXIC CHEMICALS

Chemical name	Concentration* (parts per million)
Benzene	4.5
Toluene	23
Ethylbenzene	7.4
1,2,4-Trimethylbenzene	30
m,p-Xylene	41
o-Xylene	15
Chromium	0.29
Lead	0.31

*Levels are likely to be underestimates due to non-standard sampling methods used by local resident.

The Arkansas spill sample also contained detectable levels of lead, which is toxic at such low levels that the Environmental Protection Agency has set the drinking water goal at zero. A lead-contaminated pipeline spill that came into contact with drinking water supplies would likely pose unacceptable public health risks.

SECRET CHEMICALS IN THE PIPELINE

Because of industry trade secrets, EWG was able to test for only a limited number of potential contaminants in the Mayflower crude oil sample. This constraint also makes it difficult for the federal government to assess the safety of transporting tar sands oil through the Keystone XL pipeline. This could have significant implications for communities along the proposed pipeline route, should there be additional spills into communities, farm fields and waterways. Given that significant pipeline spills happen every three days on average in the United States, it will be only a matter of time before spills take place along the Keystone pipeline route if it is constructed (McAllister 2013).

The pipeline that burst in Arkansas was carrying the same type of Canadian tar sands oil that would flow through Keystone XL, technically termed "bitumen." Because bitumen typically occurs in solid or semi-solid form, it must be diluted with significant quantities of a chemical cocktail before it can be pumped through a pipeline. The resulting mixture is called diluted bitumen or "dilbit." The exact composition of dilbit is anyone's guess since the tar sands industry claims that the identity of the diluting chemicals is a trade secret and does not disclose that information.

"An analysis of potential diluents is important to establish the potential health and environmental impacts of any spilled oil..."

– Environmental Protection Agency

The lack of such basic information was one reason the Environmental Protection Agency gave a rating of "inadequate" to the State Department's draft Environmental Impact Statement on the pipeline proposal. This rating indicates that the EPA did not think the draft document adequately assessed the potentially significant environmental risks of building and operating the pipeline (EPA 2011).

The EPA noted that "an analysis of potential diluents is important to establish the potential health and environmental impacts of any spilled oil, and responder/worker safety, and to develop response strategies" (EPA 2011). Equally importantly, the public cannot make an informed judgment on the safety of the Keystone XL pipeline if it is in the dark about what kinds of toxic chemicals will flow through it.

It is clear, however, that dilbit spills pose a unique and serious environmental and public health threat. The EPA recently emphasized that spills of diluted bitumen "require different response actions or equipment from actions for conventional oil spills" (EPA 2013b).

According to Cornell University soil scientist Corey Ptak, tar sands oil is particularly challenging to clean up and takes a long time to break down in the environment. "Heavy crude oil and crude bitumen from oil sands contain a higher percentage of long-chain hydrocarbons, asphaltenes and resins," Ptak told EWG. "These compounds are highly resistant to microbial actions, making these forms of crude more difficult to degrade than lighter crude oils. As a result, crudes from oil sands will require different management plans and potentially longer time frames to effectively remediate."

A July, 2010 pipeline spill in Marshall, Mich., provided ample evidence of how challenging it is to clean up spills of tar sands oil. In that incident, Enbridge Energy Partners reported that a rupture of a 30-inch diameter oil pipeline had released 843,000 gallons of dilbit into a nearby creek and ultimately into the Kalamazoo River. (The Keystone XL pipeline would be 36 inches in diameter, which would make a similar spill even more devastating.) Heavy oil from the Enbridge spill sank to the bottom of the river and mixed with sediment and organic matter, making the recovery process extremely difficult.

$1 billion – estimated cost of cleaning up Enbridge tar sands oil spill

After almost three years of cleanup efforts, the EPA recently determined that it will be necessary to dredge of the bottom of the Kalamazoo River to "protect public health and the welfare of the environment (EPA 2013b)." A document filed by Enbridge with the US Securities and Exchange Commission (SEC) in March said that as a result of EPA's final order, the estimated cost of cleaning up the spill had risen to a staggering $1 billion (SEC, 2013).

CONCLUSION

EWG's testing of the Arkansas spill sample highlights the risks that the Keystone XL pipeline would pose to water resources, especially in light the EPA's earlier conclusion that pipeline spills are a "very real concern" (EPA 2011). Pipeline ruptures are common: The industry's record makes clear that it's a matter of when, not if. Spills might be especially likely with tar sands oil because it takes higher temperatures and pressures to keep bitumen flowing

than with conventional crude oil.

How many drinking water supplies or acres of farmland might be devastated by pipeline ruptures for months, years or perhaps forever if Keystone XL moves forward?

Over its planned route of 1,179 miles from Alberta, Canada, to Nebraska, the Keystone XL pipeline would traverse a number of critical water resources, including aquifers that provide drinking water to millions who live in the High Plains area of the United States. This includes the Ogallala aquifer, the nation's largest underground drinking water source (Heineman 2013). The proposed pipeline route also crosses 65 rivers, streams and other water bodies designated for drinking water or for recreation, fishing and agricultural uses (USDS 2013).

The Arkansas and Michigan pipeline ruptures are clear warnings of the dangers facing America's waters, soils and homes if the Keystone XL pipeline is approved, as well as a reality check on the incredible difficulty and expense of cleaning up after spills of tar sands oil. Given the industry's track record, it is essentially inevitable that such spills will take place sooner or later in America's heartland if Keystone XL moves forward.

EWG's findings raise crucial questions: How many drinking water supplies or acres of farmland might be devastated by pipeline ruptures for months, years, or perhaps forever? Would people want to buy food from a farm that had been contaminated with crude oil containing benzene, lead and any number of other toxic chemicals? These are difficult questions that deserve serious consideration. This is especially true given that – as the Mayflower rupture demonstrates – pipelines do not fail gracefully. They fail catastrophically.

RECOMMENDATIONS

At a bare minimum:

- Oil and gas companies must be required to publicly disclose the names and amounts of all chemicals used to dilute tar sands oil.
- Oil and gas companies must be required to submit samples of the diluted for thorough independent testing to assess the concentrations of toxic chemical pollutants.
- The State Department must revise its Environmental Impact Statement, which dismissed concerns about the pipeline without taking into account the highly toxic nature of what would flow through the pipe, and address the risks of the chemicals that will be used to dilute tar sands oil flowing through Keystone XL.

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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

APR 2 2 2013

ASSISTANT ADMINISTRATOR
FOR ENFORCEMENT AND
COMPLIANCE ASSURANCE

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Economic, Energy and Business Affairs
U.S. Department of State
Washington, DC 20520

Dr. Kerri-Ann Jones
Assistant Secretary
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U.S. Department of State
Washington, DC 20520

Dear Mr. Fernandez and Dr. Jones:

In accordance with our authorities under the National Environmental Policy Act (NEPA) and Section 309 of the Clean Air Act, EPA has reviewed the Department of State's draft Supplemental Environmental Impact Statement (DSEIS) for a Presidential Permit application by TransCanada Keystone Pipeline, LP (TransCanada) to construct and operate the Keystone XL Project (Project). This DSEIS builds on the Department of State's August 2011 Final EIS, and includes information regarding a new proposed route in Nebraska.

NEPA serves an important role in the decision making process for federal actions that may have environmental effects. Through the NEPA process, federal agencies disclose and analyze the potential impacts of a proposed action and reasonable alternatives, as well as measures that could mitigate any potential harmful effects. NEPA brings transparency to the federal decision making process, requiring that other federal, state, tribal and local agencies, as well as citizens, are given a meaningful opportunity to provide comments, helping to ensure federal decisions are better informed.

EPA believes this DSEIS strengthens the analysis presented to date in the NEPA process. While we appreciate this effort, we also have several recommendations for improving the analysis and considering additional mitigation as you move forward to complete the NEPA process.

Greenhouse Gas Emissions

We commend the Department of State's efforts to estimate the lifecycle greenhouse gas (GHG) emissions associated with oil sands development and the proposed Project, to analyze the effect of the Project on Canadian oil sands production and to consider measures to reduce GHG emissions. As recognized by the DSEIS, oil sands crude is significantly more GHG intensive than other crudes, and therefore has potentially large climate impacts. The DSEIS reports that lifecycle GHG emissions from oil sands crude could be 81% greater than emissions from the average crude refined in the U.S. in 2005 on a well-to-tank basis, and 17% greater on a well-to-wheels basis.[1] This difference may be even greater depending on the assumptions made.[2] The incremental emissions from oil sands crude transported by the Project would therefore be 18.7 million metric tons CO_2-e (carbon dioxide equivalent) per year when compared to an equal amount of U.S. average crudes, based on the Project's full capacity of 830,000 barrels of oil sands crude per day.[3] To place this difference in context, we recommend using monetized estimates of the social cost of the GHG emissions from a barrel of oil sands crude compared to average U.S. crude. If GHG intensity of oil sands crude is not reduced, over a 50 year period the additional CO_2-e from oil sands crude transported by the pipeline could be as much as 935 million metric tons. It is this difference in GHG intensity - between oil sands and other crudes - that is a major focus of the public debate about the climate impacts of oil sands crude.

Although the DSEIS describes the GHG intensity of oil sands crude, the DSEIS nevertheless concludes that regardless of whether the Project permit is approved, projected oil sands production will remain substantially unchanged. This conclusion is based on an analysis of crude oil markets and projections of oil sands crude development, including the potential for other means of transport to bring oil sands crude to market. One of the alternative transport possibilities discussed in the DSEIS is the potential construction of other pipelines. As part of this discussion, the DSEIS appropriately recognizes that there is uncertainty about when, if ever, additional pipelines will be built. In light of these uncertainties, the DSEIS examines options for transporting oil sands crude by rail, and concludes that scaling up transport by rail is logistically and economically feasible, and that market forces will result in additional rail transport of oil sands crude if the Project is not built. It is this finding that supports the DSEIS' overall conclusion that approval of the permit will not by itself substantially affect GHG emissions or contribute to climate change.

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[1] DSEIS, Table 4.15-22 "GHG Emissions for Producing Gasoline from Different Crude Sources from NETL 2009 and Estimates of the Impact of Key Assumptions on the Oil Sands - U.S. Average Differential." In addition to lifecycle emissions estimates from the Department of Energy's National Energy Technology Laboratory (NETL) study, the DSEIS also provides estimates from other analyses. See discussion in DSEIS section 4.15.

[2] DSEIS, p. 4.15-106, "Adjusting the NETL results to include other product emissions could increase the differential in incremental emissions from WCSB oil sands compared to the 2005 U.S. average crude oils by roughly 30 percent."

[3] DSEIS p. 4.15-105

The market analysis and the conclusion that oil sands crude will find a way to market with or without the Project is the central finding that supports the DSEIS's conclusions regarding the Project's potential GHG emissions impacts. Because the market analysis is so central to this key conclusion, we think it is important that it be as complete and accurate as possible. We note that the discussion in the DSEIS regarding energy markets, while informative, is not based on an updated energy-economic modeling effort. The DSEIS includes a discussion of rail logistics and the potential growth of rail as a transport option, however we recommend that the Final EIS provide a more careful review of the market analysis and rail transport options. This analysis should include further investigation of rail capacity and costs, recognizing the potential for much higher per barrel rail shipment costs than presented in the DSEIS. This analysis should consider how the level and pace of oil sands crude production might be affected by higher transportation costs and the potential for congestion impacts to slow rail transport of crude.

In its discussion of practicable options for mitigating GHG emissions, the DSEIS outlines ongoing efforts by the government of Alberta to reduce the GHG emissions associated with development of oil sands crude in Alberta. EPA recommends that the Final EIS complement this discussion with an exploration of specific ways that the U.S. might work with Canada to promote further efforts to reduce GHG emissions associated with the production of oil sands crude, including a joint focus on carbon capture and storage projects and research, as well as ways to improve energy efficiency associated with extraction technologies. With regard to the estimated GHG emissions from construction and operation of the proposed Project - primarily emissions associated with electrical generation for the pumping stations - we recommend that the Department of State explore specific commitments that TransCanada might make to implement the mitigation measures recommended in the DSEIS. This would complement the significant efforts already made to reduce the risk of spills and ensure community safety. Specifically, we recommend a focus on pumping station energy efficiency and use of renewable energy, as well as investment in other carbon mitigation options.

Pipeline Safety

We have learned from the 2010 Enbridge spill of oil sands crude in Michigan that spills of diluted bitumen (dilbit)[4] may require different response actions or equipment from response actions for conventional oil spills. These spills can also have different impacts than spills of conventional oil. We recommend that these differences be more fully addressed in the Final EIS, especially as they relate to the fate and transport of the oil and the remediation that will be required. The Enbridge spill involved a 30-inch diameter pipeline, smaller than the 36-inch diameter pipeline for proposed Project, and 20,000 barrels of oil sands crude were released. In that spill, oil sands crude sank to the bottom of the Kalamazoo River, mixing with the river bottom's sediment and organic matter, making the oil difficult to find and recover. After almost three years of recovery

[4] As noted in the DSEIS, transporting oil sands crude via pipeline requires that it be mixed with a petroleum-based product (called a diluent), such as benzene, naphtha or natural gas condensate, to make a less viscous liquid called dilbit (diluted bitumen).

efforts, EPA recently determined that dredging of bottom sediments will be required to protect public health and welfare and the environment. This determination was based in large part on demonstrations that the oil sands crude associated with the Enbridge spill will not appreciably biodegrade.[5] We recommend that the Final EIS more clearly acknowledge that in the event of a spill to water, it is possible that large portions of dilbit will sink and that submerged oil significantly changes spill response and impacts. We also recommend that the Final EIS include means to address the additional risks of releases that may be greater for spills of dilbit than other crudes. For example, in the Enbridge spill, the local health department issued voluntary evacuation notices based on the level of benzene measured in the air. Given these concerns, it is important to ensure that the future response and remediation plans will protect communities from impacts due to spills.

The DSEIS also outlines specific measures that the Department of State would require TransCanada to undertake to prevent and detect oil discharges. The measures include commissioning an independent engineering analysis to review TransCanada's risk assessment of the potential impacts from oil discharges to surface and groundwater resources, as well as TransCanada's current proposals for placing mainline valves along the pipeline route and installing leak detection equipment. The DSEIS also notes that the Department of State will obtain concurrence from both EPA and PHMSA on both the scope of the engineering analysis and decisions regarding the need for any additional mitigation measures. We recommend that the Department of State provide an opportunity for public review and comment on the scope of the analysis, and an opportunity for public comment on a draft of the analysis when it is completed. We also recommend that the Final EIS consider requiring TransCanada to establish a network of sentinel or monitoring wells along the length of the pipeline, especially in sensitive or ecologically important areas, as well as where water supply wells are located and at stream crossings to provide a practical means for early detection of leaks that are below the proposed detection limit (1.5 – 2%) of the pipeline flow rate.

In addition to prevention measures, we agree with the DSEIS's suggestion that additional mitigation measures regarding preparedness to reduce the impacts of a spill may be appropriate (DSEIS, p. 4.13-79). For example, we recommend including the following measures as permit conditions:

- Requiring that the emergency response plan, as well as contingency plans address submerged oil, as well as floating oil, including in a cold weather response;

- Requiring pre-positioned response assets, including equipment that can address submerged oil;

- Requiring spill drills and exercises that include strategies and equipment deployment to address floating and submerged oil; and

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[5] Order for Removal under Section 311(c) of the Clean Water Act, March 14, 2013 (http://www.epa.gov/enbridgespill/ar/enbridge-AR-1720.pdf)

- Requiring that emergency response and oil spill response plans be reviewed by EPA.

The DSEIS also recognizes that dissolved components of the dilbit that may be transported through the pipeline, such as benzene, polycyclic aromatic hydrocarbons (PAHs), and heavy metals, could be slowly released back to the water column for many years after a release and could cause long-term chronic toxicological impacts to organisms in both the benthic and pelagic portions of the aquatic environment. We recommend that the Final EIS more clearly recognize that this characteristic of dilbit is different from the fate and transport of oil contaminants associated with conventional crude oil and refined product spills from pipelines. For that reason we recommend that as a permit condition TransCanada be required to develop a plan for long term sampling/monitoring in the event of an oil discharge to assess and monitor these impacts as part of the spill response plan. In addition, we recommend that the permit require TransCanada to provide detailed Material Safety Data Sheets and information about the diluent and the source crude oil to support response preparations and address safety concerns in advance of any spills.

Alternative Pipeline Routes

CEQ regulations implementing NEPA require the consideration of project alternatives in an EIS, and characterize the alternatives analysis as the "heart" of an EIS.[6] The DSEIS has been significantly improved by considering more alternative routes, including an alternative that would avoid crossing the Sand Hills Region in Nebraska, reducing impacts to this fragile ecosystem. Another significant issue in the consideration of alternative routes for this Project has been the potential for impacts to the Ogallala Aquifer in the event of a spill. The alternative route in Nebraska has avoided most of the impacts to the Sand Hills Region, but still crosses the Ogallala Aquifer. The alternative laid out in the DSEIS that would avoid the Ogallala Aquifer is the I-90 Corridor Alternative, which largely follows the path of existing pipelines. The I-90 Corridor Alternative would significantly reduce the length of pipeline crossing the Northern High Plains Aquifer system, which includes the Ogallala formation, and would further reduce the potential for adverse impacts to critical groundwater resources.

We are concerned, however, that the DSEIS does not provide a detailed analysis of the Keystone Corridor Alternative routes, which would parallel the existing Keystone Pipeline and likely further reduce potential environmental impacts to groundwater resources. By determining that these routes are not reasonable, the DSEIS does not provide an analysis of their potential impacts sufficient to enable a meaningful comparison to the proposed route and other alternatives. The Keystone Corridor Alternatives were determined not to be reasonable alternatives primarily on the basis that these routes are longer than the proposed Project's route, and that additional pipeline miles would be needed to connect to Bakken MarketLink project, which would allow the proposed Project to also transport crude from North Dakota and Montana. As we have indicated in the past, we believe these alternative routes could further reduce risks to

[6] 40 C.F.R. 1502.14

groundwater resources. We recommend that the Final EIS either provide more detailed information as to why these alternatives were not considered reasonable or analyze these alternatives in more detail.

Community and Environmental Justice Impacts

The DSEIS provides a comprehensive analysis of community demographics, including minority, low-income, and tribal populations, along TransCanada's proposed pipeline route. We are especially appreciative of the effort to identify and contact each of the Local Emergency Planning Committees regarding the status of their emergency response plans, and to provide that information in the DSEIS. We also commend your recognition that environmental justice communities may be more vulnerable to health impacts from a spill, and appreciate your efforts to consider communities' access to health care, including consideration of "Health Professional Shortage Areas and Medically Underserved Areas" located along the proposed pipeline route.

EPA appreciates TransCanada's commitment to conduct cleanup and restoration and to provide alternative water supplies to affected communities in the event of an oil discharge affecting not only surface waters, but also groundwater. We recommend that these commitments be clearly documented as proposed permit conditions. We believe this would give important assurances to potentially affected communities of TransCanada's responsibilities in the event of an oil discharge that affects either surface or groundwater resources.

Conclusion

Based on our review, we have rated the DSEIS as EO-2 ("Environmental Objections – Insufficient Information") (see enclosed "Summary of Rating Definitions and Follow-up Actions").

We look forward to continuing to work with you and to provide assistance as you prepare the Final EIS. We also look forward to working with you as you determine whether approving the proposed project serves the national interest under Executive Order 13337 "Issuance of Permits With Respect to Certain Energy-Related Facilities and Land Transportation Crossings on the International Boundaries of the United States".

Please feel free to contact me or have your staff contact Susan Bromm, Director, Office of Federal Activities, at (202) 564-5400 if you have any questions or would like to discuss our comments.

Sincerely,

Cynthia Giles

Enclosure

Summary of Rating Definitions and Follow-up Action

Environmental Impact of the Action

LO--Lack of Objections
The EPA review has not identified any potential environmental impacts requiring substantive changes to the proposal. The review may have disclosed opportunities for application of mitigation measures that could be accomplished with no more than minor changes to the proposal.

EC--Environmental Concerns
The EPA review has identified environmental impacts that should be avoided in order to fully protect the environment. Corrective measures may require changes to the preferred alternative or application of mitigation measures that can reduce the environmental impact. EPA would like to work with the lead agency to reduce these impacts.

EO--Environmental Objections
The EPA review has identified significant environmental impacts that must be avoided in order to provide adequate protection for the environment. Corrective measures may require substantial changes to the preferred alternative or consideration of some other project alternative (including the no action alternative or a new alternative). EPA intends to work with the lead agency to reduce these impacts.

EU--Environmentally Unsatisfactory
The EPA review has identified adverse environmental impacts that are of sufficient magnitude that they are unsatisfactory from the standpoint of public health or welfare or environmental quality. EPA intends to work with the lead agency to reduce these impacts. If the potentially unsatisfactory impacts are not corrected at the final EIS stage, this proposal will be recommended for referral to the CEQ.

Adequacy of the Impact Statement

Category 1--Adequate
EPA believes the draft EIS adequately sets forth the environmental impact(s) of the preferred alternative and those of the alternatives reasonably available to the project or action. No further analysis or data collection is necessary, but the reviewer may suggest the addition of clarifying language or information.

Category 2--Insufficient Information
The draft EIS does not contain sufficient information for EPA to fully assess environmental impacts that should be avoided in order to fully protect the environment, or the EPA reviewer has identified new reasonably available alternatives that are within the spectrum of alternatives analyzed in the draft EIS, which could reduce the environmental impacts of the action. The identified additional information, data, analyses, or discussion should be included in the final EIS.

Category 3--Inadequate
EPA does not believe that the draft EIS adequately assesses potentially significant environmental impacts of the action, or the EPA reviewer has identified new, reasonably available alternatives that are outside of the spectrum of alternatives analyzed in the draft EIS, which should be analyzed in order to reduce the potentially significant environmental impacts. EPA believes that the identified additional information, data, analyses, or discussions are of such a magnitude that they should have full public review at a draft stage. EPA does not believe that the draft EIS is adequate for the purposes of the NEPA and/or Section 309 review, and thus should be formally revised and made available for public comment in a supplemental or revised draft EIS. On the basis of the potential significant impacts involved, this proposal could be a candidate for referral to the CEQ.

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Heavy Oil and Natural Bitumen Resources in Geological Basins of the World

Open File-Report 2007-1084

U.S. Department of the Interior
U.S. Geological Survey

Heavy Oil and Natural Bitumen Resources in Geological Basins of the World

By Richard F. Meyer, Emil D. Attanasi, and Philip A. Freeman

Open File-Report 2007–1084

U.S. Department of the Interior
U.S. Geological Survey

U.S. Department of the Interior
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U.S. Geological Survey
Mark D. Myers, Director

U.S. Geological Survey, Reston, Virginia: 2007

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(available online at http://pubs.usgs.gov/of/2007/1084)

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Heavy Oil and Natural Bitumen Resources in Geological Basins of the World

By Richard F. Meyer, Emil D. Attanasi, and Philip A. Freeman

Abstract

Heavy oil and natural bitumen are oils set apart by their high viscosity (resistance to flow) and high density (low API gravity). These attributes reflect the invariable presence of up to 50 weight percent asphaltenes, very high molecular weight hydrocarbon molecules incorporating many heteroatoms in their lattices. Almost all heavy oil and natural bitumen are alteration products of conventional oil. Total resources of heavy oil in known accumulations are 3,396 billion barrels of original oil in place, of which 30 billion barrels are included as prospective additional oil. The total natural bitumen resource in known accumulations amounts to 5,505 billion barrels of oil originally in place, which includes 993 billion barrels as prospective additional oil. This resource is distributed in 192 basins containing heavy oil and 89 basins with natural bitumen. Of the nine basic Klemme basin types, some with subdivisions, the most prolific by far for known heavy oil and natural bitumen volumes are continental multicyclic basins, either basins on the craton margin or closed basins along convergent plate margins. The former includes 47 percent of the natural bitumen, the latter 47 percent of the heavy oil and 46 percent of the natural bitumen. Little if any heavy oil occurs in fore-arc basins, and natural bitumen does not occur in either fore-arc or delta basins.

Introduction

Until recent years conventional, light crude oil has been abundantly available and has easily met world demand for this form of energy. By year 2007, however, demand for crude oil worldwide has substantially increased, straining the supply of conventional oil. This has led to consideration of alternative or insufficiently utilized energy sources, among which heavy crude oil and natural bitumen are perhaps the most readily available to supplement short- and long-term needs. Heavy oil has long been exploited as a source of refinery feedstock, but has commanded lower prices because of its lower quality relative to conventional oil. Natural bitumen is a very viscous crude oil that may be immobile in the reservoir. It typically requires upgrading to refinery feedstock grade (quality). When natural bitumen is mobile in the reservoir, it is generally known as extra-heavy oil. As natural asphalt, bitumen has been exploited since antiquity as a source of road paving, caulk, and mortar and is still used for these purposes in some parts of the world. The direct use of mined asphalt for road paving is now almost entirely local, having been replaced by manufactured asphalt, which can be tailored to specific requirements.

This study shows the geological distribution of known heavy oil and natural bitumen volumes by basin type. These data are presented to advance a clearer understanding of the relationship between the occurrence of heavy oil and natural bitumen and the type of geological environment in which these commodities are found. The resource data presented were compiled from a variety of sources. The data should not be considered a survey of timely resource information such as data published annually by government agencies and public reporting services. With the exception of Canada, no such data source on heavy oil and natural bitumen accumulations is available. The amounts of heavy oil yet unexploited in known deposits represent a portion of future supply. To these amounts may be added the heavy oil in presently poorly known and entirely unexploited deposits. Available information indicates cumulative production accounts for less than 3 percent of the discovered heavy oil originally in place and less than 0.4 percent of the natural bitumen originally in place.

Terms Defined for this Report

- Conventional (light) Oil: Oil with API gravity greater than 25°.
- Medium Oil: Oil with API gravity greater than 20°API but less than or equal to 25°API.
- Heavy Oil: Oil with API gravity between 10°API and 20°API inclusive and a viscosity greater than 100 cP.
- Natural Bitumen: Oil whose API gravity is less than 10° and whose viscosity is commonly greater than 10,000 cP. It is not possible to define natural bitumen on the basis of viscosity alone because much of it, defined on the basis of gravity, is less viscous than 10,000 cP. In addition, viscosity is highly temperature-

dependent (fig. 1), so that it must be known whether it is measured in the reservoir or in the stock tank. In dealing with Russian resources the term natural bitumen is taken to include both maltha and asphalt but excludes asphaltite.

- Total Original Oil in Place (TOOIP): Both discovered and prospective additional oil originally in place.
- Original Oil in Place-Discovered (OOIP-Disc.): Discovered original oil in place.
- Reserves (R): Those amounts of oil commonly reported as reserves or probable reserves, generally with no further distinction, and quantities of petroleum that are anticipated to be technically but not necessarily commercially recoverable from known accumulations. Only in Canada are reserves reported separately as recoverable by primary or enhanced methods. Russian reserve classes A, B, and C1 are included here (See Grace, Caldwell, and Hether,1993, for an explanation of Russian definitions.)
- Prospective Additional Oil in Place: The amount of resource in an unmeasured section or portion of a known deposit believed to be present as a result of inference from geological and often geophysical study.
- Original Reserves (OR): Reserves plus cumulative production. This category includes oil that is frequently reported as estimated ultimately recoverable, particularly in the case of new discoveries.

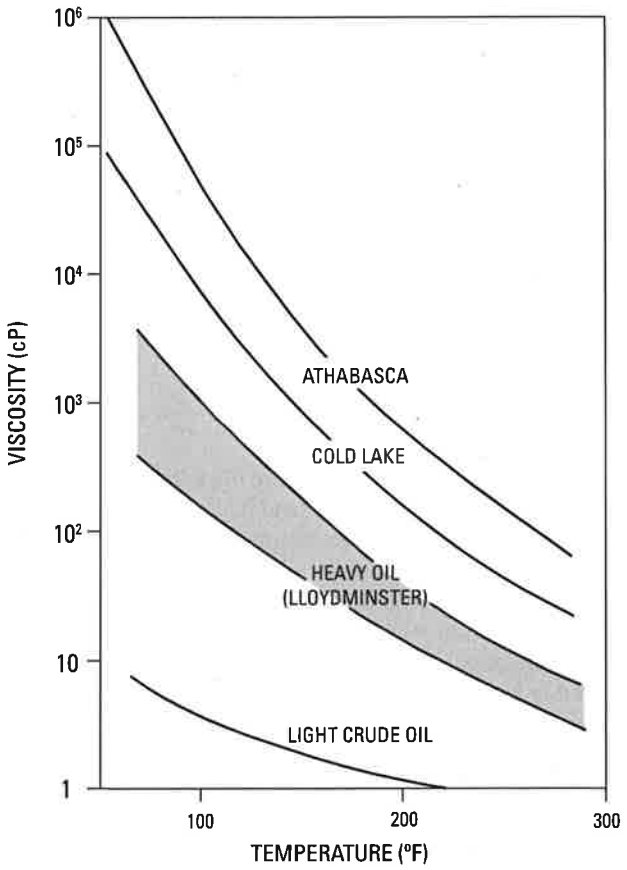

Figure 1. Response of viscosity to change in temperature for some Alberta oils (cP, centipoise), (Raicar and Proctor, 1984).

Chemical and Physical Properties

Fundamental differences exist between natural bitumen, heavy oil, medium oil, and conventional (light) oil, according to the volatilities of the constituent hydrocarbon fractions: paraffinic, naphthenic, and aromatic. When the light fractions are lost through natural processes after evolution from organic source materials, the oil becomes heavy, with a high proportion of asphaltic molecules, and with substitution in the carbon network of heteroatoms such as nitrogen, sulfur, and oxygen. Therefore, heavy oil, regardless of source, always contains the heavy fractions, the asphaltics, which consist of resins, asphaltenes, and preasphaltenes (the carbene-carboids) (Yen, 1984). No known heavy oil fails to incorporate asphaltenes. The large asphaltic molecules define the increase or decrease in the density and viscosity of the oil. Removal or reduction of asphaltene or preasphaltene drastically affects the rheological properties of a given oil and its aromaticity (Yen, 1984). Asphaltenes are defined formally as the crude oil fraction that precipitates upon addition of an n-alkane, usually n-pentane or n-heptane, but remains soluble in toluene or benzene. In the crude oil classification scheme of Tissot and Welte (1978), the aromatic-asphaltics and aromatic-naphthenics characterize the heavy oil and natural bitumen deposits of Canada and Venezuela and are the most important of all crude oil classes with respect to quantity of resources. The aromatic-intermediate class characterizes the deposits of the Middle East (Yen, 1984).

Some of the average chemical and physical properties of conventional, medium, and heavy crude oils and natural bitumen are given in table 1, in order to show their distinguishing characteristics. The data are derived from multiple sources, some old and others adhering to standards employed in different countries. The conversion factors outlined in table 2 were used to convert published data to a uniform standard. Some of the properties in table 1 are important with respect to heavy oil and natural recovery from the ground and other properties in table 1 serve as the basis for decisions for upgrading and refinery technologies. Moving across table 1 from conventional oil to natural bitumen, increases may be seen in density (shown as reductions in API gravity), coke, asphalt, asphaltenes, asphaltenes + resins, residuum yield (percent volume), pour point, dynamic viscosity, and the content of copper, iron, nickel, vanadium among the metals and in nitrogen and sulfur among the non-metals. Values diminish for reservoir depth, gasoline and gas-oil yields, and volatile organic compounds (VOC and BTEX –Benzene, Toluene, Ethylbenzene, and

Xylenes). The significance of these differences is often reflected in the capital and operating expenses required for the recovery, transportation, product processing, and environmental mitigation of the four oil types. The principal sources of analytical data for table 1 are Environmental Technology Centre (2003), Hyden (1961), Oil & Gas Journal Guide to Export Crudes (2006), U.S. Department of Energy, National Energy Technology Laboratory (1995), and various analyses published in technical reports.

The resins and asphaltenes play an important role in the accumulation, recovery, processing, and utilization of petroleum. The resins and asphaltenes are the final form of naphtheno-aromatic molecules. The carbon skeleton appears to comprise three to five polyaromatic sheets, with some heterocyclic (N-S-O) compounds. These crystallites may combine to form high molecular weight aggregates, with the high viscosity of heavy oils related to the size and abundance of the aggregates. Most asphaltenes are generated from kerogen evolution in response to depth and temperature increases in sedimentary basins. Different types of asphaltenes may be derived from the main kerogen types. Asphaltenes are not preferentially mobilized, as are light hydrocarbons during migration from source rocks to reservoir beds, where they are less abundant if the crude oil is not degraded (Tissot, 1981).

Some heavy oil and natural bitumen originates with chemical and physical attributes shown in table 1 as immature oil which has undergone little if any secondary migration. The greatest amount of heavy oil and natural bitumen results from the bacterial degradation under aerobic conditions of originally light crude oils at depths of about 5,000 feet or less and temperatures below 176°F. The consequence of biodegradation is the loss of most of the low molecular weight volatile paraffins and naphthenes, resulting in a crude oil that is very dense, highly viscous, black or dark brown, and asphaltic. An active water supply is required to carry the bacteria, inorganic nutrients, and oxygen to the oil reservoir, and to remove toxic by-products, such as hydrogen sulfide, with low molecular weight hydrocarbons providing the food (Barker, 1979). The low molecular weight components also may be lost through water washing in the reservoir, thermal fractionation, and evaporation when the reservoir is breached at the earth's surface (Barker, 1979). The importance of this process to the exploitation of heavy oil and natural bitumen lies in the increase of NSO (nitrogen-sulfur-oxygen) compounds in bacterially-altered crude oil and the increase in asphaltenes (Kallio, 1984).

Bacterial degradation of crude oil may also take place under anaerobic conditions, thus obviating the need for a fresh water supply at shallow depths (Head, Jones, and Larter, 2003; Larter and others, 2006). This proposal envisions degradation even of light oils at great depths so long as the maximum limiting temperature for bacterial survival is not exceeded. This theory does not account in any obvious way for the high percentage in heavy oil and natural bitumen of polar asphaltics, that is, the resins and asphaltenes.

Oil mass loss entailed in the formation of heavy oil and natural bitumen deposits has been the subject of numerous research studies. Beskrovnyi and others (1975) concluded that three to four times more petroleum was required than the reserves of a natural bitumen for a given deposit. Based upon material balance calculations in the Dead Sea basin, Tannenbaum, Starinsky, and Aizenshtat (1987) found indications that 75% of the original oil constituents in the C15+ range had been removed as a result of alteration processes. By accounting for the lower carbon numbers as well, they estimated that the surface asphalts represented residues of only 10-20% of the original oils. Head, Jones, and Larter (2003) diagram mass loss increasing from essentially zero for conventional oil to something more than 50% for heavy oils, which of themselves are subject to no more than 20% loss. Accompanying the mass loss is a decrease in API gravity from 36° to 5-20°; decrease in gas/oil ratio from 0.17 kg gas/kg oil; decrease in gas liquids from 20% to 2%; increase in sulfur from 0.3wt% to 1.5+wt%; and decrease in C15+ saturates from 75% to 35%. This calculation of mass loss shows: (1) the enormous amount of oil initially generated in heavy oil and natural bitumen basins, especially Western Canada Sedimentary and Eastern Venezuela basins; and (2) the huge economic burden imposed by this mass loss on the production-transportation-processing train of the remaining heavy oil and natural bitumen.

Origins of Heavy Oil and Natural Bitumen

It is possible to form heavy oil and natural bitumen by several processes. First, the oil may be expelled from its source rock as immature oil. There is general agreement that immature oils account for a small percentage of the heavy oil (Larter and others, 2006). Most heavy oil and natural bitumen is thought to be expelled from source rocks as light or medium oil and subsequently migrated to a trap. If the trap is later elevated into an oxidizing zone, several processes can convert the oil to heavy oil. These processes include water washing, bacterial degradation and evaporation. In this case, the biodegradation is aerobic. A third proposal is that biodegradation can also occur at depth in subsurface reservoirs (Head, Jones, and Larter, 2003; Larter and others, 2003; Larter and others, 2006). This explanation permits biodegradation to occur in any reservoir that has a water leg and has not been heated to more than 176° F. The controls on the biodegradation depend on local factors rather than basin-wide factors. Because the purpose of this report is to describe the geologic basin setting of the known heavy oil and natural bitumen deposits, it is beyond the scope of this report to argue the source or genesis of heavy oil and natural bitumen for each basin of the world.

Data Sources

Data for heavy oil resource occurrences and quantities for individual oilfields and reservoirs have been compiled from many published reports and commercial data bases. The most important of these include Demaison (1977), IHS Energy Group (2004), NRG Associates (1997), Parsons (1973), Roadifer (1987), Rühl (1982), and the U.S. Department of Energy, National Energy Technology Laboratory (1983, 2005)

Data for natural bitumen deposits in the United States are summarized in U.S Department of Energy, National Energy Technology Laboratory (1991), but information for Utah is taken from Oblad and others (1987) and Ritzma (1979). Although there is no single data source for deposits outside the United States, there is a rich literature, particularly for Russia and the countries of the Former Soviet Union. For Canada, reliance is placed on reports of the Alberta Energy and Utilities Board (2004) and Saskatchewan Industry and Resources (2003).

Resource Estimates

We consider the total original oil in place (OOIP) to be the most useful parameter for describing the location and volume of heavy oil and natural bitumen resources. Resource quantities reported here are based upon a detailed review of the literature in conjunction with available databases, and are intended to suggest, rather than define the resource volumes that could someday be of commercial interest. If only a recoverable volume of heavy oil for the accumulation was published, the discovered OOIP was computed according to the protocol set forth in table 3.

Natural bitumen originally in place is often reported in the literature. Where only a recoverable estimate is published, the in-place volumes were calculated according to the protocols given for heavy oil; this is especially the case for bitumen deposits above 4°API gravity, to which we arbitrarily refer as extra-heavy oil.

Poorly known deposits of heavy oil and natural bitumen are included in the category of prospective additional resources, as described in table 3. In no case are values for prospective additional resource volumes calculated as in the case of discovered resources but were taken directly from the published literature.

Table 4 summarizes the resources and essential physical parameters of the heavy oil and natural bitumen contained in each of the basin types. These characteristics affect heavy oil and natural bitumen occurrence and recovery. Recovery can be primary, as in the case of cold production without gravel packing, if the gas to oil ratio is high enough to provide necessary reservoir energy. Otherwise, recovery generally necessitates the application of enhanced recovery methods, such as thermal energy or the injection of solvents.

Recovery Methods

How the reservoir parameters apply to enhanced recovery is summarized from Taber, Martin, and Seright (1997a, 1997b) in table 5, which covers the most commonly used, or at least attempted enhanced oil recovery (EOR) methods. Of these methods, immiscible gas injection, polymer flooding, and *in situ* combustion (fireflood) have met with limited success for heavy oil and natural bitumen. Steam injection (cyclic steam, huff 'n puff) has been most successful, frequently by use of cyclic steam, followed by steam flooding. Surface mining and cold *in situ* production are usually considered to be primary recovery methods. They can be suited to the extraction of heavy oil and natural bitumen under proper conditions.

Most of the process descriptions which follow are taken from Taber, Martin, and Seright (1997b). Many processes may result in the process agent, such as nitrogen or carbon dioxide, remaining immiscible with the reservoir hydrocarbon or else becoming miscible with it. The miscibility is dependent upon the minimum miscibility pressure (MMP) and determines the way in which the process agent achieves EOR. While this summary discussion shows the breadth of the EOR processes operators have tried and continue to try as experimental projects, thermal EOR methods account for most of the heavy oil that is commercially produced. Data on the frequency of the applications are taken, unless otherwise cited, from the Oil and Gas Journal Historical Review, 1980-2006 (2006), particularly the Oil and Gas Journal 2000 and 2006 EOR Surveys.

Nitrogen gas drive is low in cost and therefore may be used in large amounts. It is commonly used with light oils for miscible recovery. However, it may also be used for an immiscible gas flood. The Oil and Gas Journal 2000 Survey includes one immiscible nitrogen gas drive in a sandstone reservoir with 16°API oil at 4,600 feet depth. It was reported to be producing 1,000 barrels per day (b/d) of enhanced production. The Journal's 2006 Survey reports one each heavy oil nitrogen miscible and nitrogen immiscible projects. The miscible project is 19°API, located in the Bay of Campeche, with 19 wells, but with no report of production capacity. The immiscible project has oil of 16°API at 4,600 feet in sandstone. For this project total production is reported to be 1,500 b/d of which 1,000 b/d is enhanced by immiscible nitrogen injection.

Of the 77 CO2 projects in the Journal 2000 Survey, 70 are for miscible CO2 and none entails heavy oil. This is true also in the Journal 2006 Survey, where all 86 CO2 projects are devoted to light oil, above 28°API. In the Journal 2000 Survey, five of the seven immiscible CO2 projects are applied to heavy oil reservoirs, four in clastics and one in limestone. The latter, in the West Raman field in Turkey, involves oil of 13°API, lies at 4,265 feet, and produces 8,000 b/d. The reservoir contains nearly two billion barrels of original oil in place. Recoverable reserves remain low because of the recalcitrance of the reservoir. Steam flooding has been unsuccessful. By the date of the Journal 2006, there are eight immiscible CO2 projects, with five of them entailing heavy oil amounting to 7,174 b/d. The

two largest projects are light oil and heavy oil and are each in carbonate reservoirs.

Polymer/chemical flooding includes micellar/polymer, alkaline-surfactant-polymer (ASP), and alkaline fluids (Taber, Martin, and Seright, 1997a, 1997b). Recovery is complex, leading to the lowering of interfacial tension between oil and water, solubilization of oil in some micellar systems, emulsification of oil and water, wettability alteration, and enhancement of mobility. Limitations and costs indicate for these floods the desirability of clean clastic formations. The Journal 2000 Survey shows five heavy oil polymer/chemical floods of 15°API in sandstone reservoirs at about 4,000 feet. They were producing about 366 b/d and the projects were deemed successful or promising. Projects such as these are below the desirable gravity limits and are more viscous than desired at 45 cP.

Polymer floods improve recovery over untreated water flood by increasing the viscosity of the water, decreasing thus the mobility of the water, and contacting a larger volume of the reservoir. The advantages of a polymer flood over a plain water flood are apparent. The Journal 2000 Survey lists 22 polymer flood projects, of which five involve heavy oil. These five are within the range of the polymer screen, although the gravities are marginal, lying from 13.5°API to a bit above 15°API. The five were producing 7,140 b/d, of which 2,120 b/d were attributed to EOR. The Journal 2006 Survey shows 20 polymer floods, with five exploring heavy oil reservoirs. Three of the five are producing 7,140 b/d total oil and 2,120 b/d of enhanced production.

The Journal 2000 Survey shows four hot water floods, one of which is heavy oil with a gravity of 12°API, viscosity of 900 cP, and starting saturation of only 15 percent. Project production was 300 b/d. Two of three hot water floods included in the Journal 2006 Survey are intended to enhance production of heavy oil. The two yield about 1,700 b/d of total oil and 1,700 b/d of enhanced hot water flood oil.

In situ combustion (fire flood) is theoretically simple, setting the reservoir oil on fire and sustaining the burn by the injection of air. Usually, the air is introduced through an injector well and the combustion front moves toward to the production wells. A variant is to include a water flood with the fire, the result being forward combustion with a water flood. Another variant is to begin a fire flood, then convert the initial well to a producer and inject air from adjacent wells. The problem with this reverse combustion is that it doesn't appear to work.

In situ combustion leads to oil recovery by the introduction of heat from the burning front, which leads to reduction in viscosity. Further, the products of steam distillation and thermal cracking of the reservoir oil are carried forward to upgrade the remaining oil. An advantage of the process is that the coke formed by the heat itself burns to supply heat. Lastly, the injected air adds to the reservoir pressure. The burning of the coke sustains the process so that the process would not work with light oil deficient in asphaltic components. The process entails a number of problems, some severe, but the Journal 2000 Survey shows 14 combustion projects, of which five are light oil and the remaining nine are heavy, between 13.5°API and 19°API. Viscosities and starting oil saturations are relatively high. It is notable that the heavy oil projects are in sandstones and the light oil in carbonates. The heavy oil *in situ* combustion projects were producing about 7,000 b/d. The Journal 2006 Survey includes nine heavy oil combustion projects among a total of twenty-one. The heavy oil projects yield about 7,000 b/d of combustion-enhanced oil, which ranges from 13.5°API to 19°API.

Steam injection for EOR recovery is done in two ways, either by cyclic steam injection (huff 'n puff) or continuous steam flood. Projects are frequently begun as cyclic steam, whereby a high quality steam is injected and soaks the reservoir for a period, and the oil, with lowered viscosity from the heat, is then produced through the injection well. Such soak cycles may be repeated up to six times, following which a steam flood is initiated. In general, steam projects are best suited to clastic reservoirs at depths no greater than about 4,000 feet, and with reservoir thicknesses greater than 20 feet and oil saturations above 40% of pore volume. For reservoirs of greater depth the steam is lowered in quality through heat loss to the well bore to where the project becomes a hot water flood. Steam is seldom applied to carbonate reservoirs in large part due to heat loss in fractures.

The Journal 2000 Survey lists 172 steam drive projects. Of these, four in Canada give no gravity reading, thirteen are medium oil from 22°API to 25°API, and the rest are heavy oil. The largest of all is at Duri field in Indonesia and this oil is 22°API. For the project list as a whole, the average gravity is 14°API, with a maximum value of 30°API and a minimum of 4°API. The average viscosity is 37,500 cP, with maximum and minimum values of 5,000,000 cP and 6 cP. Oil saturations range from 35% to 90%, the average being 68%. Most importantly, production from the project areas was 1.4 million b/d and of this, 1.3 million b/d was from steam drive EOR.

All but three of the 120 steam projects found in the Journal 2006 Survey entail recovery of heavy oil. The oil averages 12.9°API, with a low value of 8°API and a high of 28°API (one of the three light oil reservoirs). The viscosity averages 58,000 cP, with a high value of 5 million cP and a low of 2 cP. The projects are yielding over 1.3 million b/d, virtually all being steam EOR.

Maps

The geographic distribution of basins reporting heavy oil and natural bitumen, as identified by their Klemme basin types, appears on Plate 1. A diagram of the Klemme basin classification illustrates the architectural form and the geological basin structure by type. This plate also includes histograms of the total original oil in place resource volumes of both heavy oil and natural bitumen. Plates 2 and 3, respectively, depict the worldwide distribution of heavy oil and natural bitumen resources originally in place. Each map classifies basins

by the reported volumes of total original oil in place. A table ranks the basins by total original oil in place volumes besides indicating Klemme basin type and reporting discovered original oil in place and prospective additional oil in place. Plates 2 and 3 also include an inset map of the geographic distribution of original heavy oil or natural bitumen by 10 world regions (see table 6 for regional listing of countries reporting heavy oil or natural bitumen.)

Basin outlines of the sedimentary provinces are digitally reproduced from the AAPG base map compiled by St. John (1996). The basin outlines of St. John (1996) are unaltered. However, the reader should note that the basin outlines are considered to be generalizations useful for displaying the resource distributions but are less than reliable as a regional mapping tool. Also, some basin names have been changed to names more commonly used by geologists in the local country. These equivalent names and the original names from Bally (1984) and St. John (1996) are detailed in table 1-1 in Appendix 1. The basin outline for Eastern Venezuela as shown does not include the island of Trinidad where both heavy oil and natural bitumen resources occur. For this report, resources from Trinidad and Tobago are reported in the Eastern Venezuela basin totals. In a few cases a single basin as outlined on the plates is composed of multiple basins to provide more meaningful local information. This is particularly true in the United States, where the AAPG-CSD map was employed (Meyer, Wallace, and Wagner, 1991). In each case, the individual basins retain the same basin type as the basin shown on the map and all such basins are identified in Appendix 1.

Basins having heavy oil or natural bitumen deposits are listed in table 2-1 in Appendix 2 along with the Klemme basin type, countries and U.S. states or Canadian provinces reporting deposits and other names cited in literature. The Klemme basin classification diagram in Plate 1 is reprinted in fig. 3-1 in Appendix 3 for the reader's convenience. The tables from Plates 2 and 3 are reprinted as table 4-1 and table 4-2 for the reader's convenience.

Klemme Basin Classification

Many classifications of petroleum basins have been prepared. In one of the earliest, Kay (1951) outlined the basic architecture of geosynclines, with suggestions as to their origins. Kay's work preceded the later theory of plate tectonics. Klemme (1977, 1980a, 1980b, 1983, 1984) gives a summary description of petroleum basins together with their classification, based upon basin origin and inherent geological characteristics. This classification is simple and readily applicable to the understanding of heavy oil and natural bitumen occurrence. The Klemme basin types assigned to the heavy oil and natural bitumen basins described in this report correspond to the assignments made in St. John, Bally, and Klemme (1984). In some cases of multiple type designations in St. John, Bally, and Klemme (1984) a unique type designation was resolved by reference to Bally (1984) or Bally and Snelson (1980). Only a few of the basins originally designated as multiple types in St. John, Bally and Klemme (1984) appear to contain heavy oil and natural bitumen.

Table 7 summarizes the criteria upon which Klemme based his classification. The general description of the resource endowment associated to the Klemme basin classification is based upon oilfield (and gasfield) data of the world as of 1980 without regard to the density or other chemical attributes of the hydrocarbons they contain (Klemme, 1984). At the time of Klemme's work, the average density U.S. refinery crude oil was about 33.7°API (Swain, 1991). A decline in the average to about 30.6°API by 2003 perhaps signifies the increasing importance of heavy oil in the mix (Swain, 2005).

Generally, basins may be described as large or small and linear or circular in shape. They may also be described by the ratio of surface area to sedimentary volume. The basement profile or basin cross-section, together with the physical description, permits the interpretation of the fundamental basin architecture. The basin can then be placed within the relevant plate tectonic framework and assigned to one of four basin types, of which two have sub-types. A diagram of the Klemme basin types appears on Plate 1, color-coded to the basins on the map.

In the following section we provide descriptions of the basin types from Klemme (1980b, 1983, 1984) followed by discussion of the heavy oil and natural bitumen occurrences within those same basin types, summary data for which are given in table 4. Because most heavy oil and natural bitumen deposits have resulted from the alteration of conventional and medium oil, the factors leading to the initial conventional and medium oil accumulations are relevant to the subsequent occurrence of heavy oil and natural bitumen.

Type I. Interior Craton Basins

The sediment load in these basins is somewhat more clastic than carbonate. Reservoir recoveries are low and few of the basins contain giant fields. Traps are generally related to central arches, such as the Cincinnati arch, treated here as a separate province (Plates 1-3), or the arches of the Siberian platform (see below for further explanation). Traps also are found in smaller basins over the craton, such as the Michigan basin. The origin of these depressions is unclear although most of them began during the Precambrian (Klemme, 1980a, 1980b).

The six Type I basins having heavy oil contain less than 3 billion barrels of oil in place and of this 93% occurs in the Illinois basin alone. Four Type I basins that contain natural bitumen have 60 billion barrels of natural bitumen in place, with nearly 99% in the Tunguska basin in eastern Siberia and the rest in the Illinois basin. The Tunguska basin covers most of the Siberian platform, around the borders of which are found cratonic margin basins of Type IIA. For convenience all the resource is assigned to the Tunguska basin. The prospec-

tive additional resource of 52 billion barrels is almost certainly an absolute minimum value for this potentially valuable but difficult to access area (Meyer and Freeman, 2006.)

Type II. Continental Multicyclic Basins

Type IIA. Craton margin (composite)

These basins, formed on continental cratonic margins, are generally linear, asymmetrical in profile, usually beginning as extensional platforms or sags and ending as compressional foredeeps. Therefore they are multicyclic basins featuring a high ratio of sediment volume to surface area. Traps are mainly large arches or block uplifts and may be found in rocks of either the lower (platform) or upper (compression) tectonic cycle. About 14% of conventional oil discovered in the world by 1980 is from marginal cratonic basins (Klemme, 1980a, 1980b).

Type IIA basins are of moderate importance with respect to heavy oil, with about 158 billion barrels of oil in place distributed among 28 basins. Three Type IIA basins, the Western Canada Sedimentary, Putumayo, and Volga-Ural, have combined total heavy oil resource of 123 billion barrels of oil in place, or 78% of the total for Type IIA basins.

In comparison, natural bitumen in 24 Type IIA basins accounts for 2,623 billion barrels of natural bitumen in place, or nearly 48% of the world natural bitumen total. The Western Canada Sedimentary basin accounts for 2,334 billion barrels of natural bitumen in place, or about 89%. Of the Canadian total, 703 billion barrels of natural bitumen in place is prospective additional oil, largely confined to the deeply buried bitumen in the carbonate that underlie the Peace River and part of the Athabasca oil sand deposit in an area known as the Carbonate Triangle. The significance of the Canadian deposits lies in their concentration in a few major deposits: Athabasca, from which the reservoir is exploited at or near the surface and shallow subsurface, and Cold Lake and Peace River, from which the bitumen is extracted from the subsurface. Two other basins contain much less but still significant amounts of natural bitumen, the Volga-Ural basin in Russia (263 billion barrels of natural bitumen in place) and the Uinta basin in the United States (12 billion barrels of natural bitumen in place). The Volga-Ural deposits are numerous, but individually are small and mostly of local interest. The Uinta deposits are much more concentrated aerially, but are found in difficult terrain remote from established transportation and refining facilities.

Type IIB. Craton accreted margin (complex)

These basins are complex continental sags on the accreted margins of cratons. Architecturally, they are similar to Type IIA basins, but begin with rifting rather than sags. About three-quarters of Type IIA and IIB basins have proven productive, and they contain approximately one-fourth of the world's total oil and gas (Klemme, 1980a, 1980b).

The 13 Type IIB basins contain a moderate amount of heavy oil (193 billion barrels of oil in place). The two most significant basins are in Russia, West Siberia and Timan-Pechora. These, together with most of the other Type IIB heavy oil basins, are of far greater importance for their conventional and medium oil resources.

Five Type IIB basins hold 29 billion barrels of natural bitumen in place. Only the Timan-Pechora basin contains significant natural bitumen deposits, about 22 billion barrels of natural bitumen in place. Unfortunately, this resource is distributed among a large number of generally small deposits.

Type IIC. Crustal collision zone (convergent plate margin)

These basins are found at the crustal collision zone along convergent plate margins, where they are downwarped into small ocean basins. Although they are compressional in final form, as elongate and asymmetrical foredeeps, they begin as sags or platforms early in the tectonic cycle. Type IIC downwarp basins encompass only about 18 percent of world basin area, but contain nearly one-half of the world's total oil and gas. These basins are subdivided into three subtypes, depending on their ultimate deformation or lack thereof: Type IICa, closed; Type IICb, trough; and Type IICc, open (Klemme, 1980a, 1980b).

Although basins of this type begin as downwarps that opened into small ocean basins (Type IICc), they may become closed (Type IICa) as a result of the collision of continental plates. Upon closing, a large, linear, asymmetric basin with sources from two sides is formed, resembling a Type IIA basin. Further plate movement appears to destroy much of the closed basin, leaving a narrow, sinuous foredeep, that is, a Type IICb trough. Relatively high hydrocarbon endowments in the open and the closed types may be related to above-normal geothermal gradients, which accentuates hydrocarbon maturation and long-distance ramp migration. Traps are mostly anticlinal, either draping over arches or compressional folds, and are commonly related to salt flowage.

Type IICa basins, with their architectural similarity to Type IIA basins, are the most important of the three Type IIC heavy oil basins. The 15 basins account for 1,610 billion barrels of the heavy oil in place, with the Arabian, Eastern Venezuela, and Zagros basins containing 95% of the total. Of particular interest is the Eastern Venezuela basin which includes large accumulations of conventional and medium oil, while at the same time possessing an immense resource of both heavy oil and natural bitumen.

Type IICa basins also are rich in natural bitumen, with a total of 2,507 billion barrels of natural bitumen in place among the six. About 83% of this occurs in Venezuela, mostly in the southern part of the Eastern Venezuelan basin known as the Orinoco Oil Belt. Here the reservoir rocks impinge upon the

Guyana craton in much the same fashion as the reservoir rocks of the Western Canada Sedimentary basin lap onto the Canadian shield. The only other significant Type IICa accumulation of natural bitumen is found in the North Caspian basin (421 billion barrels of natural bitumen in place).

Fourteen Type IICb basins contain modest amounts of heavy oil (32 billion barrels of oil in place) and even less of natural bitumen (5 billion barrels of natural bitumen in place in seven basins). Much of this resource is found in the Caltanisetta and Durres basins, on either side of the Adriatic Sea. Durres basin resources are aggregated with the South Adriatic and the province is labeled South Adriatic on the plates. Significant amounts of the Caltanisetta resource occurs offshore.

The amount of heavy oil in the 12 Type IICc basins is substantial (460 billion barrels of oil in place). The Campeche, by far the largest, and Tampico basins in Mexico and the North Slope basin in the United States account for 89% of the heavy oil. The Campeche field, which is actually an assemblage of closely associated fields, is found about 65 miles offshore of the Yucatan Peninsula in the Gulf of Mexico. The North Slope basin, on the north coast of Alaska, occurs in an area of harsh climate and permafrost, which makes heavy oil and natural bitumen recovery by the application of thermal (steam) methods difficult both physically and environmentally. The U.S. fields in the East Texas, Gulf Coast, and Mississippi Salt Dome basins account for only 5% of the heavy oil in basins of this type.

Only a small amount of natural bitumen (24 billion barrels) has been discovered in eight Type IICc basins. Two of these, the North Slope and South Texas Salt Dome basins, are significant for possible future development.

Type III. Continental Rifted Basins

Type IIIA. Craton and accreted zone (rift)

These are small, linear continental basins, irregular in profile, which formed by rifting and simultaneous sagging in the craton and along the accreted continental margin. About two-thirds of them are formed along the trend of older deformation belts and one-third are developed upon Precambrian shields. Rifts are extensional and lead to block movements so that traps are typically combinations. Oil migration was often lateral, over short distances. Rift basins are few, about five percent of the world's basins, but half of them are productive. Because of their high recovery factors, Type IIIA basins accounted for 10% of the world's total recoverable oil and gas in 1980 (Klemme, 1980a, 1980b).

Globally, there are 28 Type IIIA heavy oil basins, containing 222 billion barrels of oil in place The Bohai Gulf basin in China accounts for 63% of the heavy oil, with an additional 11% derived from the Gulf of Suez and 10% from the Northern North Sea. Outside of these, most Type IIIA basins contain just a few deposits. The five basins in Type IIIA have almost 22 billion barrels of natural bitumen in place, but half of that is located in the Northern North Sea basin.

Type IIIB. Rifted convergent margin (oceanic consumption)

Type IIIBa basins are classified as back-arc basins on the convergent cratonic side of volcanic arcs. They are small, linear basins with irregular profiles (Klemme, 1980a, 1980b).

Not unlike Type IIIA basins, the volume of heavy oil found in the Type IIIBa basins is small. Seventeen heavy oil basins contain 49 billion barrels of oil in place and 83% of this amount is in Central Sumatra.

Just 4 billion barrels of natural bitumen in place are identified in the Type IIIA basin called Bone Gulf. Small amounts are also known to occur in the Cook Inlet and Tonga basins.

Type IIIBb basins are associated with rifted, convergent cratonic margins where wrench faulting and subduction have destroyed the island arc. They are small, linear, and irregular in profile.

The 14 Type IIIBb basins containing heavy oil account for only 134 billion barrels of oil in place. These basins are only moderately important on a global scale, but have been very important to the California oil industry. The seven such basins of California - Central Coastal, Channel Islands, Los Angeles, Sacramento, San Joaquin, Santa Maria, and Ventura – equal 129 billion barrels of oil in place or 96%.

There are nine Type IIIBb basins that report natural bitumen deposits. They contain 4 billion barrels of natural bitumen in place, about half of which is in the Santa Maria basin.

Types IIIBa and IIIBb basins comprise about seven percent of world basin area, but only one-quarter of the basins are productive for oil of all types. However, the productive ones, which represent only two percent of world basin area, yield about seven percent of total world's oil and gas (Klemme, 1983). Some of these productive basins, particularly those located in California, have high reservoir recovery factors.

Type IIIBc basins are small and elongate, irregular in profile, and occupy a median zone either between an oceanic subduction zone and the craton or in the collision zone between two cratonic plates. They result from median zone wrench faulting and consequent rifts. Such basins make up about three and one-half percent of world basin area and contribute two and one-half percent of total world oil and gas.

Type IIIBc basins are important to the occurrence of heavy oil (351 billion barrels of oil in place). Although there are nine basins of this type, 92% of the heavy oil is concentrated in the Maracaibo basin. The Maracaibo basin also yields 95% of the 178 billion barrels of natural bitumen in place in the five basins containing this type of oil. This makes the Maracaibo basin unique: no other basin type is so completely dominated by a single basin.

Type IIIC. Rifted passive margin (divergence)

These basins, often aptly called pull-apart basins, are extensional, elongate, and asymmetric. Located along major oceanic boundaries of spreading plates, they are divergent and occupy the intermediate zone between thick continental crust and thin oceanic crust. They appear to begin with a rifting stage, making possible the later sedimentary fill from the continent. Type IIIC basins, comprising 18 percent of the world's basin area, are mostly offshore and are often in water as deep as 5,000 feet. For this reason their development has been slow but is accelerating as traditional, easily accessible basins reach full development and world demand for petroleum increases (Klemme, 1980a, 1980b).

Twenty-eight Type IIIC basins yield 158 billion barrels of heavy oil in place, but one, the offshore Campos basin, contains 66% of this heavy oil. These continental margin basins must at some point in their histories have been sufficiently elevated to permit their generated conventional oil to be degraded. It is possible that the heavy oil could be very immature, having undergone only primary migration and later elevation. The geologic history of such basins does not encourage this view. However, the oil could well have been degraded bacterially at depth according to the recently proposed mechanisms suggested by Head, Jones, and Larter (2003) and Larter and others (2006). In a pull-apart basin the sediments would have accumulated rapidly and at depth, the expressed oil then was subject to degradation. The problem with degradation at depth is the loss of mobility unless it can be demonstrated that the oil was never elevated and, in fact, the Campos basin oil is deep, occurring at an average depth of nearly 8,400 feet.

The bitumen resource in Type IIIC basins is small (47 billion barrels of natural bitumen in place in seven basins), as are nearly all bitumen occurrences in comparison with the Western Canada Sedimentary and Eastern Venezuela basins. But the 38.3 billion barrels of natural bitumen in place in the Ghana basin of southwestern Nigeria is exploitable and the amount of the resource may be understated. Like many bitumen deposits it awaits more detailed evaluation.

Type IV. Delta (Tertiary to recent)

Deltas form along continental margins as extensional sags, are circular to elongate, and show an extremely high ratio of sediment fill to surface area. Architecturally, they are modified sags comprised of sediment depocenters and occur along both divergent and convergent cratonic margins. Although by 1980 delta basins provide two and one-half percent of world basin area and perhaps six percent of total oil and gas (Klemme, 1980a, 1980b), they account for more of the conventional resource endowment with the recent successful exploration in frontier deep water areas.

The three Type IV delta basins produce scant heavy oil (37 billion barrels of oil in place) and no natural bitumen. This is related to the extremely high ratio of sediment fill to surface area and that these basins exhibited rapid burial of the source organic matter. Burial is constant and uninterrupted, providing very limited opportunity for degradation of the generated petroleum.

Type V. Fore-Arc Basins

Fore-arc basins are located on the ocean side of volcanic arcs. They result from both extension and compression, are elongate and asymmetrical in profile, and architecturally are the result of subduction. Fore-arc basins are few in number and generally not very productive (Klemme, 1980a, 1980b).

Very small amounts of heavy oil are found in the Barbados basin. Although a natural bitumen deposit is reported in the Shumagin basin, volume estimates are not available.

Essentially no heavy oil or natural bitumen is found in fore-arc basins because these basins do not generate large quantities of petroleum of any type and therefore provide relatively little material to be degraded.

Regional Distribution of Heavy Oil and Natural Bitumen

The preceding discussion has been concerned with the distribution of heavy oil and natural bitumen in the world's geological basins. This is of paramount interest in the exploration for the two commodities and for their exploitation. The chemical and physical attributes of the fluids and the reservoirs which contain them do not respect political boundaries.

At the same time it is necessary to understand the geography of the heavy oil and natural bitumen for both economic and political reasons. These factors will be dealt with in detail in a subsequent report. The bar graphs on Plates 2 and 3 give the regional distribution of total and discovered original oil in-place for heavy oil and natural bitumen, respectively. The distribution of the resources is given in table 8. The western hemisphere accounts for about 52 percent of the world's heavy oil and more than 85 percent of its natural bitumen. The Middle East and South America have the largest in-place volumes of heavy oil, followed by North America. North and South America have, by far, the largest in-place volumes of natural bitumen. Very large resource deposits are also known in eastern Siberia but insufficient data are available to make more than nominal size estimates.

Summary

From the preceding basin discussion, Klemme basin Type IICa is by far the most prolific in terms of heavy oil. For natural bitumen Klemme basin Type IIA and Type IICa are the most prolific. The basin types involved are architecturally analogous, beginning with depositional platforms or sags

and ending up as foredeeps. They differ only in their modes of origin. What they have in common is truncation against cratonic masses updip from rich source areas. This situation permitted immense accumulations of conventional oil at shallow depths, with near ideal conditions for oil entrapment and biodegradation resulting in formation of heavy oil and bitumen accumulations. The prospective resources from the prospective additional resource deposits in these basins are larger than the discovered resources of many basin types.

The Klemme basin classification system includes elements of basin development and architecture that control basin type. The observed pattern of the heavy oil and natural bitumen occurrences across basin types is consistent with the formation of heavy oil and natural bitumen through the process of degradation of conventional oil. Only relatively small quantities of heavy oil were found in the Interior Craton (Type I), Deltas (Type IV) and Fore-Arc basins (Type V).

Type IICa basins, including the Arabian, Eastern Venezuela, and Zagros, have the largest endowments of heavy oil and also contain the largest amounts of conventional oil. Large volumes of heavy oil are also found in both Type IICc basins, notably, the Campeche, Tampico, and North Slope basins, and in Type IIIBc basins, primarily Maracaibo basin. For natural bitumen, the Western Canada Sedimentary and Eastern Venezuela basins have similar development histories and basin architectural features. Some basin development patterns promote the formation of greater volumes of heavy oil and natural bitumen than others. This is seen most clearly in present occurrences of heavy oil and natural bitumen in the Type IICa and Type IICc basins, with their rich source areas for oil generation and up-dip migration paths to entrapment against cratons. Conventional oil may easily migrate through the tilted platforms until the platforms are breached at or near surface permitting deveopment of asphaltic seals.

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Tables 1–8

Table 1. Some chemical and physical attributes of crude oils (averages).

[cP, centipoise; wt%, weight percent; mgKOH/g, milligrams of potassium hydroxide per gram of sample; sp gr, specific gravity; vol%, volume percent; ppm, parts per million; Concarbon, Conradson carbon; VOC, volatile organic compounds; BTEX, benzene, toluene, ethylbenzene, and xylenes]

Attribute	Unit	Conventional oil (131 basins, 8148 deposits)	Medium oil (74 basins, 774 deposits)	Heavy oil (127 basins, 1199 deposits)	Natural bitumen (50 basins, 305 deposits)
API gravity	degrees	38.1	22.4	16.3	5.4
Depth	feet	5,139.60	3,280.20	3,250.00	1,223.80
Viscosity (77°F)	cP	13.7	34	100,947.00	1,290,254.10
Viscosity (100°F)	cP	10.1	64.6	641.7	198,061.40
Viscosity (130°F)	cP	15.7	34.8	278.3	2,371.60
Conradson Carbon	wt%	1.8	5.2	8	13.7
Coke	wt%	2.9	8.2	13	23.7
Asphalt	wt%	8.9	25.1	38.8	67
Carbon	wt%	85.3	83.2	85.1	82.1
Hydrogen	wt%	12.1	11.7	11.4	10.3
Nitrogen	wt%	0.1	0.2	0.4	0.6
Oxygen	wt%	1.2		1.6	2.5
Sulfur	wt%	0.4	1.6	2.9	4.4
Reid vapor pressure	psi	5.2	2.6	2.2	
Flash point	°F	17	20.1	70.5	
Acid number	mgKOH/g	0.4	1.2	2	3
Pour point	°F	16.3	8.6	19.7	72.9
C1-C4	vol%	2.8	0.8	0.6	
Gasoline + naphtha	vol%	31.5	11.1	6.8	4.4
Gasoline + naphtha	sp gr	0.76	0.769	0.773	0.798
Residuum	vol%	22.1	39.8	52.8	62.2
Residuum	sp gr	0.944	1.005	1.104	1.079
Asphaltenes	wt%	2.5	6.5	12.7	26.1
Asphaltenes + resins	wt%	10.9	28.5	35.6	49.2
Aluminum	ppm	1.174	1.906	236.021	21,040.03
Copper	ppm	0.439	0.569	3.965	44.884
Iron	ppm	6.443	16.588	371.05	4,292.96
Mercury	ppm	19.312	15	8.74	0.019
Nickel	ppm	8.023	32.912	59.106	89.137
Lead	ppm	0.933	1.548	1.159	4.758
Titanium	ppm	0.289	0.465	8.025	493.129
Vanadium	ppm	16.214	98.433	177.365	334.428
Residue Concarbon	wt%	6.5	11.2	14	19
Residue Nitrogen	wt%	0.174	0.304	0.968	0.75
Residue Nickel	ppm	25.7	43.8	104.3	
Residue Sulfur	ppm	1.5	3.2	3.9	
Residue Vanadium	ppm	43.2	173.7	528.9	532
Residue viscosity (122°F)	cP	1,435.80	4,564.30	23,139.80	
Total BTEX volatiles	ppm	10,011.40	5,014.40	2,708.00	
Total VOC volatiles	ppm	15,996.30	8,209.20	4,891.10	

Table 2. Conversion factors and equivalences applied to standardize data.

Standard unit in this report	Units as reported in literaure	Formula
	API gravity	
°API (degrees)	specific gravity (sp gr), (g/cm³)	= (141.5/(sp gr))-131.5
	Area	
acre	square mile (mi²)	= (1/640) mi²
	square kilometer (km²)	= 0.00405 km²
	hectare (ha)	= 0.405 ha
	Asphalt in crude	
weight percent (wt%)	Conradson Carbon Residue (CCR)	= 4.9× (CCR)
	Barrels of oil	
barrel (bbl), (petroleum, 1 barrel=42 gal)	cubic meter (m³)	= 0.159 m³
	metric tonne (t)	= 0.159× (sp gr) ×t
	Coke in crude	
weight percent (wt%)	Conradson Carbon Residue (CCR)	= 1.6× (CCR)
	Gas-oil ratio	
cubic feet gas/barrel oil (ft³ gas/bbl oil)	cubic meters gas/cubic meter oil (m³ gas/m³ oil)	= 0.18× (m³gas/m³oil)
	Parts per million	
parts per million (ppm)	gram/metric tonne (g/t)	= g/t
	milligram/kilogram (mg/kg)	= mg/kg
	microgram/gram (µg/g)	= µg/g
	milligram/gram (mg/g)	= 0.001 mg/g
	weight percent (wt%)	= 0.0001 wt%
	Parts per billion	
parts per billion (ppb)	parts per million (ppm)	= 0.001 ppm
	Permeability	
millidarcy (md)	micrometer squared (µm²)	= 1,000 µm²
	Pressure	
pound per square inch (psi)	kilopascal (kPa)	= 6.89 kPA
	megapascal (Mpa)	= 0.00689 MPa
	bar	= 0.0689 bar
	kilograms/square centimeter (kg/cm²)	= 0.0703 kg/cm²
	Specific gravity (density)	
specific gravity (sp gr), (g/cm³)	°API (degrees)	= 141.5/(131.5+°API)
	Temperature	
degrees Fahrenheit (°F)	degrees Celsius (°C)	= (1.8×°C)+32
degrees Celsius (°C)	degrees Fahrenheit (°F)	= 0.556×(°F-32)
	Viscosity (absolute or dynamic)	
centipoise (cP)	Pascal second (Pa·s)	= 0.001 Pa·s
	millipascal second (mPa·s)	= mPa·s

Table 2. Conversion factors and equivalences applied to standardize data.—Continued

Standard unit in this report	Units as reported in literaure	Formula
	Viscosity (absolute or dynamic)—Continued	
centipoise (cP)—cont.	kinematic viscosity[1]: centistroke (cSt), (mm^2/sec)	= cSt × (sp gr)
	Saybolt Universal Seconds (SUS) at 100°F, for given density	= (SUS /4.632)× (sp gr)
	Saybolt Universal Seconds (SUS) at 100°F, for given °API	= (SUS /4.632)×(141.5/(131.5+°API))
	Weight percent	
weight percent (wt%)	parts per million (ppm)	= 10,000 ppm

[1] Kinematic viscosity is equal to the dynamic viscosity divided by the density of the fluid, so at 10°API the magnitudes of the two viscosities are equal.

Table 3. Total original in place resource calculation protocol when discovered oil in place is unavailable.

Define—

- OOIP-disc.: Original Oil In Place, discovered
- RF: Recovery factor (%)
- R: Reserves, known
- OR: Reserves, original sometimes called, known recovery, ultimate production if so reported
- AP: Production, annual
- CP: Production, cumulative
- PA: Prospective additional oil in place resource
- TOOIP = Total original oil in place

Calculations are based given data, which always receives priority; CP, AP and PA are never calculated and must be from published sources. (Assume CP, AP, PA are given)—

- R = 20×AP. This assumes a 20-year life or production plan for the viscous oil.
- OR = R+CP
- RF = 0.1 for clastic reservoirs or if no lithology is reported
- RF = 0.05 for carbonate reservoirs
- OOIP-disc. = OR/RF
- TOOIP = OOIP-disc. + PA

Table 4. Heavy oil and natural bitumen resources in billions of barrels of oil (BBO) and average characteristics of heavy oil and natural bitumen by basin type. Average values for gravity, viscosity, depth, thickness permeability are weighted by volume of oil in place discovered in each heavy oil or natural bitumen deposit by basin type; except for API gravity of heavy oil Type I, where because of relatively few deposits and several outlier values, a trimmed weighted mean value is shown.

[Volumes may not add to totals due to independent rounding; BBO, billions of barrels of oil; cP, centipoise]

Basin type	Total original oil in place (BBO)	Discovered oil in place (BBO)	API gravity (degrees)	Viscosity (cP @ 100°F)	Depth (feet)	Thickness (feet)	Porosity (percent)	Permeability (millidarcy)	Temperature (°F)
				Heavy oil					
I.....	3	2	15.9	724	1,455	11	15.3	88	122
IIA.....	158	157	16.3	321	4,696	36	22.8	819	102
IIB.....	181	181	17.7	303	3,335	96	27.2	341	82
IICa.....	1,610	1,582	15.5	344	3,286	150	24	242	144
IICb.....	32	32	15.4	318	3,976	161	16.9	2,384	126
IICc.....	460	460	17.8	455	6,472	379	19.6	1,080	159
IIIA.....	222	222	16.3	694	4,967	279	24.9	1,316	159
IIIBa.....	49	49	19.2	137	558	838	24.9	2,391	122
IIIBb.....	134	134	15.8	513	2,855	390	31.9	1,180	116
IIIBc.....	351	351	13.5	2,318	4,852	142	20.1	446	145
IIIC.....	158	158	17.2	962	7,227	273	25.1	868	159
IV.....	37	37	17.9	-	7,263	1,195	27.9	1,996	155
V.....	<1	<1	18	-	1,843	135	30	-	144
All types	3,396	3,366	16	641	4,213	205	23.7	621	134
				Natural bitumen					
I.....	60	8		-	20	317	5.5	100	
IIA.....	2,623	1,908	6.8	185,407	223	53	0.4	611	173
IIB.....	29	26	4.5	-		209	13.1	57	113
IICa.....	2,509	2,319	4.4	31,789	806	156	29.8	973	174
IICb.....	5	5	6.8	-	8,414	1,145	4.7	570	181
IICc.....	24	23	5	1,324	3,880	82	32.4	302	263
IIIA.....	22	22	8.7	-	4,667	882	30.3	1,373	85
IIIBa.....	4	4	-	-	-	-	-	-	-
IIIBb.....	3	3	6.7	500,659	3,097	586	28.6	2,211	89
IIIBc.....	178	178	9.5	1,322	8,751	52	34	751	139
IIIC.....	47	14	7.3	-	900	103	23.1	2,566	117
IV.....	0	0							
V.....	0	0							
All types	5,505	4,512	4.9	198,061	1,345	110	17.3	952	158

Table 5. Enhanced oil recovery (EOR) methods for heavy oil showing primary reservoir threshold criteria.

[modified from Taber, Martin, and Seright (1997a,b); cP, centipoises; PV, pore volume; ft, feet; md, millidarcy; °F, degrees Fahrenheit, wt%, weight percent]

Method	Gravity (°API)	Viscosity (cP)	Oil composition	Oil saturation (%PV)	Lithology	Net thickness (ft)	Average permeability (md)	Depth (ft)	Temperature (°F)
Immiscible gases									
Immiscible gases[a]	>12	<600	Not critical	>35	Not critical	Not critical	Not critical	>1,800	Not critical
Enhanced waterflood									
Polymer	>15	<150	Not critical	>50	Sandstone preferred	Not critical	>10[b]	<9,000	>200-140
Thermal/mechanical									
Combustion	>10	<5,000	Asphaltic components	>50	Highly porous sandstone	>10	>50[c]	<11,500	>100
Steam	>8	<200,000	Not critical	>40	Highly porous sandstone	>20	>200[d]	<4500	Not critical
Surface mining	>7	0 cold flow	Not critical	>8 wt% sand	Mineable oil sand	>10[e]	Not critical	>3:1 overburden: sand ratio	Not critical

[a] Includes immiscible carbon dioxide flood.

[b] >3 md for some carbonate reservoirs if the intent is to sweep only the fracture systems.

[c] Transmissibility > 20md-ft/cP.

[d] Transmissibility > 50md-ft/cP.

[e] See depth.

Table 6. Listing of countries reporting deposits of heavy oil and/or natural bitumen grouped by region. (See inset maps of regional distribution on Plates 2 and 3.)

North America	South America	Europe	Africa	Transcaucasia	Middle East	Russia	South Asia	East Asia	Southeast Asia and Oceania
Canada	Argentina	Albania	Algeria	Azerbaijan	Bahrain	Russia	Bangladesh	China	Australia
Mexico	Barbados	Austria	Angola	Georgia	Iran		India	Japan	Brunei
United States	Bolivia	Belarus	Cameroon	Kazakhstan	Iraq		Pakistan	Taiwan	Indonesia
	Brazil	Bosnia	Chad	Kyrgyzstan	Israel				Malaysia
	Colombia	Bulgaria	Congo (Brazzaville)	Tajikistan	Jordan				Myanmar
	Cuba	Croatia	Democratic Republic of Congo (Kinshasa)	Turkmenistan	Kuwait				Philippines
	Ecuador	Czech Republic	Egypt	Uzbekistan	Neutral Zone				Thailand
	Guatemala	France	Equatorial Guinea		Oman				Tonga
	Peru	Germany	Gabon		Qatar				Vietnam
	Suriname	Greece	Ghana		Saudi Arabia				
	Trinidad & Tobago	Hungary	Libya		Syria				
	Venezuela	Ireland	Madagascar		Turkey				
		Italy	Morocco		Yemen				
		Malta	Nigeria						
		Moldova	Senegal						
		Netherlands	South Africa						
		Norway	Sudan						
		Poland	Tunsia						
		Romania							
		Serbia							
		Slovakia							
		Spain							
		Sweden							
		Switzerland							
		Ukraine							
		United Kingdom							

Table 7. Attributes of Klemme basin types.

[Sources for attributes 1-15 are Klemme (1980a, 1980b, 1984) and attributes 16 and 17 are from this report]

	Type I	Type IIA	Type IIB	Type IICa
	Craton interior	**Continental multicycle basins, craton margin**	**Continental multicycle basins: craton/acreted zone rift-faulted**	**Continental interior multicycle basins: close collision zone at paleoplate margin**
1. Crustal zone	Continental craton	Continental craton	Contnental craton and accreted zone	Ocean crust early stages then continental crust of craton and accreted zone
2. Tectonic setting	Continenal crust within interior of craton, near or upon Precambrian sheld areas	Continental crust on exterior margin of craton, basins become multicylic ion Paleozoic or Mesozoic when a second cycle of sediments derived from uplife encroaches	Continental crust, or on margin of craton	Convergent margin along collision zone of paleoplates
3. Regional stress	Extensional	1st cycle: extension, 2nd cycle: compression	(1st) extension with rifting, (2nd) extensional sag	(1st) regional extension and platform deposits, then rifting, formation of linear sag, (2nd) compression with creation of foredeep
4. Basin size, shape	Large, circular to elongate	Moderate to large, circular to elongate	Large, circular	Large, elongate
5. Basin profile	Symmetrical	Asymmetrical	Irregular to asymmetrical	Asymmetrical
6. Sediment ratio[1]	Low	High	High	High
7. Architectural sequence	Sag	1st cycle: platform or sag, 2nd cycle: foredeep	(1st) rift, (2nd) large circular sag	(1st) platform or sag, (2nd) foredeep
8. Special features	Unconformities, regional arches, evaporite caps	Large traps, basins and arches, evaporite caps	Large traps, basins and arches, evaporite caps	Large traps and basins, evaporite caps, regional arches, regional source seal, fractured reservoirs
9. Basin lithology[2]	Clastic 60%, carbonate 40%	Clastic 75%, carbonate 25%	Clastic 75%, carbonate 25%	Clastic 35%, carbonate 65%
10. Depth of production[3]	Shallow	Shallow 55%, moderate 25%, deep 5%[5]	Shallow 55%, moderate 25%, deep 5%[5]	Shallow 45%, moderate 30%, deep 25%
11. Geothermal gradient	Low	Low	High	High
12. Temperature	Cool	Cool	Cool	High
13. Age	Paleozoic	Paleozoic, Mesozoic	Paleozoic, Mesozoic	Upper Paleozoic, Mesozoic, Tertiary
14. Oil and gas recovery[4]	Low, few giant fields	Average	Generally average	High
15. Traps	Associated with central arches and stratigraphic traps along basin margins	Basement uplifts, mostly arches or blocks	Basement uplifts, mostly combination of structural stratigraphic	Basement uplifts, arches and fault blocks
16. Propensity for heavy oil	Low	Low	Low	High
17. Propensity for natural bitumen	Low	High	Low	High

[1]Sediment ratio: ratio of sediment volume to basin surface area.

[2]Basin lithology: percentages apply to reservoir rocks, not to the basin fill.

[3]Depth of production: shallow, 0-6000 ft.; medium, 6000-9000 ft.; deep, >9000 ft.

[4]Oil and gas recovery (barrels of oil equivalent per cubic mile of sediment): low, <60,000; average, >=60,000 but <300,000; high, >=300,000.

[5]Does not add to 100% in source, Klemme (1980a,b).

Table 7. Attributes of Klemme basin types.—Continued

	Type IICb	Type IICc	Type IIIA	Type IIIBa
	Continental interior multicycle basins: foredeep portion of collision zone at paleoplate margin	**Continental interior multicycle basins: open collision zone at paleoplate margin**	**Continental rifted basins: craton/accreted zone, rift-faulted, with small linear sag**	**Continental rifted basins: back arc rift-faulted convergent margin**
1. Crustal zone	Ocean crust early stages then continental crust of craton and accreted zone	Ocean crust early stages then continental crust of craton and accreted zone	Continental craton and accreted zone	Contintental accreted zone with oceanic crust in early stages
2. Tectonic setting	Convergent margin along collision zone of paleoplates, but retain only proximal or foredeep portion of original sediment suite	Convergent margin along collision zone of paleoplates	Continental, on margin of craton. About two-thirds of Type IIIA basins form along trend of older deformation; remainder on Precambrian shields	Back arc basins along accreted zone of continent, with continental crust involved in later stages of development and ocean crust in the initial stages
3. Regional stress	(1st) regional extension and platform deposits, then rifting, formation of linear sag, (2nd) compression with creation of foredeep	(1st) regional extension and platform deposits, then rifting, formation of linear sag, (2nd) compression with creation of foredeep	(1st) extension with local wrench faulting during rifting, (2nd) sag	(1st) extension with local wrench faulting compression, (2nd) extension and compression
4. Basin size, shape	Large, elongate	Large, elongate	Small to moderate, fault controlled, elongate	Small, elongate
5. Basin profile	Asymmetrical	Asymmetrical	Irregular	Irregular
6. Sediment ratio[1]	High	High	High	High but variable
7. Architectural sequence	(1st) platform or sag, (2nd) foredeep	(1st) platform or sag, (2nd) foredeep	(1st) extension with local wrench faulting druing rifting, (2nd) sag	Rift faulting leading to linear sag, may be followd by wrench faulting
8. Special features	Large traps and basins, evaporite caps, regional arches, regional source seal, fractured reservoirs	Large traps and basins, evaporite caps, regional arches, regional source seal, fractured reservoirs, unconformities	Large traps, evaporite caps, unconformities, regional source seal	Large traps, and unconformities
9. Basin lithology[2]	Clastic 50%, carbonate 50%	Clastic 35%, carbonate 65%	Clastic 60%, carbonate 40%	Clastic 90%, carbonate 10%
10. Depth of production[3]	Shallow 45%, moderate 30%, deep 25%	Shallow 45%, moderate 30%, deep 25%	Moderate 55%, shallow 30%, deep 15%	Shallow 70%, moderate 20%, deep 10%
11. Geothermal gradient	High	High	High	High
12. Temperature	High	High	Normal to high	Normal to high
13. Age	Upper Paleozoic, Mesozoic, Tertiary	Upper Paleozoic, Mesozoic, Tertiary	Upper Paleozoic, Mesozoic, Paleogene, Neogene	Upper Mesozoic, Paleogene and Neogene
14. Oil and gas recovery[4]	Generally low	High	Generally high	Variable
15. Traps	Basement uplifts, arches and fault blocks	Basement uplifts, arches and fault blocks	Basement uplifts, combination structural/stratigraphic; result in fault block movement	Basement uplifts, fault blocks and combination
16. Propensity for heavy oil	Low	Moderate	Moderate	Low
17. Propensity for natural bitumen	Low	Low	Low	Low

Table 7. Attributes of Klemme basin types.—Continued

	Type IIIBb	Type IIIBc	Type IIIC	Type IV	Type V
	Continental rifted basins: transverse rift-faulted convergent margin	**Continental rifted basins: median rift-faulted convengent margin**	**Continental rifted basins: rift-faulted divergent margin, may be subdivided into (a) parallel, or (b) transverse basins**	**Deltas**	**Fore-arc basins**
1. Crustal zone	Contintental accreted zone with oceanic crust in early stages	Contintental accreted zone with oceanic crust in early stages	Ocean crust in early stage, then continental crust of craton and accreted zone	Ocean crust in early stage, then continental crust of craton and accreted zone	Continetal accreted crust and oceanic crust
2. Tectonic setting	Back arc basins along accreted zone of continent, with continental crust involved in later stages of development and ocean crust in the initial stages	Back arc basins along accreted zone of continent, with continental crust involved in later stages of development and ocean crust in the initial stages	Rift faulting along a divergent, passive or pull-apart continental margin	Almost any location: divergent and convergent margins along open or confined coastal areas	Fore-arc basins located on oceanward side of the volcanic arc in subduction or consumption zone
3. Regional stress	(1st) extension and wrench compression, (2nd) extension and compression	(1st) extension and wrench compression, (2nd) extension and compression	Extension leading to rift or wrench faulting	Extension as sag develops but uncertain as to the initial cause of sag, roots being deeply buried	Compression and extension
4. Basin size, shape	Small, elongate	Small, elongate	Small to moderate, elongate	Moderate, circular to elongate	Small, elongate
5. Basin profile	Irregular	Irregular	Asymmetrical	Depocenter	Asymmetrical
6. Sediment ratio[1]	High but variable	High but variable	High	Extremely high	High
7. Architectural sequence	Rift faulting leading to linear sag, may be followd by wrench faulting	Rift faulting leading to linear sag, may be followd by wrench faulting	Linear sage with irregular profile	Roots of deltas deeply buried; extension leads to half-sag with sedimentary fill thickening seaward.	Small linear troughs
8. Special features	Large traps, and unconformities	Large traps, unconformities, and regional arches	Possible unconformities and regional source seals	None	Large traps, and unconformities
9. Basin lithology[2]	Clastic 90%, carbonate 10%	Clastic 90%, carbonate 10%	Clastic 70%, carbonate 30%	Clastic 100%	Clastic 90%, carbonate 10%
10. Depth of production[3]	Shallow 70%, moderate 20%, deep 10%	Shallow 70%, moderate 20%, deep 10%	Deep 60%, moderate 30%, shallow 10%	Deep 65%, moderate 30%, shallow 5%	Shallow 70%, deep 20%, moderate 10%
11. Geothermal gradient	High	Normal to high	Low	Low	High
12. Temperature	Normal to high	Normal to high	Cool	Normal to low	High to normal
13. Age	Upper Mesozoic, Paleogene and Neogene	Upper Mesozoic, Paleogene and Neogene	Upper Mesozoic, Paleogene and Neogene	Paleogene, Neogene, and Quaternary	Upper Mesozoic, Tertiary
14. Oil and gas recovery[4]	Variable	Variable	Low	High	High but variable
15. Traps	Basement uplifts, fault blocks and combination	Basement uplifts, fault blocks and combination	Fault blocks and combination	Primarily tensional growth (roll-over) anticlines and flowage: basement not involved	Fault blocks and combination
16. Propensity for heavy oil	Low	Moderate	Low	Low	Nil
17. Propensity for natural bitumen	Low	Low	Low	Nil	Nil

Table 8. Regional distribution of heavy oil and natural bitumen (billion barrels).

[Volumes may not add to totals due to independent rounding]

Region[1]	Discovered orginal oil in place	Prospective additional	Total original oil in place
	Heavy oil		
North America	650	2	651
South America	1099	28	1127
Europe	75	0	75
Africa	83	0	83
Transcaucasia	52	0	52
Middle East	971	0	971
Russia	182	0	182
South Asia	18	0	18
East Asia	168	0	168
Southeast Asia and Oceania	68	0	68
Total	3366	29	3396
	Natural bitumen		
North America	1671	720	2391
South America	2070	190	2260
Europe	17	0	17
Africa	13	33	46
Transcaucasia	430	0	430
Middle East	0	0	0
Russia	296	51	347
South Asia	0	0	0
East Asia	10	0	10
Southeast Asia and Oceania	4	0	4
Total	4512	993	5505

[1] See table 6 for a list of countries reporting deposits of heavy oil and/or natural bitumen grouped by regions.

Appendixes 1–4

Appendix 1. Map Basin Name Conventions

Table 1-1. List of geologic provinces where province names used in this report differ from names used in St. John, Bally and Klemme (1984).

Geological province name in this report	Geological province name in St. John, Bally, and Klemme (1984)
Amu Darya	Tadzhik
Arkla	Louisiana Salt Dome
Baikal	Lake Baikal
Barinas-Apure	Llanos de Casanare
Carnarvon	Dampier
Central Montana Uplift	Crazy Mountains
Central Sumatra	Sumatra, Central
East Java	Java, East
East Texas	East Texas Salt Dome
Eastern Venezuela	Maturin
Forest City	Salina-Forest City
Gulf of Alaska	Alaska, Gulf of
Gulf of Suez	Suez, Gulf of
Guyana	Guiana
Junggar	Zhungeer
Kutei	Mahakam
Mae Fang	Fang
Minusinsk	Minisinsk
North Caspian	Caspian, North
North Caucasus-Mangyshlak	Caucasus, North
North Egypt	Western Desert
North Sakhalin	Sakhalin, North
North Sumatra	Sumatra, North
North Ustyurt	Ust Urt
Northern North Sea	North Sea, Northern
Northwest Argentina	Argentina, Northwest
Northwest German	German, Northwest
Northwest Shelf	Dampier
Ordos	Shanganning
Progreso	Guayaquil
Sacramento	Sacramento/San Joaquin
Salinas	Salinas (Mexico)
San Joaquin	Sacramento/San Joaquin
South Adriatic	Adriatic, South
South Palawan	Palawan, South
South Sumatra	Sumatra, South
Timan-Pechora	Pechora
Turpan	Tulufan

Table 1-1. List of geologic provinces where province names used in this report differ from names used in St. John, Bally and Klemme (1984).—Continued

Geological province name in this report	Geological province name in St. John, Bally, and Klemme (1984)
Upper Magdalena	Magdalena, Upper
West Java	Java, West, Sunda
West of Shetlands	Shetlands, West
Western Canada Sedimentary	Alberta
Yukon-Kandik	Yukon/Kandik

The following basins listed in bold type are from the digital mapping file of St. John (1996) and require further explanation:

- **Anadarko**: includes provinces more commonly known as the *Anadarko,* Central Kansas Uplift, Chautauqua Platform, *Las Animas Arch,* Nemaha Anticline-Cherokee Basin, *Ozark Uplift,* Sedgwick, and South Oklahoma Folded Belt (provinces in italics report neither heavy oil nor natural bitumen.)
- **Sacramento/San Joaquin**: separated into two distinct provinces, Sacramento and San Joaquin.
- **North Sea, Southern**: : includes both the Anglo-Dutch and Southern North Sea basins.
- **South Adriatic**: includes both the Durres and South Adriatic basins.

Other comments:

Three separate outlines for Marathon, Ouachita, and Eastern Overthrust are shown as a common province Marathon/Ouachita/Eastern Overthrust in the original St John (1996) but only Ouachita Basin had reported volumes of natural bitumen resources.

Deposits reported for Eastern Venezuela basin include deposits on the island of Trinidad, which are a likely extension of the rock formations from the surface expression of the basin outline.

The plates attach the name of Barinas Apure to the polygonal province labeled Llanos de Casanare in St. John (1996). Barinas Apure is the province name commonly used in Venezuela and Llanos de Casanare is the province name commonly used in Colombia for the same geologic province.

Appendix 2. Basins, Basin Type and Location of Basins having Heavy Oil and Natural Bitumen Deposits

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.

Geological province	Klemme basin type	Country	State/Province	Other names
Aegian	IIIBc	Greece		North Aegean Trough (North Aegean Sea Basin)
Akita	IIIBa	Japan		Akita Basin, Japan Accreted Arc/Accreted Terrane
Amu-Darya	IICa	Tajikistan, Uzbekistan		Tadzhik, Surkhan-Vaksh, Badkhyz High (Murgab Basin), Afghan-Tajik
Amur	IIIBc	Georgia		
Ana Maria	IIIBb	Cuba		Zaza Basin, Greater Antilles Deformed Belt
Anabar-Lena	IIA	Russia		
Anadarko	IIA	United States	Kans.	
Anadyr	IIIBb	Russia		
Angara-Lena	IIA	Russia		
Anglo-Dutch	IIB	Netherlands		Central Graben, North Sea, Southern
Appalachian	IIA	United States	Ky., N.Y.	
Aquitaine	IIIA	France		Ales, Aquitaine, Lac Basin, Parentis, Massif Central, Pyrenean Foothills-Ebro Basin
Arabian	IICa	Bahrain, Iran, Iraq, Jordan, Kuwait, Neutral Zone, Oman, Qatar, Saudi Arabia, Syria		Arabian Basin, Rub Al Khali, Aneh Graben, Aljafr Sub-basin, Oman Platform, Mesopotamian Foredeep, Palmyra Zone, Oman Sub-Basin, Euphrates/Mardin, Ghaba Salt Basin, Greater Ghawar Uplift, Haleb, Qatar Arch, South Oman Salt Basin, Widyan Basin
Arkla	IICc	United States	Ark., La.	Louisiana Salt Dome
Arkoma	IIA	United States	Ark., Okla.	
Assam	IICb	India		
Atlas	IICb	Algeria		Moroccan-Algerian-Tunisian Atlas, Hodna-Constantine
Bahia Sul	IIIC	Brazil		J Equitinhonha
Baikal	IIIA	Russia		Lake Baikal
Balearic	IIIA	Spain		Western Mediterranean, Gulf of Valencia, Barcelona Trough (Catalano-Balearic Basin), Iberic Cordillera
Baltic	I	Sweden		
Baluchistan	IICb	Pakistan		Sulaiman-Kirthar
Barbados	V	Barbados		Lesser Antilles, Northeast Caribbean Deformed Belt
Barinas-Apure	IIA	Venezuela, Colombia		Barinas-Apure Basin, Llanos de Casanare
Barito	IIIBa	Indonesia		Barito Basin
Bawean	IIIBa	Indonesia		
Beibu Gulf	IIIBa	China		Beibuwan (Gulf of Tonkin) Basin
Bengal	IICa	Bangladesh, India		Bengal (Surma Sub-basin), Tripura-Cachar, Barisal High (Bengal Basin), Ganges-Brahmaputra Delta
Beni	IIA	Bolivia		Foothill Belt
Big Horn	IIA	United States	Mont., Wyo.	
Black Mesa	IIB	United States	Ariz.	Dry Mesa, Dineh Bi Keyah
Black Warrior	IIA	United States	Ala., Miss.	
Bohai Gulf	IIIA	China		Bohai Wan (Huabei-Bohai) Basin, Huabei, Pohal, Luxi Jiaoliao Uplift

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.—Continued

Geological province	Klemme basin type	Country	State/Province	Other names
Bombay	IIIC	India		
Bonaparte Gulf	IIIC	Australia		Berkeley Platform (Bonaparte Basin)
Bone Gulf	IIIBa	Indonesia		Bone
Bresse	IIIA	France		Jura Foldbelt
Browse	IIIC	Australia		
Brunei-Sabah	IICc	Brunei, Malaysia		Baram Delta
Cabinda	IIIC	Angola, Congo (Brazzaville), Democratic Republic of Congo (Kinshasa)		Lower Congo Basin, West-Central Coastal
Caltanisetta	IICb	Italy, Malta		Caltanissetta Basin, Ibleian Platform, Sicilian Depression
Cambay	IIIA	India		Cambay North, Bikaner-Nagam, Bombay (in part)
Campeche	IICc	Mexico		Tabasco-Campeche, Yucatan Boderland and Platform, Tobasco, Campeche-Sigsbee Salt, Villahermosa Uplift
Campos	IIIC	Brazil		Cabo Frio High (Campos Basin)
Cantabrian	IIIA	Spain		Offshore Cantabrian Foldbelt (Cantabrian Zone), Spanish Trough-Cantabrian Zone
Carnarvon	IIIC	Australia		Dampier, Northwest Shelf, Carnarvon Offshore, Barrow-Dampier Sub-Basin
Carpathian	IICb	Austria, Czech Republic, Poland, Ukraine		Carpathian Flysch, Carpathian Foredeep, Bohemia, Carpathian-Balkanian
Celtic	IIIA	Ireland		Celtic Sea Graben System, Ireland-Scotland Platform
Central Coastal	IIIBb	United States	Calif.	Coastal, Santa Cruz, Salinas Valley, Northern Coast Range
Central Kansas Uplift	IIA	United States	Kans.	Anadarko
Central Montana Uplift	IIA	United States	Mont.	Crazy Mountains
Central Sumatra	IIIBa	Indonesia		Central Sumatra Basin
Ceram	IICa	Indonesia		North Seram Basin, Banda Arc
Channel Islands	IIIBb	United States		Southern California Borderlands
Chao Phraya	IIIA	Thailand		Phitsanulok Basin, Thailand Mesozoic Basin Belt
Chautauqua Platform	IIA	United States	Okla.	Anadarko
Cincinnati Arch	I	United States	Ky., Ohio	
Cook Inlet	IIIBa	United States	Alaska	Susitna Lowlands
Cuanza	IIIC	Angola		Kwanza Basin, West-Central Coastal
Cuyo	IIB	Argentina		Alvear Sub-basin (Cuyo Basin), Cuyo-Atuel
Dead Sea	IICa	Israel, Jordan		Syrian -African Arc, Levantine, Jafr-Tabuk, Sinai
Denver	I	United States	Colo., Nebr.	Denver-Julesberg
Diyarbakir	IICa	Syria, Turkey		Bozova-Mardin High (Southeast Turkey Fold Belt), Euphrates/Mardin, Zagros Fold Belt
Dnieper-Donets	IIIA	Ukraine		Dnepr-Donets Graben
Doba	IIIA	Chad		
Durres	IICb	Albania		Ionian Basin (zone), South Adriatic, Pre-Adriatic
East China	IIIBa	China, Taiwan		Diaoyu Island Depression (East China Sea Basin)
East Java	IIIBa	Indonesia		Bawean Arch (East Java Basin)
East Texas	IICc	United States	Tex.	East Texas Salt Dome, Ouachita Fold Belt
Eastern Venezuela	IICa	Venezuela, Trinidad and Tobago		Maturin, Eastern Venezuela Basin, Orinoco Oil Belt, Guarico Sub-basin, Trinidad-Tabago

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.—Continued

Geological province	Klemme basin type	Country	State/Province	Other names
Espirito-Santo	IIIC	Brazil		Abrolhos Bank Sub-Basin (Espirito Santo Basin)
Fergana	IIIBc	Kyrgyzstan, Tajikistan, Uzbekistan		
Florida-Bahama	IIIC	Cuba, United States	Fla.	Almendares-San Juan Zone, Bahia Honda Zone, Llasvvillas Zone, Florida Platform, Greater Antilles Deformed Belt
Forest City	I	United States	Kans., Nebr.	Salina-Forest City, Salina, Chadron Arch
Fort Worth	IIA	United States	Tex.	Bend Arch, Fort Worth Syncline, Llano Uplift, Ouachita Overthrust
Gabon	IIIC	Gabon		Gabon Coastal Basin (Ogooue Delta), West-Central Coastal
Gaziantep	IICa	Syria, Turkey		
Ghana	IIIC	Ghana, Nigeria		Benin-Dahomey, Dahomey Coastal
Gippsland	IIIA	Australia		Gippsland Basin
Green River	IIA	United States	Colo., Wyo.	
Guangxi-Guizou	IIB	China		Bose (Baise) Basin, South China Fold Belt
Gulf Coast	IICc	United States	La., Tex.	Mid-Gulf Coast, Ouachita Folded Belt, Burgos
Gulf of Alaska	V	United States	Alaska	
Gulf of Suez	IIIA	Egypt		Gulf of Suez Basin, Red Sea Basin
Guyana	IIIC	Suriname		Guiana, Bakhuis Horst, Guyana-Suriname
Illinois	I	United States	Ill., Ky.	
Indus	IICb	India		Punjab (Bikaner-Nagaur Sub-basin), West Rajasthan
Ionian	IICb	Greece		Epirus, Peloponesus
Irkutsk	IIA	Russia		
Jeanne d'Arc	IIIC	Canada	N.L.	Labrador-Newfoundland Shelf
Jianghan	IIIA	China		Tung-T'Ing Hu
Junggar	IIIA	China		Zhungeer, Anjihai-Qigu-Yaomashan Anticlinal Zone (Junggar)
Kansk	IIA	Russia		
Krishna	IIIC	India		Krishna-Godavari Basin
Kura	IIIBc	Azerbaijan, Georgia		Kura Basin
Kutei	IIIBa	Indonesia		Mahakam
Kuznets	IIB	Russia		
Laptev	IIB	Russia		
Los Angeles	IIIBb	United States	Calif.	
MacKenzie	IV	Canada	N.W.T.	Beaufort Sea, MacKenzie Delta
Mae Fang	IIIA	Thailand		Fang, Mae Fang Basin, Tenasserim-Shan
Maracaibo	IIIBc	Venezuela, Colombia		Maracaibo Basin, Catatumbo
Mauritius-Seychelles	IIIC	Seychelles		
Mekong	IIIC	Vietnam		Mekong Delta Basin
Michigan	I	United States	Mich.	
Middle Magdalena	IIIBc	Colombia		Middle Magdalena Basin
Minusinsk	IIB	Russia		Minisinsk
Mississippi Salt Dome	IICc	United States	Ala., Miss.	
Moesian	IICb	Bulgaria, Moldova, Romania		Moesian Platform-Lom Basin, Alexandria Rosiori Depression (Moesian Platform), Carpathian-Balkanian, West Black Sea

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.—Continued

Geological province	Klemme basin type	Country	State/Province	Other names
Molasse	IICb	Austria, Germany, Italy, Switzerland		Molasse Basin
Morondava	IIIC	Madagascar		
Mukalla	IIIC	Yemen		Sayhut Basin, Masila-Jeza
Natuna	IIIA	Indonesia		
Nemaha Anticline-Cherokee Basin	IIA	United States	Kans., Mo.	Anadarko
Neuquen	IIB	Argentina		Agrio Fold Belt (Neuquen Basin)
Niger Delta	IV	Cameroon, Equatorial Guinea, Nigeria		Abakaliki Uplift (Niger Delta)
Niigata	IIIBa	Japan		Niigata Basin, Yamagata Basin, Japan Volcanic Arc/Accreted Terrane
Nile Delta	IV	Egypt		Nile Delta Basin
North Caspian	IICa	Kazakhstan, Russia		Akatol' Uplift, Alim Basin, Beke-Bashkuduk Swell Pri-Caspian, Kobyskol' Uplift, South Emba, Tyub-Karagan
North Caucasus-Mangyshlak	IICa	Russia		Indolo-Kuban-Azov-Terek-Kuma Sub-basins, North Buzachi Arch, Middle Caspian, North Caucasus
North Egypt	IICa	Egypt		Western Desert, Abu Gharadiq
North Sakhalin	IIIBb	Russia		Sakhalin North
North Slope	IICc	United States	Alaska	Arctic Coastal Plains, Interior Lowlands, Northern Foothills, Southern Foothills, Colville
North Sumatra	IIIBa	Indonesia		North Sumatra Basin
North Ustyurt	IIB	Kazakhstan		Ust-Urt
Northern North Sea	IIIA	Norway, United Kingdom		Viking Graben, North Sea Graben
Northwest Argentina	IIA	Argentina		Carandaitycretaceous Basin
Northwest German	IIB	Germany		Jura Trough, West Holstein
Olenek	I	Russia		
Ordos	IIA	China		Shanganning, Qinling Dabieshan Fold Belt
Oriente	IIA	Peru		Acre, Maranon, Upper Amazon
Otway	IIIC	Australia		
Ouachita Overthrust	IIA	United States	Ark.	
Palo Duro	IIA	United States	N. Mex.	Tucumcari
Pannonian	IIIBc	Bosnia and Herzegovina, Croatia, Hungary, Romania, Serbia		Backa Sub-basin (Pannonian Basin)
Paradox	IIB	United States	Utah	
Paris	IIB	France		Anglo-Paris Basin
Pearl River	IIIC	China		Dongsha Uplift (Pearl River Basin), Pearl River Mouth, South China Continental Slope
Pelagian	IICa	Tunisia, Libya		
Permian	IIA	United States	N. Mex., Tex.	Ouachita Fold Belt, Bend Arch, Delaware, Midland
Peten-Chiapas	IICc	Guatemala		Chapayal (South Peten) Basin, North Peten (Paso Caballos), Sierra De Chiapas-Peten, Yucatan Platform
Piceance	IIA	United States	Colo.	
Po	IICb	Italy		Crema Sub-Basin (Po Basin)
Polish	IIIA	Poland		Danish-Polish Marginal Trough, German-Polish

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.—Continued

Geological province	Klemme basin type	Country	State/Province	Other names
Potiguar	IIIC	Brazil		Boa Vista Graben (Potiguar Basin), North-Northeastern Region
Potwar	IICb	Pakistan		Bannu Trough (Potwar Basin), Kohat-Potwar
Powder River	IIA	United States	Mont., Wyo.	
Pripyat	IIIA	Belarus		Pripyat Graben
Progreso	IIIBb	Ecuador		Guayaquil, Gulf Of Gayaquil, Jambeli Sub-basin of Progresso Basin, Santa Elena
Putumayo	IIA	Colombia, Ecuador		Napo, Cuenca Oriente Ecuatoriana
Rhine	IIIA	France, Germany		Upper Rhine Graben
Sacramento	IIIBb	United States	Calif.	Sacramento-San Joaquin
Salawati	IICa	Indonesia		Salawati Basin, Bintuni-Salawati
Salinas	IICc	Mexico		Isthmus Of Tehuantepec, Salinas Sub-basin, Isthmus Saline, Saline Comalcalco
San Joaquin	IIIBb	United States	Calif.	Sacramento-San Joaquin
San Jorge	IIIA	Argentina		Rio Mayo, San Jorge Basin
San Juan	IIB	United States	Ariz., Colo., N. Mex.	
Santa Maria	IIIBb	United States	Calif.	
Santos	IIIC	Brazil		
Sarawak	IICc	Malaysia		Central Luconia Platform
Sedgwick	IIA	United States	Kans.	Anadarko
Senegal	IIIC	Senegal		Bove-Senegal Basins
Sergipe-Alagoas	IIIC	Brazil		Sergipe-Alagoas Basin
Shumagin	V	United States	Alaska	
Sirte	IIIA	Libya		Agedabia Trough (Sirte Basin)
Songliao	IIIA	China		
South Adriatic	IICb	Italy		Adriatic, Marche-Abruzzi Basin (Pede-Apenninic Trough), Plio-Pleist Foredeep, Scaglia
South African	IIIC	South Africa		Agulhas Arch (South African Coastal Basin)
South Burma	IIIBb	Burma		Central Burma Basin, Irrawaddy
South Caspian	IIIBc	Azerbaidjan		South Caspian OGP (Apsheron-Kobystan Region), Emba, Guriy Region
South Oklahoma Folded Belt	IIA	United States	Okla., Tex.	Anadarko
South Palawan	IIIBa	Philippines		China Sea Platform, Palawan Shelf
South Sumatra	IIIBa	Indonesia		Central Palembang Depression (South Sumatra Basin)
South Texas Salt Dome	IICc	United States	Tex.	
South Yellow Sea	IIIA	China		Central Uplift (South Huanghai Basin), Subei Yellow Sea
Southern North Sea	IIB	United Kingdom		Central Graben (North Sea Graben system), Dutsh Bank Basin (East Shetland Platform), Witch Ground Graben
Sudan	IIIA	Sudan		Kosti Sub-Basin (Melut Basin), Muglad Basin, Sudd Basin
Sunda	IIIBa	Indonesia		
Surat	IIB	Australia		
Sverdrup	IICc	Canada	N.W.T.	Mellville
Taiwan	IIIBa	Taiwan		Taihsi Basin

Table 2-1. List of geological basin names, the Klemme basin type, countries, U.S. states or Canadian provinces reporting deposits of heavy oil and/or natural bitumen, and other names cited in literature.—Continued

Geological province	Klemme basin type	Country	State/Province	Other names
Talara	IIIBb	Peru		Talara Basin
Tampico	IICc	Mexico		Tampico-Tuxpan Embayment, Chicontepec, Tampico-Misantla
Tarakan	IIIBa	Indonesia		Bera Sub-basin (Tarakan Basin), Pamusian-Tarakan
Taranto	IICb	Italy		Abruzzi Zone (Apennine Range). Marche-Abruzzi Basin (Pede-Apenninic Trough), Latium, Calabrian
Tarfaya	IIIC	Morocco		Aaiun-Tarfaya
Tarim	IIIA	China		
Thrace	IIIBc	Turkey		Thrace-Gallipoli Basin, Zagros Fold Belt
Timan-Pechora	IIB	Russia		Belaya Depression (Ural Foredeep), Brykalan Depression, Pechora-Kozhva Mega-Arch, Varendey-Adz'va
Timimoun	IIB	Algeria		Sbaa
Tonga	IIIBa	Tonga		
Tunguska	I	Russia		Baykit Antecline
Turpan	IIIA	China		Tulufan
Tyrrhenian	IIIA	Italy		
Uinta	IIA	United States	Utah	
Upper Magdalena	IIIBc	Colombia		Upper Magdalena Basin
Ventura	IIIBb	United States	Calif.	Santa Barbara Channel
Veracruz	IIIC	Mexico		
Verkhoyansk	IIA	Russia		
Vienna	IIIBc	Austria, Slovakia		Bohemia
Vilyuy	IIA	Russia		
Volga-Ural	IIA	Russia		Aksubayevo-Nurlaty Structural Zone, Bashkir Arch, Belaya Depression, Melekess Basin, Tatar Arch, Vishnevo-Polyana Terrace
Washakie	IIA	United States	Wyo.	
West Java	IIIBa	Indonesia		Arjuna Sub-Basin (West Java Basin), Northwest Java
West of Shetlands	IIIC	United Kingdom		Faeroe, West of Shetland
West Siberia	IIB	Russia		West Siberia
Western Canada Sedimentary	IIA	Canada, United States	Alta., Mont., Sask.	Alberta, Western Canada Sedimentary, Sweetgrass Arch
Western Overthrust	IIA	United States	Ariz., Mont., Nev., Utah	Central Western Overthrust, Great Basin Province, Southwest Wyoming, South Western Overthrust
Williston	I	Canada, United States	N. Dak., Sask.	Sioux Uplift
Wind River	IIA	United States	Wyo.	
Yari	IIA	Colombia		Yari Basin
Yenisey-Khatanga	IIA	Russia		
Yukon-Kandik	IIIBb	United States	Alaska	Yukon-Koyukuk
Zagros	IICa	Iran, Iraq		Zagros Fold Beltzagros or Iranian Fold Belt, Sinjar Trough, Bozova-Mardin High, Euphrates/Mardin

Appendix 3. Klemme Basin Classificaton Figure from Plate 1

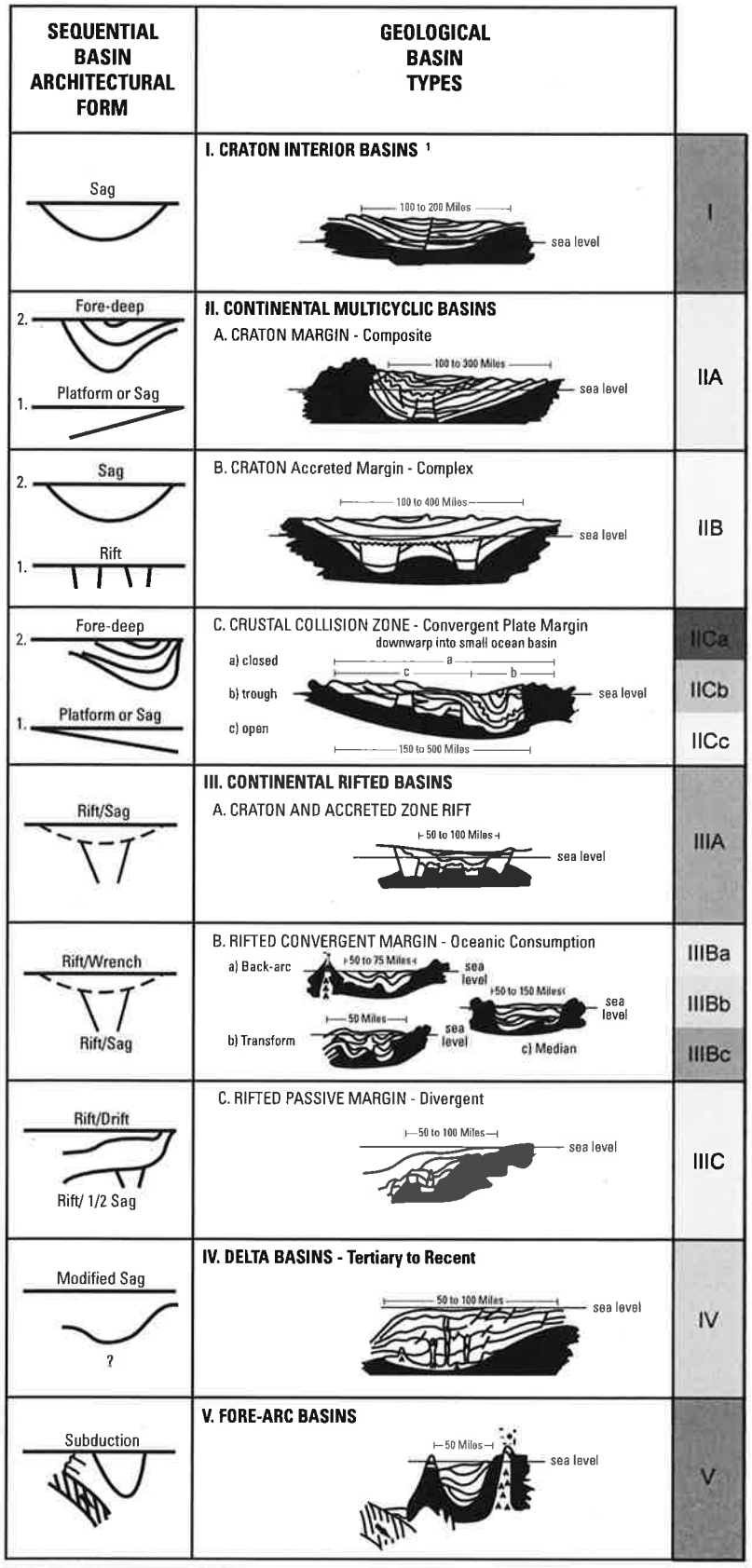

Figure 2-1. Diagram of Klemme basin types from plate 1. Modified from St. John, Bally, and Klemme (1984).

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[1] Cincinati Arch is classified as I-Arch

Appendix 4. Tables from the Plates

Table 4-1. 50 heavy oil basins ranked by volumes of total original heavy oil in place (TOHOIP), showing natural bitumen volumes where reported. Table repeated from plate 2.

[billions of barrels, BBO, 10^9 barrels]

Rank	Geological province	Klemme basin type	Total original heavy oil in place	Original heavy oil in place-discovered	Prospective additional heavy oil in place	Total original natural bitumen in place	Original natural bitumen in place-discovered	Prospective additional natural bitumen in place
1	Arabian	IICa	842	842				
2	Eastern Venezuela	IICa	593	566	27.7	2,090	1,900	190
3	Maracaibo	IIIBc	322	322		169	169	
4	Campeche	IICc	293	293		0.060	0.060	
5	Bohai Gulf	IIIA	141	141		7.63	7.63	
6	Zagros	IICa	115	115				
7	Campos	IIIC	105	105				
8	West Siberia	IIB	88.4	88.4				
9	Tampico	IICc	65.3	65.3				
10	Western Canada Sedimentary	IIA	54.9	54.9		2,330	1,630	703
11	Timan-Pechora	IIB	54.9	54.9		22.0	22.0	
12	San Joaquin	IIIBb	53.9	53.9		< 0.01	< 0.01	
13	Putumayo	IIA	42.4	42.4		0.919	0.919	
14	Central Sumatra	IIIBa	40.6	40.6				
15	North Slope	IICc	37.0	37.0		19.0	19.0	
16	Niger Delta	IV	36.1	36.1				
17	Los Angeles	IIIBb	33.4	33.4		< 0.01	< 0.01	< 0.01
18	North Caspian	IICa	31.9	31.9		421	421	
19	Volga-Ural	IIA	26.1	26.1		263	263	
20	Ventura	IIIBb	25.2	25.2		0.505	0.505	
21	Gulf of Suez	IIIA	24.7	24.7		0.500	0.500	
22	Northern North Sea	IIIA	22.8	22.8		10.9	10.9	
23	Gulf Coast	IICc	19.7	19.7				
24	Salinas	IICc	16.6	16.6				
25	Middle Magdalena	IIIBc	16.4	16.4				
26	Pearl River	IIIC	15.7	15.7				
27	North Ustyurt	IIB	15.0	15.0				
28	Brunei-Sabah	IICc	14.7	14.7				
29	Diyarbakir	IICa	13.5	13.5				

Table 4-1. 50 heavy oil basins ranked by volumes of total original heavy oil in place (TOHOIP), showing natural bitumen volumes where reported. Table repeated from plate 2.—Continued

[billions of barrels, BBO, 10^9 barrels]

Rank	Geological province	Klemme basin type	Total original heavy oil in place	Original heavy oil in place-discovered	Prospective additional heavy oil in place	Total original natural bitumen in place	Original natural bitumen in place-discovered	Prospective additional natural bitumen in place
30	Northwest German	IIB	9.48	9.48				
31	Barinas-Apure	IIA	9.19	9.19		0.38	0.38	
32	North Caucasus-Mangyshlak	IICa	8.60	8.60		0.060	0.060	
33	Cambay	IIIA	8.28	8.28				
34	Santa Maria	IIIBb	8.06	8.06		2.03	2.02	< 0.01
35	Central Coastal	IIIBb	8.01	8.01		0.095	0.025	0.070
36	Big Horn	IIA	7.78	7.78				
37	Arkla	IICc	7.67	7.67				
38	Moesian	IICb	7.39	7.39				
39	Assam	IICb	6.16	6.16				
40	Oriente	IIA	5.92	5.92		0.250	0.250	
41	Molasse	IICb	5.79	5.79		0.010	0.010	
42	Doba	IIIA	5.35	5.35				
43	Morondava	IIIC	4.75	4.75		2.21	2.21	
44	Florida-Bahama	IIIC	4.75	4.75		0.48	0.48	
45	Southern North Sea	IIB	4.71	4.71				
46	Durres	IICb	4.70	4.70		0.37	0.37	
47	Caltanisetta	IICb	4.65	4.65		4.03	4.03	
48	Neuquen	IIB	4.56	4.56				
49	North Sakhalin	IIIBb	4.46	4.46		< 0.01	< 0.01	
50	Cabinda	IIIC	4.43	4.43		0.363	0.363	

Table 4-2. 33 natural bitumen basins ranked by volumes of total original natural bitumen in place (TONBIP). Table repeated from plate 3.

[billions of barrels, BBO, 10^9 barrels]

Rank	Geological province	Klemme basin type	Total original natural bitumen in place	Original natural bitumen in place-discovered	Prospective additional natural bitumen in place
1	Western Canada Sedimentary	IIA	2,330	1,630	703
2	Eastern Venezuela	IICa	2,090	1,900	190
3	North Caspian	IICa	421	421	
4	Volga-Ural	IIA	263	263	
5	Maracaibo	IIIBc	169	169	
6	Tunguska	I	59.5	8.19	51.3
7	Ghana	IIIC	38.3	5.74	32.6
8	Timan-Pechora	IIB	22.0	22.0	
9	North Slope	IICc	19..0	19.0	
10	Uinta	IIA	11.7	7.08	4.58
11	Northern North Sea	IIIA	10.9	10.9	
12	South Caspian	IIIBc	8.84	8.84	
13	Bohai Gulf	IIIA	7.63	7.63	
14	Paradox	IIB	6.62	4.26	2.36
15	Black Warrior	IIA	6.36	1.76	
16	South Texas Salt Dome	IICc	4.88	3.87	1.01
17	Cuanza	IIIC	4.65	4.65	
18	Bone Gulf	IIIBa	4.46	4.46	
19	Caltanisetta	IICb	4.03	4.03	
20	Nemaha Anticline-Cherokee Basin	IIA	2.95	0.70	2.25
21	Morondava	IIIC	2.21	2.21	
22	Yenisey-Khatanga	IIA	2.21	2.21	
23	Santa Maria	IIIBb	2.03	2.02	<0.01
24	Junggar	IIIA	1.59	1.59	
25	Tarim	IIIA	1.25	1.25	
26	West of Shetlands	IIIC	1.00	1.00	
27	Putumayo	IIA	0.919	0.919	
28	Illinois	I	0.890	0.300	0.590
29	South Oklahoma Folded Belt	IIA	0.885	0.058	0.827
30	South Adriatic	IICb	0.510	0.510	
31	Ventura	IIIBb	0.505	0.505	
32	Gulf of Suez	IIIA	0.500	0.500	
33	Florida-Bahama	IIIC	0.477	0.477	

Atomic and Nuclear Methods in Fossil Energy Research

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PREFACE

The increased demand on fossil fuels for energy production has resulted in expanded research and development efforts on direct use of fossil fuels and conversion of fossil fuels into synthetic fuels. These efforts have focused on the efficiency of the energy production and/or conversion processes, and of the emission control technology, as well as delineation of the health and environmental impacts of those processes and their by-products. A key ingredient of these studies is the analytical capability necessary to identify and quantify those chemicals of interest in the process and by-produce streams from coal combustion, oil shale retorting, petroleum refining, coal liquifaction and gasification. These capabilities are needed to analyze a formidable range of materials including liquids, solids, gases and aerosols containing large numbers of criteria and pollutants including potentially hazardous polynuclear aromatic hydrocarbons, organo-sulfur and organo-nitrogen species, trace elements and heavy metals, among others.

Taking notice of these developments we sought to provide a forum to discuss the latest information on new and novel applications of a subset of those necessary analytical capabilities, namely atomic and nuclear techniques. Consequently, we organized the conference on Atomic and Nuclear Methods in Fossil Fuel Energy Research, which was held in Mayaguez, Puerto Rico from December 1 to December 4, 1980.

The wide range of atomic and nuclear techniques which were discussed in the invited and contributed papers included:

neutron activation analysis
plasma emission spectroscopy
ion selection electrodes
atomic absorption spectroscopy
hydride generation
fission track etching
x-ray fluorescence analysis
Rutherford backscattering
gas chromatography

mass spectroscopy
photoelectron spectroscopy
gamma-ray spectroscopy
radioactive tracer studies
electron microprobe analysis
nuclear magnetic resonance spectroscopy

These papers were arranged into sessions according to the following major problem areas:

methods of elemental analysis
investigations of mechanisms and structures
characterization of species

The problem areas were keynoted by excellent plenary state-of-the-art review papers given by J. W. Fruchter, W. D. Ehmann, R. A. Cahill, H. D. Buenafama, R. B. Finkelman, B. R. T. Simoneit, O. P. Strauz, H. L. Retcofsky, T. Novakov, R. P. Skowronski, J. R. Speight and C. B. Cecil, well-known authorities in their fields. The contributed papers followed by presenting recent experimental results in those areas reviewed in the plenary papers.

This conference provided an unusual opportunity for attendees with expertise and interests in the diverse areas of atomic analytical techniques and nuclear analytical techniques to communicate together and discuss topics of mutual interest. We feel these discussions were very fruitful and educational for all the attendees and will lead to beneficial collaborations in the future.

The Atomic and Nuclear Methods in Fossil Energy Research Topical Conference was co-sponsored by the American Nuclear Society, Isotopes and Radiation Division; the American Chemical Society, Divisions of Analytical Chemistry, Nuclear Chemistry and Technology, Petroleum Chemistry, and Fuel Chemistry; U.S. Department of Energy, Center for Energy and Environment Research; and University of Puerto Rico - Mayaguez Campus. The organizing committee for this conference was composed of R. H. Filby, General Chairman; B. S. Carpenter and R. C. Ragaini, Technical Co-chairmen; J. W. Morgan, K. B. Pedersen, E. Ricci, R. E. Sinclair, J. R. Vogt, and N. A. Wogman, members of the Technical Committee.

The editors would like to acknowledge the unselfish giving of time and talent of those whose help made this conference a success. We wish to express our added appreciation to the session chairmen for their overall contributions: J. R. Vogt, B. S. Carpenter, H. D. Buenafama, R. C. Ragaini, J. S. Fruchter, R. H. Filby, and R. P. Skowronski. We thank the following for their various support roles so important to the success of this conference: J. R. Hiegel and D. Price of the National Bureau of Standards; R. Farmakes and S. H. Krapp of the American Nuclear Society; and S. Shives and E. Macpher-

son of Washington State University. A special thanks is extended to S. Hendrickson of Lawrence Livermore National Laboratory for the compilation and preparation of the abstracts.

Although proper prearrangements are critical to any meeting, implementation is equally critical. For the invaluable on-site assistance we received during the various phases of the conference, we are indebted to many, but especially to K. B. Pedersen for arranging so much of the local assistance. We are indebeted specifically to the staff of the Center for Energy and Environment Research; S. E. Alemany, Chancellor, University of Puerto Rico - Mayaguez Campus; and M. Vargas-Cesani, Recinto Universitario de Mayaguez. We wish to express our appreciation to D. Bennett, C. Filby and N. Wogman for coordination of all the last minute crises, both before and during the meeting. Finally, we would like to thank R. E. Sinclair, Applied Physics Technology, for organizing the coffee breaks which were sponsored by Canberra, ORTEC, Nicolet, and Nuclear Data.

The location of the conference, Mayaguez, Puerto Rico, not only provided an excellent secluded atmosphere for informal working groups to be formed, but also was an equally enjoyable and superb setting for those who accompanied the attendees. The location was surpassed only by the cordiality of the hosts, J. Guzman, H. J. Santiago, J. A. Petrillo and staff, at the Mayaguez Hilton, the conference hotel. Competing with scenery and cordiality of the hotel was the exquisite cuisine prepared by D. Hannig, the hotel's chef.

The editors wish to thank the authors, who supplied manuscripts for all the papers included in these proceedings. We also extend our appreciation to J. Busis, Plenum Press, for the aid in publishing these proceedings. Finally, we would like to extend a "thank you" to each and every person connected with the conference for a job well done.

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CHANGING REFINERY CONFIGURATION FOR HEAVY AND SYNTHETIC CRUDE PROCESSING

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INTRODUCTION

Reduced availability of light conventional crudes in the future will create demand for new crude sources that will necessitate refinery configuration changes. The production of heavy crudes, synthetic crudes, and bitumen blends is growing, and the supply of bitumen-derived crudes is expected to reach almost three million barrels per day by the year 2015[1]. A plethora of synthetic crudes and bitumen blends have become available, all of which pose different challenges for today's refiners. Some crudes are both higher in contaminant levels and have a composition that makes them more difficult to upgrade. Coupled with the demand for increased production of ultra-clean diesel and gasoline, innovative refinery configuration changes will be needed to accommodate these new feedstocks. The potential processing schemes under consideration range from simple hydrotreating for contaminant removal, to hydrocracking and fluid catalytic cracking for conversion of gas oil to high-quality transportation fuels. It is the integration of these process technologies, however, that offers the greatest economic potential. This paper focuses on the processing of heavy and synthetic crude blends using innovative process integration across several technology platforms to produce clean fuels.

MARKET SITUATION

World oil demand is projected to continue increasing, at a rate of about 1.5% per year, with increased growth of transportation fuels coupled with a relatively flat heavy oil demand. The Energy Information Administration recently predicted that the demand for crude oil in the United States will increase at an average rate of 1.1% through to the year 2030[2]. Most of the 400,000-barrel annual increase in crude consumption in the United States will be used for transportation fuels, with gasoline representing about 45% of total petroleum consumption. The demand for distillate fuels is growing at a faster rate than the demand for gasoline. These changes in demand

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have been accompanied by well-known ultra-low-sulfur regulations for diesel and gasoline, forcing a significant capital investment through the end of decade.

The production of light and medium crudes from the Western Canadian sedimentary basin is dropping by about 20% every five years. Given the increasing demand for oil, and declining production of conventional crudes in both the United States and Canada, the Canadian oil sands will become a key source of future crude supply. The volume of oil in place in the various deposits is estimated at 1.7 trillion barrels, of which 174 billion barrels is recoverable with existing technology. This places the size of the established reserves second only to Saudi Arabia. As recovery technologies improve, the size of those recoverable reserves could increase significantly. The proximity of the source, security of supply, and competitive pricing will drive the refinery investment needed to accommodate these crudes.

Today, the level of production has reached one million barrels per day, and it is expected to increase to almost three million barrels per day by 2015. Oil sands crudes are expected to represent more than 75% of the crude produced in Western Canada. While most of the imports into the United States are in PADDs II and IV, further pipeline expansions will increase penetration in PADD II and the Pacific Northwest (Northern PADD V). New pipeline systems are expected in the future, reaching Texas (PADD III) and a new sea port in British Columbia. From this new port, marine shipments to both California (Southern PADD V) and the Far East are expected.

As will be shown later, the composition and contaminant levels of bitumen-derived crudes does not make them an easy replacement for conventional crudes, especially since most existing refineries have limited capacity to accept poorer quality feedstocks. These crudes are fundamentally different, so refiners need to understand them and also be prepared for the changes needed to process them.

Bitumen-derived Crudes

The term "synthetic crude" has never been strictly defined, but it has come to mean a blend of naphtha, distillate, and gas oil range materials, with no resid (1050°F+, 565°C+ material). Canadian synthetic crudes first became available in 1967 when Suncor (then Great Canadian Oil Sands) started to market a blend produced by hydrotreating the naphtha, distillate, and gas oil generated in a delayed coking unit. The light, sweet synthetic crude marketed by Suncor today is called Suncor Oil Sands Blend A (OSA). Syncrude Canada Ltd. started production in 1978, marketing a fully-hydrotreated blend utilizing fluidized-bed coking technology as the primary upgrading step. Today, this product is referred to as Syncrude Sweet Blend (SSB). Husky Oil started up a heavy conventional crude upgrader in 1990 using a combination of ebullated-bed hydroprocessing and delayed coking technologies. Their sweet synthetic crude is traded as Husky Sweet Blend (HSB). The Athabasca Oils Sands Project (AOSP) started producing a sweet synthetic crude in 2003 called Premium Albian Synthetic (PAS) using ebullated-bed hydroprocessing technology. There are also small volumes of two synthetic crudes produced at the Consumers' Co-op refinery called NSA and NSB.

The quality of the kerosene and diesel in these synthetic crude blends has been a major concern in the past. Bitumen is itself a very aromatic feed, and the choice of primary upgrading

technology has an effect on the final distillate quality. Husky's HSB and AOSP's PAS crudes are produced using an ebullated-bed resid hydrocracking technology, and hence have full-range diesel cetane numbers above 40. Suncor's OSA crude is produced using delayed coking, giving the full-range diesel a cetane number very close to, or just below 40. Syncrude's SSB crude is produced using the more-severe fluidized-bed coking technology, and the diesel has a cetane number of about 33. The kerosene cut of SSB has a smoke point of just 13 mm. Starting in June of 2006, Syncrude will start to produce Syncrude Sweet Premium (SSP), where the distillate has been further upgraded to give a full-range diesel cetane number of 40, and a kerosene smoke point of 19 mm.

Table 1 compares the basic composition and quality of Syncrude SSB against Brent crude[3]. The SSB crude has lower sulfur and no resid (as shipped), it contains significantly less naphtha-range material, and more distillate and VGO. Note that due to pipeline contamination, synthetic crudes like SSB can have some resid component when actually received in the refinery.

Table 1 – Comparison of Syncrude SSB and Brent Crudes

	Brent	*SSB Produced*	*SSB Received**
Gravity, API	**38.6**	**31.8**	**33.5**
Sulphur, wt%	**0.29**	**0.1**	**0.2**
RVP, psi	**8.2**	**4.6**	**N/A**
Yield, vol%			
C_4-	**2.9**	**3.4**	**N/A**
C_5- 350°F	**28.6**	**15**	**21**
350 - 650°F	**29.6**	**44**	**40.5**
650 - 350°F	**29.8**	**37.6**	**31.5**
1050°F plus	**9.1**	**0**	**3**
Properties			
Kerosene smoke pt, mm 350 - 650°F	**25**	**13**	**---**
Cetane	**50**	**30**	**---**
Pour	**-5**	**-55**	**---**

While sweet synthetic blends make up the majority of the synthetic crudes on the market, some sour synthetic blends are also available. Suncor markets a range of sour synthetic blends, each tailored to meet specific refinery processing capabilities. Suncor OSE crude is a blend of hydrotreated coker naphtha with non-hydrotreated coker distillate and coker heavy gas oil. Suncor's OSV crude is a blend of hydrotreated coker naphtha with straight-run distillate and straight-run VGO. There are several other sour blends available (OSH, OCC, etc), each with its own processing characteristics. While these sour crude blends still contain no resid fraction, they are generally sold to medium and heavy sour crude refineries. Note that AOSP markets a heavy sour blend called Albian Heavy Synthetic (AHS) which is a blend of their sweet PAS crude with

unconverted oil from their ebullated-bed resid hydrocracking unit. Since this crude does have a resid component, it is not really a synthetic crude as it has been defined here, but neither is it a bitumen blend, like those described below.

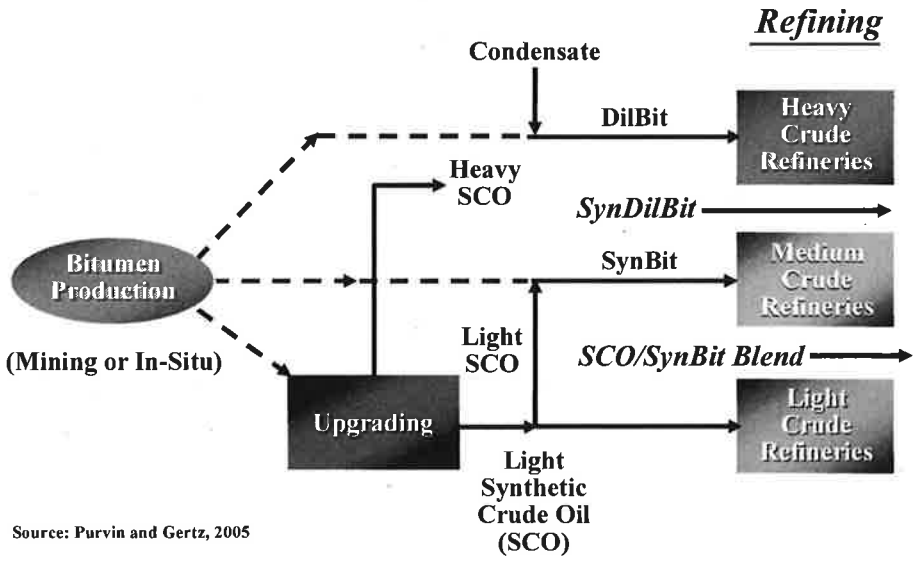
Together, the various synthetic crudes make up the majority of the bitumen-derived crudes on the market today. However, over 400 KB/D of bitumen are produced and shipped to market without having been upgraded. The bitumen must be diluted with a lighter hydrocarbon stream to meet the specifications required for shipping in pipelines. DilBits are blends of bitumen and condensate, typically natural gas condensate. They normally contain 25 – 30 lv% condensate and 70 – 75 lv% bitumen. The most common streams are Cold Lake Blend (CLB), Bow River (BRH), and various Lloyd blends (LLB, LLK, WCB). Since the majority of condensate is C_5 to C_{12} material, and the majority of bitumen is C_{30}+ boiling range material, these crudes have become known as "dumbbell crudes." There is a lot of material boiling at each end of the boiling point curve, but little in the middle.

Natural gas condensate is in short supply in Northern Alberta where the bitumen is produced. Condensate sells at a significant premium to light sweet crudes for this reason, and some condensate is actually being shipped by rail back to Alberta from the United States. To address the shortage of diluent, and the problem with dumbbell crudes, producers have started to market SynBits, blends of sweet synthetic crude (typically OSA) and bitumen. SynBits have a more continuous boiling point curve than DilBits, with a significant portion of distillate-range material in the blend. However, since the synthetic crude diluent has a much lower API gravity than condensate, more diluent is needed, so SynBits are typically 50 lv% synthetic crude and 50 lv% bitumen. The most common SynBits on the market today are Christina Lake Blend (CSB) and MacKay Heavy (MKH), both of which are blends of bitumen produced by Steam Assisted Gravity Drainage (SAGD) and OSA crudes.

SynDilBits are actually blends of condensate, hydrotreated synthetic crude, and bitumen. They typically contain about 65 lv% bitumen, with the remaining volume split between the two diluent streams. The most common of these streams are Wabasca Heavy (WH) and Western Canadian Select (WCS).

Light sweet synthetic crudes, heavy sour synthetic crudes, DilBits, SynBits, and SynDilBits all target different refineries. Figure 1[4] on the next page summarizes how bitumen is upgraded, or blended with other streams to make various types of crude blends. Light sweet synthetic crudes are usually sold to light crude refineries, while heavy sour synthetic crudes and DilBits are normally processed in heavy crude refineries. SynBits and SynDilBits are usually sold to medium crude refineries, or blended with additional synthetic crude for processing in a light crude refinery.

Figure 1 – Disposition of Bitumen-derived Crudes

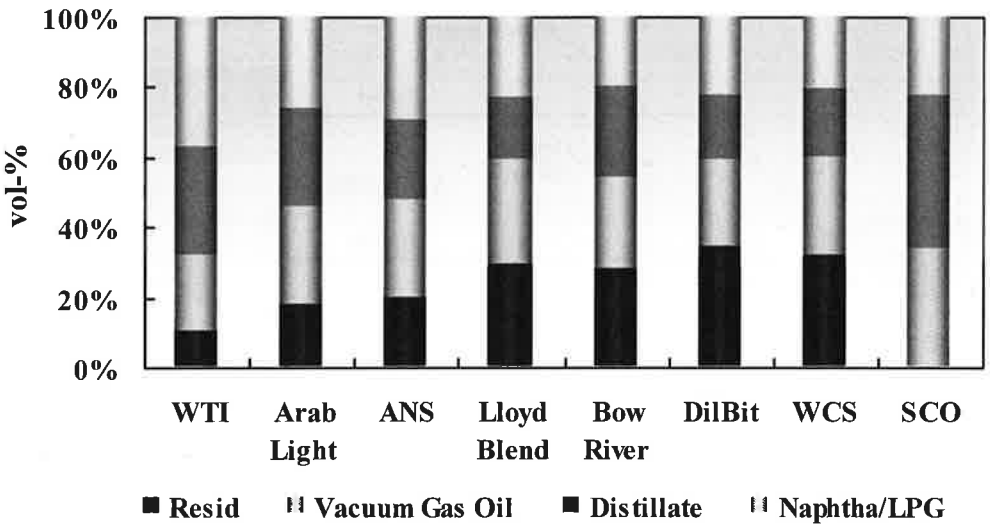


WCS is a somewhat unique SynDilBit in that it has a proprietary formula developed by EnCana, Talisman, Canadian Natural Resources Limited (CNRL), and Petro-Canada. They wanted to reduce the large number of heavy crudes being marketed from Western Canada, and achieve consistency in the heavy crude blends being shipped from Canada. Each batch contains specified amounts of the following crudes; LLW, LLC, CLB, CSB, MKH, and BR[6]. As such, each batch contains condensate, hydrotreated synthetic crude (OSA), heavy conventional crude, medium conventional crude, Cold Lake bitumen, and Athabasca bitumen. Each batch is blended to meet the following specifications; API gravity of 19 - 22°, carbon residue of 7 – 9 wt%, sulfur of 2.8 – 3.2 wt%, and a total acid number (TAN) of 0.7 – 1.0 mg KOH/g.

WCS may well become the new marker heavy crude from Western Canada, and efforts are being made to have it traded on the New York Commodities Exchange. Production of WCS started in January of 2005, with shipments currently at 250 KB/D, but expected to increase to more than 500 KB/D by 2008.

Each synthetic crude and bitumen blend has its own unique processing characteristics. The compositions of three conventional light crudes, two heavy conventional crudes, two bitumen blends, and one synthetic crude are compared and contrasted in Figure 2. Compared to the marker West Texas Intermediate (WTI) crude, a typical synthetic crude has no resid, 50% more VGO, 50% more distillate, and only half the naphtha. DilBits and WCS have about three times the volume of resid material than WTI, 50% more VGO, but only half the distillate range material, and half the naphtha.

Figure 2 – Crude Composition



PROCESSING IMPLICATIONS

The processing implications to the refinery will be a function of the type of bitumen-derived crude imported. First and foremost, refiners used to processing WTI will have a three-fold increase in the amount of resid coming to the refinery if they replace WTI with WCS. Beyond the implications to the crude and vacuum unit, extra resid conversion capacity will certainly be required. Refineries already seem to be anticipating this need. There is about 200 KB/D of incremental delayed coking capacity in various stages of planning in PADD II, PADD IV, and northern PADD V[1]. The addition of coking capacity can bring a new set of issues to these refiners. Not only must the refinery deal with the coke disposal issue, they must deal with the cracked products a coker generates. Coker naphtha typically has a high sulfur and nitrogen content, but is also rich in olefins and diolefins. Coker naphtha cannot simply be added to the straight-run feed to the naphtha hydrotreater protecting the catalytic reforming unit. The coker distillate is also high in sulfur and nitrogen, and has a very low cetane number due to its high aromatic content. The heavy gas oil produced in a coker from bitumen has a particularly high aromatic content, and therefore makes a poor FCC feedstock. Each of these streams will also contain differing levels of silica from the antifoam agent used in the coke drums which can poison the catalyst in downstream hydroprocessing units. Coupled with the high VGO component of all synthetic crudes and bitumen blends, there is a twofold impact on the FCC unit. Higher throughputs are required to process the additional VGO, and the feedstock is poor quality.

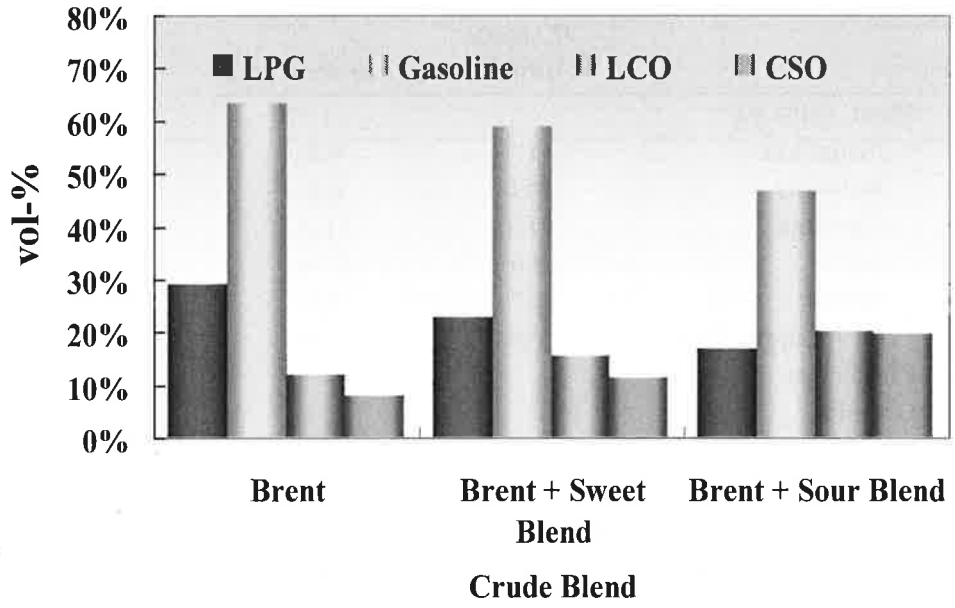
Refiners used to processing the VGO from a sweet conventional crude, like Western Canadian Mixed Sweet (MSW) crude, will see a significant shift in their FCC yield pattern if they start to process VGO from most bitumen-derived crudes. Table 2 shows commercial data from a Canadian refinery, and the impact of switching their FCC feed from a VGO from 100% MSW crude, to a VGO from SSB crude[3]. Conversion in the FCC unit dropped by more than 20% as the yields of LCO and decant oil tripled.

Table 2 – FCC Yield Impact of Processing Synthetic VGO

	Western Canadian	*Synthetic Crude*
Yield, vol% FF		
Propylene	**11.7**	**6.2**
Butylene	**14.0**	**6.9**
Gasoline	**61.1**	**51.3**
LCO	**9.6**	**27.6**
Decant	**2.7**	**9.3**
C_3 + Liquid Yield	**116**	**110**
Properties		
Gasoline		
RON	**91.9**	**96.5**
MON	**80.9**	**84.3**

Most refiners will not see such a drastic change, as they will most likely replace only a portion of their current crude diet with synthetic crudes or bitumen blends. The impact will be closer to that shown in Figure 3. This graph shows the FCC yields for the VGOs from three crude blends; 100% Brent crude, 75% Brent plus 25% of a sweet synthetic blend, like Syncrude SSB, and 75% Brent plus 25% of a sour synthetic blend, like Suncor OSE. As the sweet synthetic and the sour synthetic blends are added to the FCC feed, the yields of LPG and gasoline drop off, while the yields of light cycle oil and slurry oil increase. This has been one of the historical problems refiners have experienced while trying to process synthetics. The loss of FCC conversion has adversely affected the value of the synthetic blends. This adverse impact increases dramatically as the percentage of synthetic crude in the diet increases. All the VGO-range material in OSE crude is coker heavy gas oil. This underscores the impact coker gas oil has on FCC performance, so if a refiner is also adding a new coker to convert the surplus resid in a bitumen blend like WCS, the overall impact on FCC yields could be even more pronounced.

Figure 3 – Impact of 25% Synthetic on FCC Yields



Most aspects of the refinery operation will be affected by the shift to synthetic crudes or bitumen blends. If a refiner decides to import a sweet synthetic crude like SSB, their reformer feed will become richer. With more naphthenes in the reformer feed, hydrogen production will increase. They will, however, have limitations blending distillate fuels. Depending on the other crudes being processed, the refinery could be limited to just 20% SSB in their crude diet if running for maximum jet production (smoke point limit), or about 35% if running for maximum diesel production (cetane number limit). These limits will be relaxed when Syncrude starts producing SSP in June of 2006. There are also fewer distillate blending constraints with other sweet synthetics like OSA, HSB, or PAS. The production of ULSD is more difficult than indicated by the low sulfur level of the distillate cuts of the crude. The sulfur and nitrogen species left in the kerosene and diesel cuts are the most refractory, difficult-to-treat species that could not be removed in the upgrader's relatively high-pressure hydrotreaters. FCC conversion and gasoline yield will drop significantly when using any of the currently-available synthetic crudes, and the production of lube base stock may be impossible due to the aromatic nature of the synthetic VGO. The large percentage of VGO-range material in these crudes may result in the FCC unit capacity becoming a bottleneck. Since synthetic crudes contain no resid, they can be used to increase refinery throughput without having to increase resid conversion capacity.

If a refiner chooses to process one of the sour synthetic crudes, all of the benefits and limitations discussed above for a sweet synthetic crude still apply, but much more severe hydrotreating will be required at the refinery to produce ULSD or acceptable-quality FCC feed. The sulfur and nitrogen content of these crudes is very high compared to a conventional light sweet crude. Additional sulfur plant capacity may be required. The TAN number of these crudes may also become an issue. Crudes with straight-run VGO components from bitumen, like OSV, have higher TAN numbers. Metallurgy upgrades may be required to handle these crudes. Sour synthetic crudes produced from coker products are more aromatic in nature, but the TAN numbers are much lower.

DilBits have both a high resid content and a high sulfur content. They are relatively low in distillate content, and high in VGO content. Significant resid conversion capacity and hydrotreating capacity would have to be added to a light sweet crude refinery to process a DilBit. The FCC unit will again be limited by the large volume of the FCC feed. Although better quality than a coker-derived VGO, the straight-run VGO from bitumen is still very aromatic, and makes a poor FCC feed. The TAN number of these crudes can be high, depending on the source of the bitumen. For example, Athabasca bitumens are reported to have higher TAN numbers. The large volume of condensate in the blend may pose a problem for some refiners. All the light naphtha may not be able to be blended into the gasoline pool without exceeding RVP specifications. As already mentioned, some refiners are already shipping the condensate back to Alberta by rail, both to alleviate any light ends constraint, but also to realize the higher netbacks for condensate. Depending on the gasoline to diesel (G/D) ratio of the refinery involved, there may be insufficient material for the distillate pool.

SynBits generally have a lower resid content than DilBits, but more distillate and VGO-range material. Since the majority of the distillate comes from a sweet synthetic crude, with the remainder from bitumen, the distillate quality is only marginal. Some hydrotreating will be required to achieve a 40 cetane number in the full-range diesel. The VGO is still a poor-quality FCC feed due to the high aromatic content. The TAN number will be lower than that found in a DilBit since about half the feed has already been severely hydrotreated. The sulfur content of a SynBit is also much lower than that of a DilBit.

REQUIRED PROCESS MODIFICATIONS

Changes will be needed in a refinery to accommodate the processing of bitumen-derived crude blends. The addition of resid conversion capacity, of which coking is the most common, to convert the large volume of resid present in a DilBit, SynBit, or SynDilBit like WCS, is almost a given. As mentioned previously, there are several new coking projects being considered for that portion of the refining market currently able to access bitumen-derived crudes via pipelines. The heavy gas oil produced in the coker, coupled with the large volume of low-quality VGO in these crudes, overwhelms FCC units designed for VGO from light sweet crudes. These VGO streams can be upgraded to premium-quality FCC feeds with hydrotreating or hydrocracking. To avoid any regret capital, one must first have a basic understanding of FCC chemistry, and how that chemistry is affected by the addition of bitumen-derived feeds to the FCC unit.

The aromatic nature of bitumen-derived VGOs makes them poor FCC feedstocks. The quality of three bitumen-derived VGOs are compared to the VGO from an Arab Light crude in Table 3. The bitumen-derived fractions have lower API gravities and lower hydrogen contents, consistent with higher levels of sulfur, nitrogen, and aromatics. A brief overview of the chemistry of aromatic conversion in an FCC unit is useful to give more insight into reasons why these feedstocks are more difficult to process.

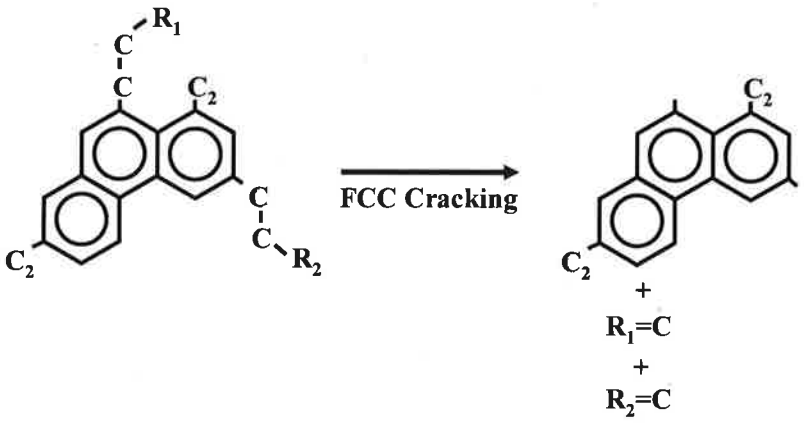
Table 3 – Bitumen-derived VGO Quality

Property	*Lt. Arabian VGO*	*Cold Lake VGO*	*Athabasca VGO*	*Coker HGO*
Gravity, °API	**22.7**	**16.3**	**14.2**	**11.4**
Sulfur, wt-%	**2.10**	**3.10**	**3.77**	**4.99**
Nitrogen, wppm	**820**	**1380**	**1854**	**4032**
Hydrogen, wt-%	**12.47**	**11.38**	**10.80**	**10.43**
Carbon Residue, wt-%	**0.50**	**0.67**	**0.50**	**0.35**
Total Aromatics, vol-%	**58**	**60**	**69**	**76**
Sim Dist D-2887, °F				
IBP / 5wt-%	**382/609**	**597/647**	**621/693**	**457/605**
10	**701**	**671**	**719**	**649**
30	**777**	**734**	**779**	**719**
50	**834**	**795**	**831**	**764**
70	**895**	**856**	**878**	**812**
90/95	**981/1020**	**942/980**	**947/972**	**878/905**
EBP	**1100**	**1054**	**1032**	**955**

Figure 4 illustrates how a three-ring aromatic compound with several alkyl side chains reacts in an FCC unit. Methyl and ethyl groups will tend to stay attached to the aromatic compounds. Alkyl side chains with a carbon number of three or greater will cleave off close to the aromatic ring. The removed alkyl side chains will initially become olefins, and may crack again into smaller components depending on the length of the chain. Paraffinic compounds with a carbon number of five or less tend not to crack. Paraffinic compounds with a carbon number of six crack slowly, and paraffinic compounds with a carbon number of seven or more will crack fairly quickly.

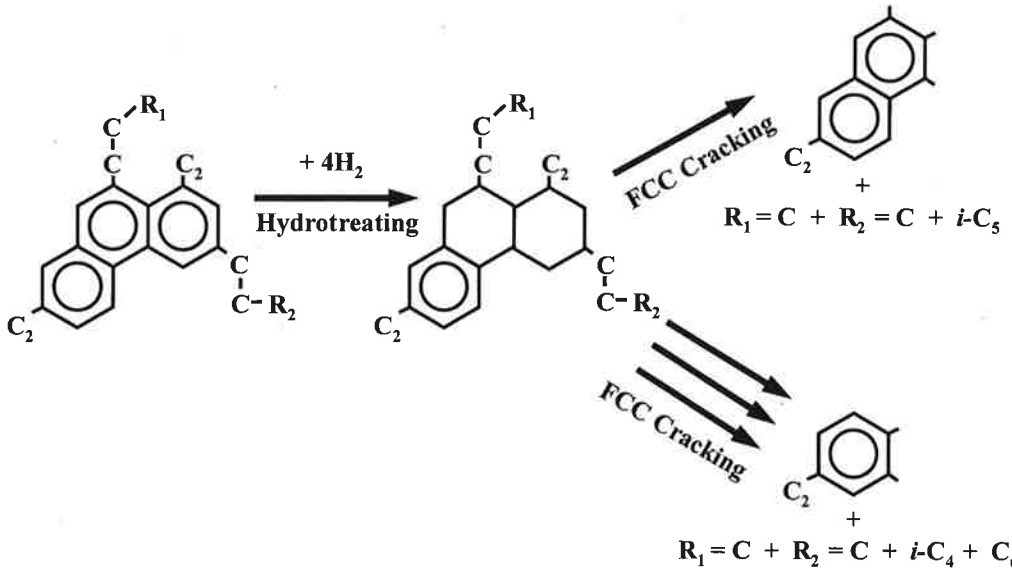
FCC cracking will not open the aromatic ring structures. Depending on the number of short alkyl side chains remaining, the compound in Figure 4 could end up in the FCC light cycle oil product, but would most likely be produced as part of the heavy cycle oil pool or the decant oil.

Figure 4 – FCC Conversion of Multi-ring Aromatic Compounds



If that same three-ring aromatic compound was partially saturated in an FCC feed pretreater before being fed to the FCC unit, the resulting products would be significantly different (Figure 5). Notice it takes only four moles of hydrogen to saturate two of the three aromatic rings. Saturated ring structures crack open far more easily in an FCC unit than aromatic ring structures. While some of the compounds would follow the upper path and partially dehydrogenate back to a two-ring aromatic compound, the majority of these partially saturated ring structures would follow the lower pathway. The longer alkyl side chains would first be cleaved off as before. The saturated rings would then crack open, leaving a single-ring aromatic structure and other paraffinic and iso-paraffinic molecules.

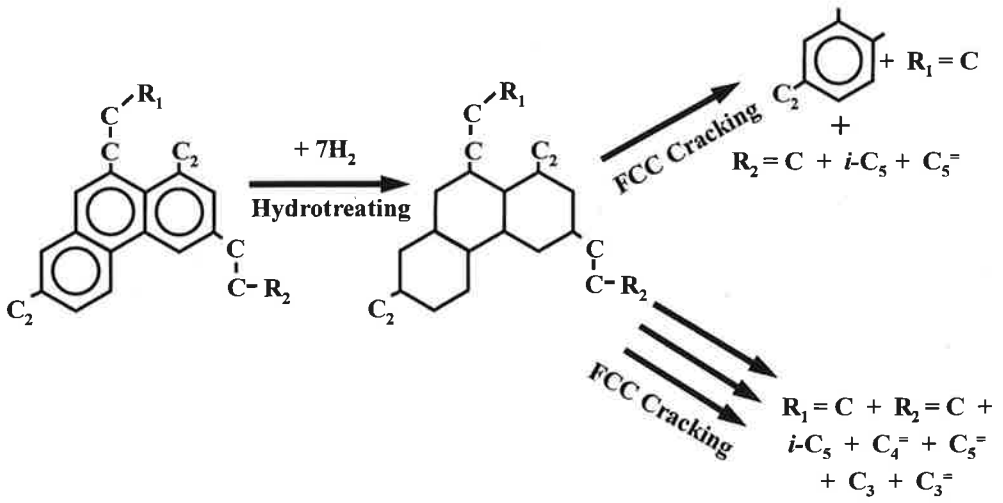
Figure 5 – FCC Conversion of Partially-saturated Aromatic Compounds



The single-ring aromatic structures created in this way are almost never benzene. The structures formed will usually be toluene and xylenes, and thus have high octane number. The iso-paraffins created will continue to crack to lighter compounds as dictated by the carbon number.

If that same three-ring aromatic compound was now fully saturated in an FCC feed pretreater before being fed to the FCC unit, the resultant products would again be significantly different (Figure 6). It would take seven moles of hydrogen to fully saturate the three-ring structure. As before, some of the structures would partially dehydrogenate back to a single-ring aromatic structure, generating several paraffinic molecules. The majority of these saturated ring structures would crack open completely, generating numerous normal paraffins, iso-paraffins, and olefins. Again, the paraffins would continue to crack to lighter compounds, depending upon their carbon number. Normal and iso-paraffinic molecules have much lower octane numbers than aromatic compounds with the same carbon number. By fully saturating the initial compound in the FCC feed pretreater, the gasoline yield would be reduced in favor of a slightly higher LPG and gas yield, and the octane number of the gasoline created would be lower.

Figure 6 – FCC Conversion of Fully-saturated Aromatic Compounds



In general, the FCC cracking of saturated and aromatic ring structures can be summarized by Table 4 below. Cracking of multi-ring aromatics produces high cycle oil yields but low gas, LPG and gasoline yields. The gasoline produced would consist mainly of paraffins and olefins. FCC cracking of single-ring aromatic compounds produces low gas, LPG and cycle oil yields, but a high gasoline yield. The gasoline produced is mostly aromatic, and therefore has a high octane number. FCC cracking of saturated ring structures produces a low yield of cycle oil, a reasonably high gasoline yield, but a higher yield of gas and LPG than cracking of single-ring aromatics. The gasoline produced would again consist mainly of paraffins and olefins, and would therefore have a lower octane number.

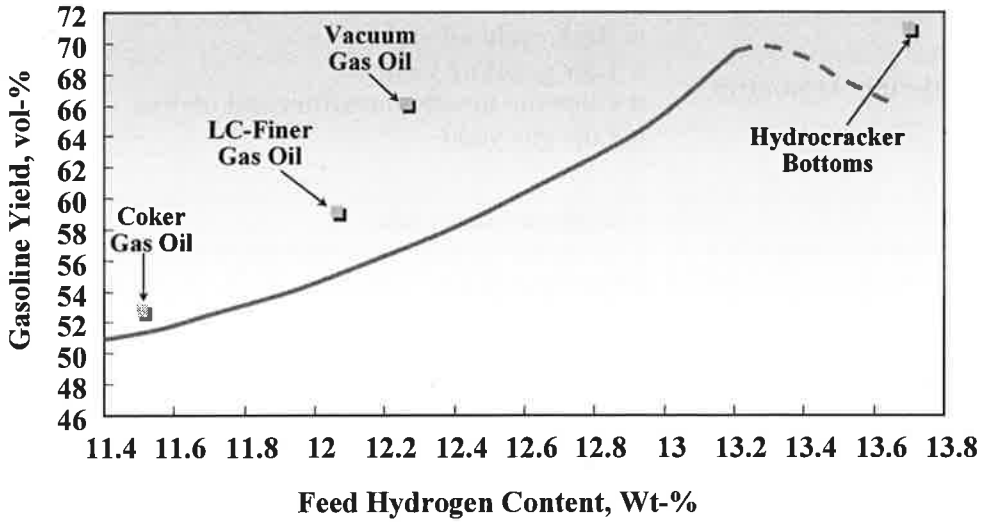
Table 4 – FCC Cracking of Aromatic Rings (Summary)

Multi-ring Aromatics	■ **High cycle oil yield** ■ **Low gasoline yield** ■ **Gasoline mostly paraffins and olefins** ■ **Low gas yield**
Mono-ring Aromatics	■ **High gasoline yield** ■ **Gasoline mostly aromatic (high octane)** ■ **Low gas yield**
Naphthenes	■ **Reasonably high gasoline yield** ■ **Gasoline mostly paraffins and olefins (lower octane)** ■ **Higher gas yield**

From this, it can be concluded that if the refiner's objective is maximum gasoline yield from his FCC unit, the feed pretreater conditions should be set to ensure that all the multi-ring aromatic compounds are saturated down to single-ring aromatics. Hydrogen addition beyond this point would only serve to lower gasoline yield and gasoline octane. Higher conversions would, of course, be achieved, but product value would not be maximized. If a refiner were trying to use his FCC unit to produce olefins for alky feed or MTBE or other chemical production, obviously a higher feed hydrogen level would increase the production of olefins.

The curve shown in Figure 7 illustrates the FCC gasoline yield as a function of the feed hydrogen content. VGOs from more aromatic crudes are near the bottom left-hand end of the curve, while VGOs from more paraffinic crudes would be located near the top right-hand end of the curve. The trend shows that gasoline yield increases with increasing feed hydrogen. Gasoline yield will not continue to rise, however, as the feed hydrogen content is increased. At some point, the gasoline yield will drop off, as the conversion to gas and LPG continues to increase. Overall, conversion will continue to increase, but the gasoline yield will drop. This drop off is not a detriment if the refiner is trying to make olefins for alky feed or petrochemical feedstock with the FCC unit, but it is detrimental if the FCC unit is designed to achieve the maximum gasoline yield. This yield response is represented by the dashed portion of the curve, although the exact inflection point is a function of the feedstock and other variables.

Figure 7 – FCC Gasoline Yield Controlled by Feed Hydrogen Content



Yui et al[5] took several intermediate VGO streams derived from Athabasca bitumen, hydrotreated them at a constant severity, and then processed them in an FCC pilot plant to determine product yields and qualities. The gasoline yield data from that study is also shown in Figure 7. Note that the hydrotreated coker gas oil had a hydrogen content of 11.5wt%, and gave a gasoline yield just above the UOP curve. The gas oil produced in the ebullated-bed resid hydrocracking unit had a higher hydrogen content after hydrotreating, and generated a gasoline yield above the curve, while the hydrotreated VGO distilled directly from bitumen produced a gasoline yield well above the curve. There is a valid reason as to why these bitumen-derived VGO streams all generated FCC gasoline yields above the standard UOP curve. One of the characteristics of these VGOs is the very low paraffin content. Some bitumen derived coker VGO samples analyzed at UOP have actually been shown to contain no paraffins of any kind. All the molecules were aromatics, naphthenes, or sulfur species which were almost all aromatic compounds. Without the paraffins to crack to gas and LPG, bitumen-derived VGOs can produce very high gasoline yields, if the hydrogen content is raised to the appropriate level.

Many people believe that the unconverted oil from a hydrocracker is the best FCC feedstock available. The Yui study obtained a sample of hydrocracker bottoms from a Canadian refinery when they were processing 100% synthetic heavy gas oil in their hydrocracker. It is clear that very high conversions and gasoline yields can be achieved by first hydrocracking the bitumen-derived heavy gas oil streams. Since the data points fall to the right of the conventional crude curve, it suggests that hydrocracker bottoms are less efficient gasoline feedstocks than conventional crudes. The important question becomes, "Is the extra hydroprocessing severity required to raise the hydrogen content from about 12.3 wt% all the way to 13.7 wt% worth an extra five or six percent in FCC gasoline yield?" UOP would suggest that hydrogen is most effectively used to increase the hydrogen content to the 12.3 to 12.5 wt% range. Beyond this level, more and more naphthenes and paraffins would be created in the FCC feed, decreasing the gasoline yield and increasing the LPG and gas yield. Since LPG has a high value to many refiners as alky feed or chemical plant feedstock, the most economic hydrogen content will be different for each application, and must be carefully determined.

Table 5 – Required FCC Feed Pre-treat Severity

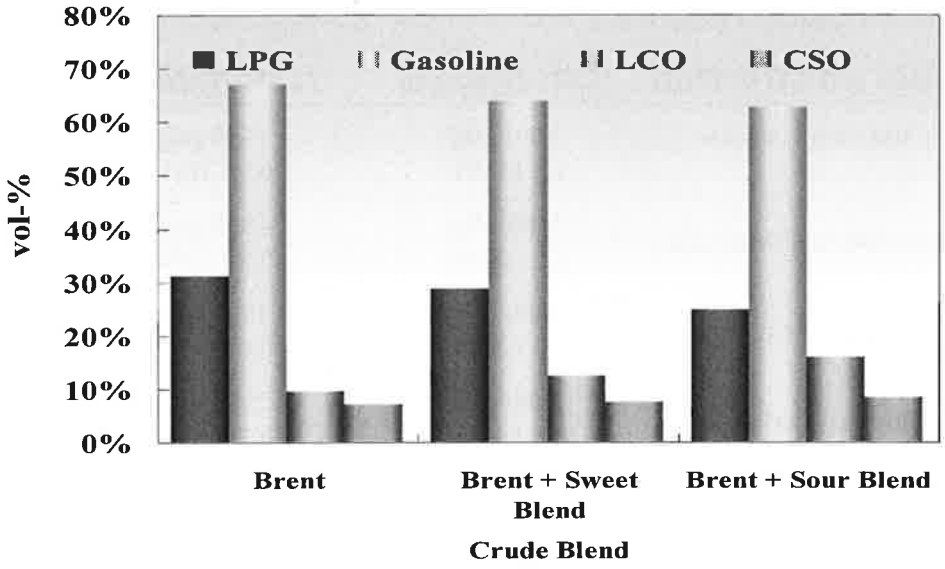
Source of VGO or HGO	*12.0% Hydrogen*	*12.5% Hydrogen*
Vacuum Column	**1400 psig 1.6 LHSV**	**1600 psig 0.9 LHSV**
Ebullated-bed Processing	**1600 psig 1.2 LHSV**	**1800 psig 0.8 LHSV**
Delayed Coker	**1700 psig 1.2 LHSV**	**1900 psig 0.8 LHSV**
Fluidized-bed Coker	**1900 psig 0.8 LHSV**	**2100 psig 0.7 LHSV**

Recognizing that the optimum hydrogen content for bitumen-derived heavy gas oil streams may be between 12.0 and 12.5 wt%, UOP has estimated a rough set of operating conditions necessary using UOP's Unionfining™ process technology to increase the hydrogen content of each of the raw feeds to those levels. As might have been expected, the vacuum heavy gas oil would require the least severe conditions, where heavy gas oil product from the fluidized-bed coking unit would require the most severe conditions. These pressure and liquid hourly space velocity combinations are thought to be close to the economic optimum, but the same results can be achieved at lower pressure if the space velocity is lowered sufficiently.

Many refiners use the UOP K Factor to estimate the hydrogen content of an FCC feedstock. The UOP K Factor works very well when trying to distinguish between aromatic and paraffinic VGO's. However, when the FCC feed is hydrotreated, the UOP K factor-hydrogen content relationship begins to break down. Thus, if the FCC feed has been severely hydrotreated, the refiners should focus on API gravity, nitrogen content, and most importantly, hydrogen content of the FCC feed.

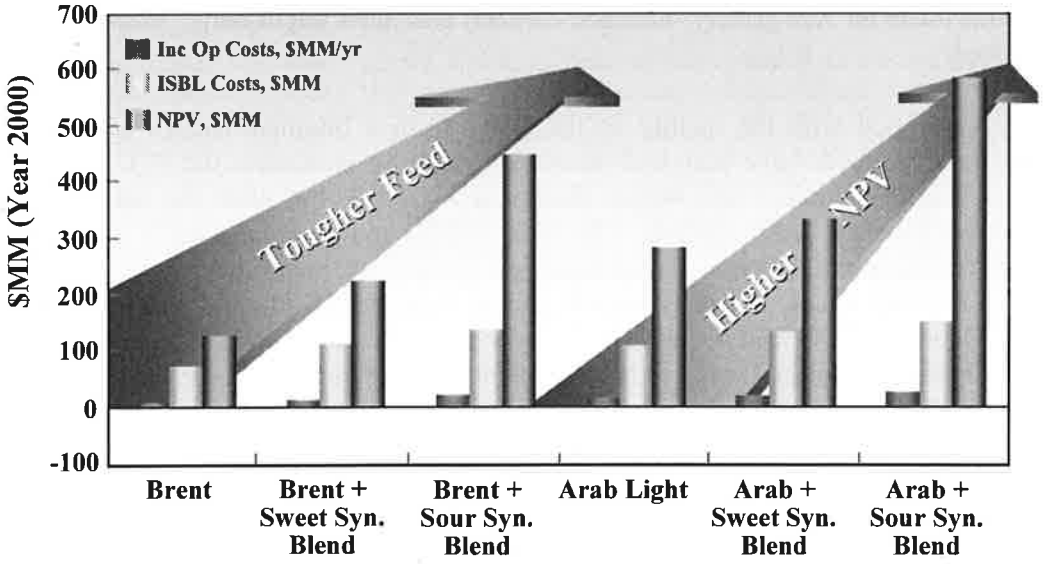
The penalty associated with the quality of the VGO from a bitumen-derived crude changes rapidly once the FCC feeds have been hydrotreated. Figure 8 summarizes the FCC yield pattern for the same Brent/synthetic VGO blends shown in Figure 3, but after the feeds had been hydrotreated to a constant sulfur content. The yield of LPG and gasoline is up in all cases, with the expected reduction in light cycle oil and slurry oil yields. The magnitude of this yield shift is not consistent between cases. There is a much greater increase in LPG and gasoline yield with synthetic VGOs in the FCC feed, especially the sour synthetic blend. When this same analysis was completed substituting the Brent crude component with the more aromatic Arab Light crude, the increases in LPG and gasoline yields were even greater, to the point that two of the three cases had gasoline yields greater than the corresponding Brent crude cases. **The most difficult to treat feeds gained the most advantage by hydrotreating.**

Figure 8 – FCC Yields on Hydrotreated VGO Feeds



In 2000, UOP completed an internal study that compared the economics of FCC feed pretreating with post-treating of FCC products. UOP studied the impact of blending 25% of two different synthetic crudes into a conventional crude diet. The FCC yield patterns discussed above were part of that study. One of the conclusions from that study was confirmation that the economics for pretreating the FCC feed improved as the quality of the raw FCC feed became worse. The incremental operating costs, capital costs, and net present values for each case are summarized in Figure 9 below. While all the capital and product prices used for this study are now six years out of date, the conclusion is still valid; the tougher the feed, the higher the NPV.

Figure 9 – FCC Feed Pre-treat Economics



The economics of FCC feed pretreating projects are driven by two factors: better yields from the FCC unit itself, and incremental conversion. Whenever a VGO-range feed is severely hydrotreated, about 10 – 15 lv% of the feed is converted to distillate and lighter products. Some of this conversion is simply a shift in the boiling point curve as sulfur and nitrogen is removed from the molecules, but some hydrocracking to lighter products does occur. Assuming the distillate and lighter products are fractionated from the hydrotreated VGO before it is fed to the FCC unit, hydrotreating the feed effectively debottlenecks the FCC unit. If a refinery has an FCC unit sized for 50 KB/D, after hydrotreating, there will only be about 43 KB/D of hydrotreated VGO left to feed to the FCC unit. This allows the refiner to acquire additional crude if the other processing units have surplus capacity, or at a minimum, it allows the refiner to purchase surplus VGO to keep the FCC unit full. In either case, it is the incremental conversion in the FCC feed pretreater that makes the economics so attractive.

UNICRACKING™ PROCESSES

As previously mentioned, all the sweet and sour synthetic crudes, as well as the bitumen blends, have a high VGO content relative to conventional crude. Hydrotreating alone can turn these low-quality VGOs into premium FCC feeds and reduce the volume of the feed available, but most refiners importing bitumen-derived crudes for the first time will still have surplus VGO that cannot be processed in their FCC unit. This problem will be made worse by the addition of the heavy gas oil generated in the coker, for those refiners choosing to process a bitumen blend. Additional VGO conversion capacity must be added. Refiners may consider an FCC expansion project, or even an entirely new FCC unit, but since they are probably going to construct a high-pressure hydrotreater to process the FCC feed, it only makes sense to consider converting the incremental VGO barrels with hydrocracking technology. Hydrocracking has the advantage in that, unlike an FCC unit, it can be used to produce both gasoline and high-quality distillate fuels.

In order to avoid increased utility cost and unnecessary quality give-away caused by excess hydrogen consumption, efficient hydrogen consumption is a critical parameter in hydroprocessing unit design and operation. With growing demand and more stringent specifications for fuels, it is recognized by industry experts that hydroprocessing technologies will be key in the future to meeting the refinery conversion capacity and quality needs. Recent advances in UOP's hydrocracking technology portfolio, such as the Advanced Partial Conversion Unicracking (APCU) process, and the other Unicracking configurations discussed below, were designed for optimal treatment of the distillate and unconverted product fractions, resulting in more efficient hydrogen utilization.

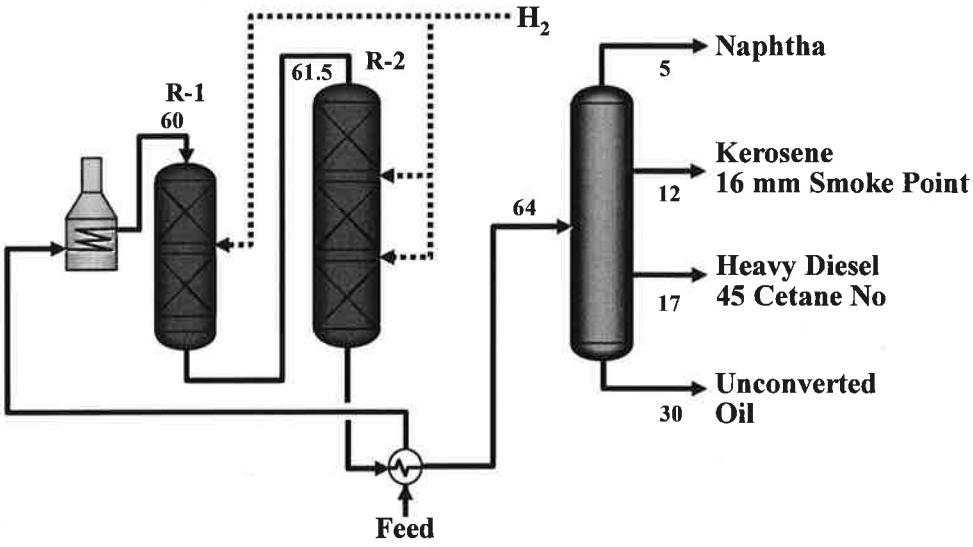
Once-Through Unicracking Unit

When most refiners think about partial-conversion hydrocracking, they think of a simple once-through Unicracking process unit. This configuration has the lowest capital cost of the various Unicracking process configurations. The unit shown in Figure 10 is sized for a fresh feed rate of 60 KB/D with an unconverted oil rate of 30 KB/D, meaning the unit is designed to give a gross conversion of 50 lv%. If the cracking reactor was loaded with a high-activity naphtha catalyst, such as UOP's HC™ 29 catalyst, the yield pattern would be very close to that shown in Figure 10. The unit would produce about 5 KB/D of naphtha, 12 KB/D of kerosene with a relatively low smoke point, and 17 KB/D of heavy diesel with a reasonable cetane number. Note that if the

kerosene and heavy diesel were combined to form a full-range diesel, the cetane number would be close to 40.

One key consideration with this simple configuration is the quality of the unconverted oil produced as FCC feed. Unconverted oil from this unit would have high hydrogen content, similar to the 13.7 wt% seen in the unconverted oil from the Canadian refinery discussed earlier. Again, if a refiner is trying to make alky feed or petrochemical feedstock with their FCC unit, this would make an excellent feed, but if the FCC unit is being run for maximum gasoline production, the hydrogen content of the unconverted oil may be too high.

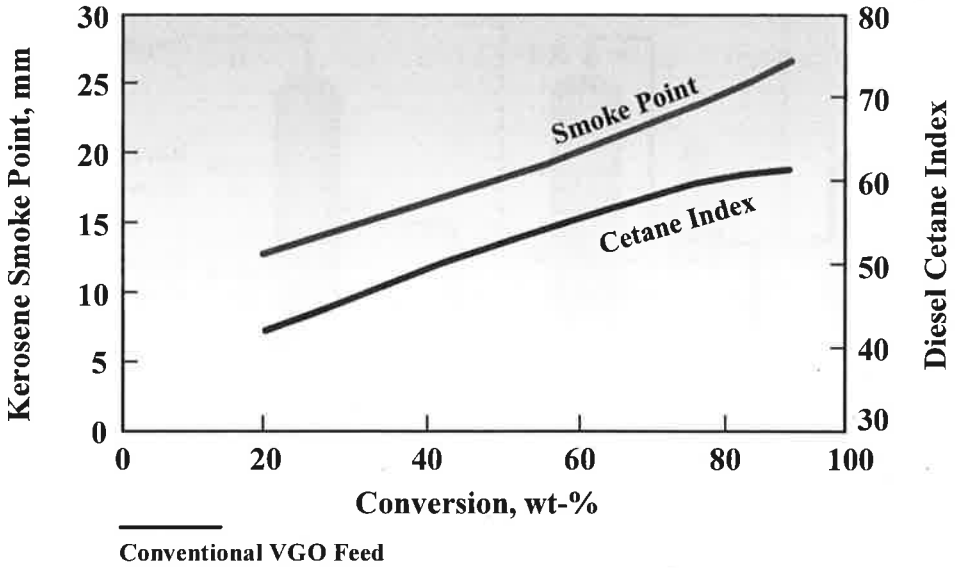
Figure 10 – Once-through Unicracking Unit



Separate-Hydrotreat Unicracking Unit

The quality of the distillate fuels produced in a low conversion once-through Unicracking unit can be poor. Figure 11 is a plot of both kerosene smoke point and diesel cetane index plotted against conversion in the Unicracking unit. While these plots are based on a VGO from conventional Arab Light crude, the message is the same for bitumen-derived VGOs. Kerosene smoke point and diesel cetane index both increase with increasing conversion. If a refiner wants to generate high-quality distillate fuels from a poor-quality VGO stream, they need to run the Unicracking unit at a high conversion level.

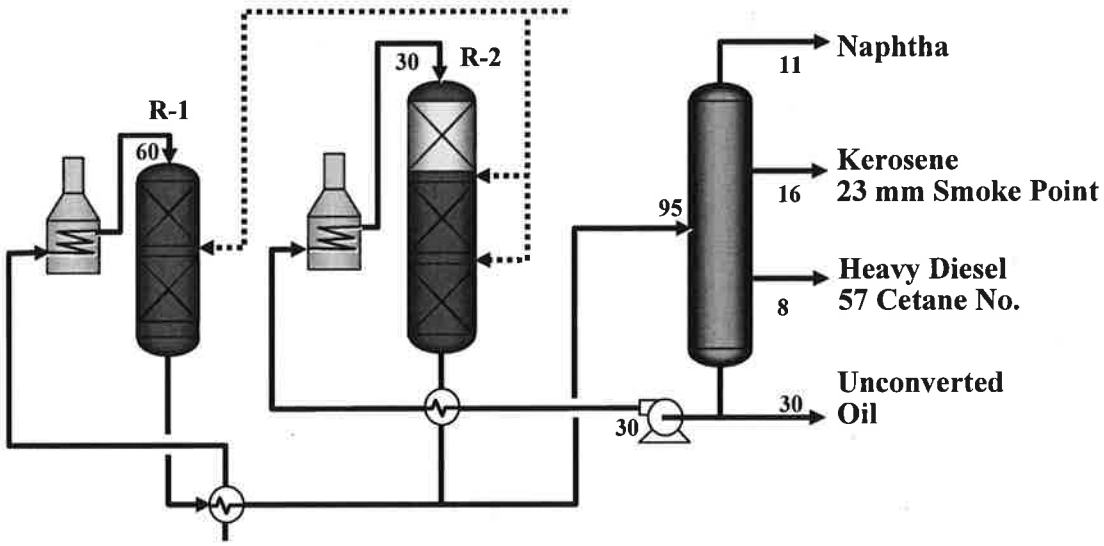
Figure 11 – Distillate Quality as a Function of Conversion



Both the distillate quality and the unconverted oil quality can be controlled by using a different Unicracking unit configuration. In UOP's separate-hydrotreat configuration shown in Figure 12, the severity in the R-1 pretreat reactor would be set to achieve the desired hydrogen content in the unconverted oil, in this example a hydrogen content of 12.5 wt%. The R-1 effluent would be routed directly to the product fractionator. About 30 KB/D of unconverted oil would be fed to the cracking reactor, this time running at 80 lv% crack-per-pass. A bed of pretreat catalyst could be added to the cracking reactor to get the organic nitrogen down to the optimum level for the cracking catalyst, about 100 wppm or less. The effluent from the cracking reactor would also be routed directly to the product fractionator.

The fractionator in the separate-hydrotreat configuration would be larger than that in the once-through configuration, but the high-pressure equipment in the cracking reactor loop would be 50% smaller, now sized for only 30 KB/D. At the high conversion level of 80 lv% crack-per-pass, the unit would generate higher yields of both naphtha and kerosene, but less heavy diesel. The quality of both distillate streams would be significantly higher than the once-through unit. Only about 3 KB/D of the 30 KB/D of unconverted oil produced in the unit would have been processed to a high hydrogen content in the cracking reactor, thereby reducing overall hydrogen consumption. The separate-hydrotreat configuration effectively allows independent control of both the unconverted oil quality and the distillate fuel quality.

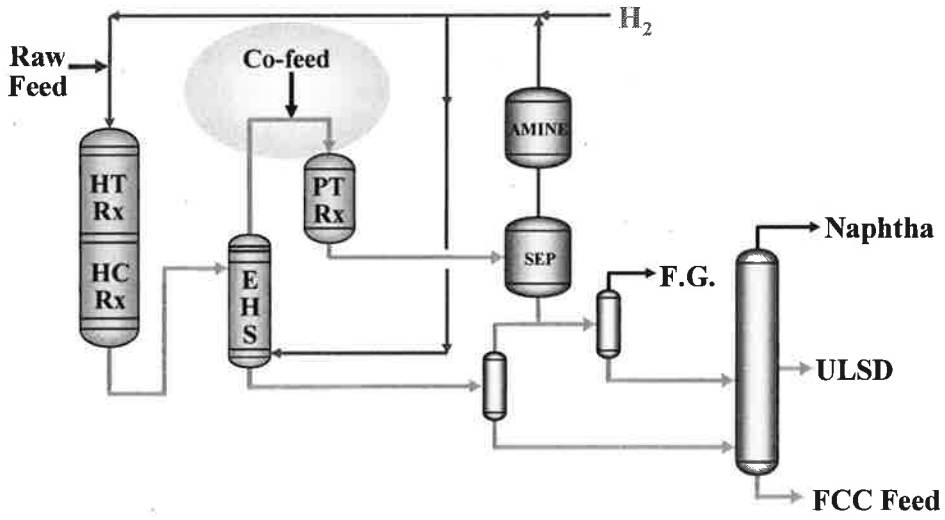
Figure 12 – Separate-Hydrotreat Unicracking Unit



Advanced Partial Conversion Unicracking (APCU) Unit

Typical once-through hydrocrackers operating at low to moderate conversion levels may not produce USLD-quality distillate. The unit pressure is often set by the need to produce low-aromatic, high-cetane diesel. This higher design pressure results in the production of unconverted oil with a high hydrogen content, and a higher overall hydrogen consumption. In the APCU unit flow scheme depicted in Figure 13, the unit is designed to produce ULSD and partially hydrotreated FCC feedstock as primary products, while operating at a significantly lower pressure. The refiner achieves two goals with the addition of this unit; increased production of ULSD, as well as improved quality of the FCC feedstock.

Figure 13 – Advanced Partial Conversion Unicracking Unit



The benefits of the APCU process compared to a conventional low-conversion hydrocracking process are reflected in more efficient use of hydrogen due to lower pressure operation, and staging of reactions in a way that minimizes over-processing and product quality giveaway. Separation of cracked products from the desulfurized FCC feed in the Enhanced Hot Separator (EHS), followed by an additional Distillate Unionfining process step, allows the distillate quality to be controlled independently of FCC feed quality. Over-treating of FCC feed is avoided and excess hydrogen consumption is minimized. The APCU process can achieve hydrogen savings of five to ten percent compared to a conventional mild hydrocracking scheme. The integration of a separate Unionfining reactor in the process enables post-treating of other refinery middle distillate streams. For refiners processing a bitumen blend following the installation of a coker, the coker diesel stream must be severely hydrotreated to meet ULSD specs. With the APCU process, that stream could be processed as a co-feed in the Distillate Unionfining reactor, using the hydrogen and heat from the Unicracking reactor, resulting in utility cost savings. The level of hydrotreating of these streams can be independently controlled to add just the right amount of hydrogen, an added benefit of APCU process.

The typical operating conditions and the resultant FCC feed quality for FCC feed pretreating, the once-through Unicracking process, the separate-hydrotreat Unicracking process, and the APCU Unicracking process are summarized in Table 6. Since the quality of the VGO from an Arab Light crude is so different from that of a WCS VGO, the operating conditions to treat both feeds are shown for comparison. Note that this comparison represents model predictions for representative feed properties and that the units are sized for typical run lengths. The table should be used as a qualitative guide to differentiate between the various processing options. This is not meant to be a substitute for a customized hydroprocessing solution.

Table 6 – Hydroprocessing Options Summary

Feed	*Unit Type*	*FCC Feed H_2, wt-%*	*FCC Feed Nitrogen, ppm*	*ULSD*	*Pressure psig*	*LHSV*	*FCC Feed %FF*	*H_2 Cons (SCFB)*
Arabian	**Unionfining Process**	**12.8**	**<1000**	**No**	**Base**	**Base**	**85**	**500**
Arabian	**Once-Through Unicracking Process**	**13.5**	**<50**	**Yes**	**Base+600**	**0.3*Base**	**50**	**1150**
WCS	**Unionfining Process**	**12.4**	**<1000**	**No**	**Base+1000**	**0.3*Base**	**80**	**1000**
WCS	**Once-Through Unicracking Process**	**13.2**	**<50**	**Yes**	**Base+1000**	**0.17* Base**	**50**	**1900**
WCS	**Separate-Hydrotreat Unicracking Process**	**12.5**	**<500**	**Yes**	**Base+1000**	**0.2*Base**	**50**	**1700**
WCS	**APCU Process**	**12.9**	**<50**	**Yes**	**Base+800**	**0.17*Base[+]**	**50**	**1800**

[+] Plus additional volume for diesel co-feed

Many refiners believe they can treat a portion of their VGO in a Unicracking unit to generate some high-quality VGO, and then blend it with lower quality VGO, like coker gas oil, to get an FCC feed with hydrogen content near the 12.5 wt% UOP believes to be close to the optimum level. While this may be true mathematically, the FCC unit in actuality is concerned only with the types of molecules being fed to it, and not the average hydrogen content. The three- and four-ring aromatics in the coker gas oil will still end up in the cycle oil or the slurry oil, and the naphthenes and paraffins in the hydrocracker bottoms will still be mostly converted to gas and LPG. Some hydrogen donor reactions will occur in the FCC unit to transfer hydrogen from the hydrogen-rich stream to the hydrogen-deficient stream, but the overall yields will still be significantly worse compared to those obtained if the entire feed stream has been hydrotreated to a hydrogen content of 12.5 wt%. Some refiners have reported significant synergies when processing Unicracking unit unconverted oil with lower-quality VGOs in their FCC unit.

EXPERIENCE AND PILOT PLANT STUDIES

UOP has been very successful in licensing technology for synthetic crude projects in Canada, the United States, and Venezuela. Our understanding of the processing issues is based on extensive pilot plant and commercial experience. UOP has completed both hydrotreating and hydrocracking pilot plant studies on numerous distillate and VGO-range feeds derived from Athabasca bitumen, Cold Lake bitumen, Lloydminster heavy conventional crudes, and Orinoco Belt heavy crudes.

UOP has licensed 22 of the 32 hydroprocessing units currently in operation, design, or construction for the upgraders in Western Canada. UOP's experience with hydrotreating, hydrocracking, FCC, coking, and reforming gives us the unique ability to look at all the units affected by import of any of these bitumen-derived crudes.

CONCLUSIONS

The supply of Canadian light and medium sweet crudes is declining. There is a vast oil sands resource in Canada which up until recently, has not been exploited to any great degree. Synthetic crudes and bitumen blends will become the dominant crudes in PADD II, PADD IV, and Northern PADD V in the next ten to fifteen years. Refiners planning to process one of the bitumen blends will have to install additional resid conversion capacity. Refiners planning to process any bitumen-based crude will have to install FCC feed pretreating capacity to maintain acceptable FCC yields. Many refiners will have to install hydrocracking capacity to convert the large volume of VGO-range barrels present in these crudes, and to meet the increasing demand for distillate fuels.

The hydrogen content of an FCC feedstock is the critical parameter to control when trying to optimize the performance of all the VGO processing units. UOP has developed novel Unicracking process flow schemes that allow the refiner to control the hydrogen content of the FCC feed independently from conversion level or distillate quality.

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Regulatory Announcement

EPA Finalizes More Stringent Emissions Standards for Locomotives and Marine Compression-Ignition Engines

The U.S. Environmental Protection Agency (EPA) is adopting standards that will dramatically reduce emissions of diesel particulate matter (PM) and nitrogen oxide (NOx) from locomotives and marine diesel engines. This three-part program aims to:

1. tighten emissions standards for existing locomotives and large marine diesel engines when they are remanufactured;
2. set near-term engine-out emissions standards, referred to as Tier 3 standards, for newly-built locomotives and marine diesel engines; and
3. set longer-term standards, referred to as Tier 4 standards, for newly-built locomotives and marine diesel engines that reflect the application of high-efficiency aftertreatment technology.

EPA is also finalizing new idle reduction requirements for newly-built and remanufactured locomotives and adopting provisions to encourage a new generation of clean switch locomotives, based on clean nonroad diesel engine standards. This rule completes an important step in EPA's ongoing National Clean Diesel Campaign (NCDC).

EPA estimates 90 percent PM reductions and 80 percent NOx reductions from Tier 4 engines meeting these standards, compared to engines meeting the current Tier 2 standards. By 2030, this program will reduce annual emissions of NOx by about 800,000 tons and PM emissions by 27,000 tons and those emission reductions continue to grow beyond 2030 as fleet turnover is completed.

EPA United States Environmental Protection Agency

Office of Transportation and Air Quality
EPA-420-F-08-004
March 2008

Changes from Proposed Rule

The final requirements will bring earlier and significantly greater emission reductions of NOx and PM from the locomotive and marine sector than the proposed program envisioned. This is accomplished by finalizing the first-ever national standards for remanufactured large commercial marine diesel engines (above 600kW) and starting Tier 4 NOx requirements for line-haul locomotives and for the largest (2000-3700 kW) marine engines two years earlier than initially proposed. These changes reflect important cooperative efforts by the regulated industry to implement cleaner technology as early as possible and will provide communities across the United States with crucial emissions reductions both in the near-term and long-term.

Background

Existing EPA regulations in 40 CFR parts 92 and 94 include standards for emissions of PM, NOx, hydrocarbons (HC) and carbon monoxide (CO) from locomotive and marine compression-ignition engines (also called marine diesel engines). These standards rely on engine-based technologies to reduce emissions. The opportunity to gain large additional public health benefits, as well as the similarities between these engines and highway diesel and nonroad engines, have led us to implement additional emission controls based on the high-efficiency aftertreatment technologies that will soon be in use by highway and nonroad engines.

Need to Reduce Locomotive and Marine Diesel Emissions

Locomotive and marine diesel engines contribute significantly to air pollution in many of the nation's cities and towns. EPA anticipates that over the next few decades, these engines may account for an even greater share of overall emissions as other emission control programs take effect for cars and trucks and other nonroad emissions sources. Estimates show that, without the emission reductions from this final action, by 2030 locomotive and marine diesel engines would contribute more than 65 percent of national mobile source diesel PM2.5, or fine particulate, emissions and 35 percent of national mobile source NOx emissions, a key precursor to ozone and secondary PM formation.

As of October 10, 2007, air quality data show that about 144 million people live in areas that violate air quality standards for ground-level ozone, also called smog, and about 88 million people live in areas that violate air quality standards from PM. These pollutants contribute to serious public health problems that include premature mortality, aggravation of respiratory and cardiovascular disease, and aggravation of existing asthma, acute respiratory symptoms and chronic bronchitis. Beyond the impact these diesel engines have on our nation's ambient air quality, exposure to diesel exhaust has been classified by EPA as being likely carcinogenic to humans. Children, people with heart and lung diseases, and the elderly are most at risk.

Locomotive and marine diesel emissions reductions will particularly benefit those who live, work, or recreate in and along our nation's coastal areas, rivers, ports, and rail lines. Such reductions will also have beneficial impacts on visibility impairment and regional haze, as well as reducing crop damage and acid rain.

Description of Engines Covered

The finalized requirements cover all locomotives and many marine diesel engines already subject to EPA emission standards, as follows:

- Locomotives: With some limited exceptions, the regulations apply to all diesel line-haul, passenger, and switch locomotives that operate extensively within the United States including newly manufactured locomotives and remanufactured locomotives that were originally manufactured after 1972.

- Marine Diesel Engines: The regulations apply to both newly manufactured marine diesel engines and remanufactured commercial marine diesel engines above 600 kilowatt (kW) or 800 horsepower (hp) with displacement less than 30 liters per cylinder installed on vessels flagged or registered in the United States. These are commonly referred to as marine diesel engines and are divided into three categories for the purposes of EPA's standards. The cut points for the standards have been revised in the final rule to ensure that the appropriate standards apply to every group of engines. Category 1 represents engines up to 7 liters per cylinder displacement. Category 2 includes engines from 7 to 30 liters per cylinder. Finally, Category 3 engines are those at or above 30 liters per cylinder. Category 3 engines are not included in this rule. They are typically used for propulsion on ocean-going vessels and will be addressed in a separate EPA rulemaking.

  Marine diesel engines covered by this proposal are used in a variety of applications. Commercial propulsion applications range from fishing and tug boats to Great Lakes freighters. Recreational propulsion applications range from sailboats to super-yachts. Auxiliary power units range from small generator sets to large auxiliary engines on ocean-going vessels.

Exhaust Emissions Standards

The final rule consists of a three-part emission control strategy.

First, the final standards for existing locomotives and marine diesel engines are more stringent when they are remanufactured. These standards take effect as soon as certified remanufacture systems are available, as early as 2008.

Second, the rule sets near-term emission standards, referred to as Tier 3 standards, for newly-built locomotive and diesel marine engines. These standards reflect the application of currently available technologies to reduce engine-out PM and NOx emissions and phase-in starting in 2009. The rule also creates new idle reduction requirements for new and remanufactured locomotives and establishes a new generation of clean switch locomotives, based on clean nonroad diesel engine standards.

Third, the final long-term emissions standards, referred to as Tier 4, apply to newly-built locomotives and marine diesel engines. These standards are based on the application of high-efficiency catalytic aftertreatment technology and would phase-in beginning in 2014 for marine

diesel engines and 2015 for locomotives. These standards are enabled by the availability of ultra-low sulfur diesel fuel with sulfur content capped at 15 parts per million, which will be available by 2012. These marine Tier 4 engine standards apply only to commercial marine diesel engines above 600 kW (800 hp).

EPA estimates this final rule will result in PM reductions of about 90 percent and NOx reductions of about 80 percent from engines meeting these standards, compared to engines meeting the current standards. The proposed standards would also yield sizeable reductions in emissions of HC, CO, and other air toxics.

Program Costs

The social cost estimates of the new program will be approximately $740 million in 2030. The impact of these costs on society are expected to be minimal, with the prices of rail and marine transportation services in 2030 estimated to increase by about 0.6 percent for locomotive transportation services and about 1.1 percent for marine transportation services.

Program Benefits

The final rule will result in substantial benefits to public health and welfare and to the environment. EPA estimates that by 2030 this comprehensive emission control program will reduce annual emissions of NOx and PM by approximately 800,000 and 27,000 tons, respectively, and the magnitude of these reductions will continue to grow well beyond 2030. We estimate that the monetized health benefits of this rule in 2030 will range from $9.2 billion to $11 billion per year, assuming a 3 percent discount rate, or between $8.4 billion to $10 billion, assuming a 7 percent discount rate. The estimated annual social cost of the program in 2030 is significantly less, at $740 million. Overall benefits are estimated to outweigh social costs by 9:1 to 15:1 depending on the discount tool.

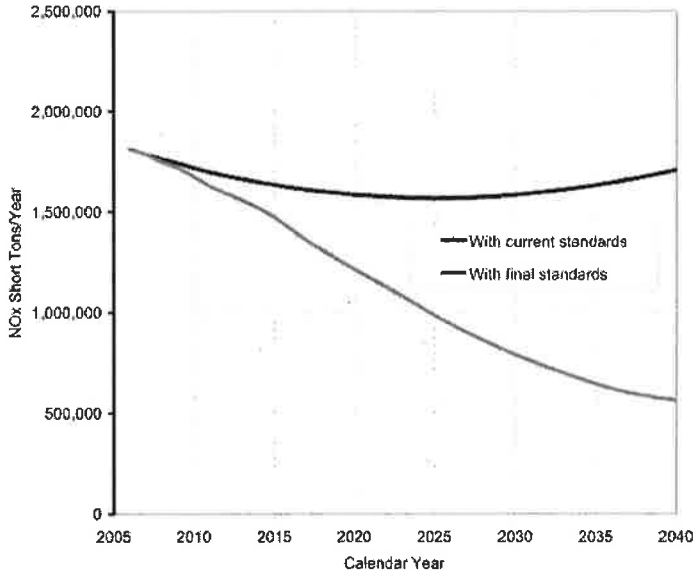

Figure 1 NOx Emissions (tons per year) Reductions from Final Rule

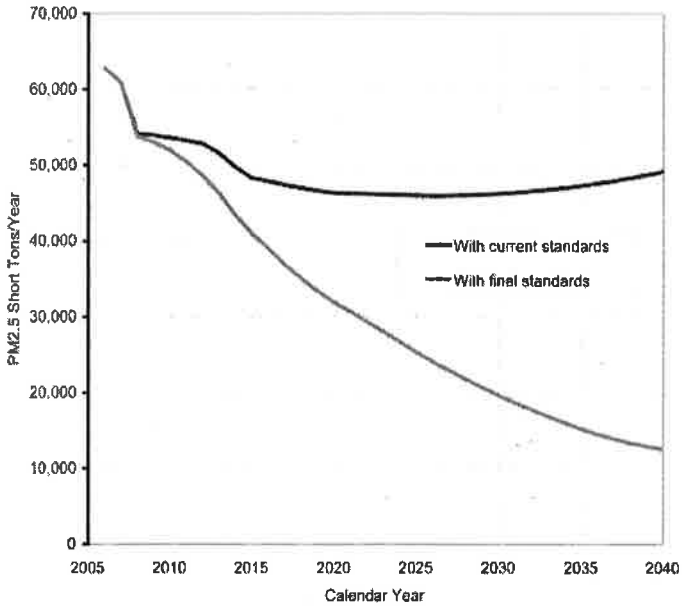

Figure 2 PM2.5 (tons per year) Reductions from Final Rule

For More Information

You can access the rule and related documents on EPA's Office of Transportation and Air Quality (OTAQ) Web site at: www.epa.gov/otaq/marine.htm or www.epa.gov/otaq/locomotv.htm.

For more information on this rule, please contact the Assessment and Standards Division information line at:

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Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children

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Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. T. Nicolai, D. Carr, S.K. Weiland, H. Duhme, O. von Ehrenstein, C. Wagner, E. von Mutius. ©ERS Journals Ltd 2003.

ABSTRACT: Conflicting results have been reported for the relationship between traffic exposure and inception of atopy. The effect of traffic on the prevalence of asthma and atopy at school age was investigated in a representative population.

Random samples of schoolchildren (n=7,509, response rate 83.7%) were studied using the International Study of Asthma and Allergies in Childhood phase-II protocol with skin-prick tests, measurements of specific immunoglobulin E and lung function. Traffic exposure was assessed *via* traffic counts and by an emission model which predicted soot, benzene and nitrogen dioxide (NO_2).

Traffic counts were associated with current asthma, wheeze and cough. In children with tobacco-smoke exposure, traffic volume was additionally associated with a positive skin-prick test. Cough was associated with soot, benzene and NO_2, current asthma with soot and benzene, and current wheeze with benzene and NO_2. No pollutant was associated with allergic sensitisation.

High vehicle traffic was associated with asthma, cough and wheeze, and in children additionally exposed to environmental tobacco smoke, with allergic sensitisation. However, effects of socioeconomic factors associated with living close to busy roads cannot be ruled out.

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During the last 50 yrs, respiratory allergic diseases have increased in children in many countries with modern living conditions. At the same time, car traffic and air-pollution levels have undergone large changes. At sufficiently high concentrations, pollutants such as particulate matter, sulphur dioxide (SO_2), car exhaust and ozone are known to be associated with respiratory symptoms. While it is well accepted that air pollution can trigger symptoms in children with established asthma [1], its influence on the inception of asthma and allergies is not known. It would be of major concern if the increasing prevalence was, at least in part, due to traffic-related air pollution.

In cities, car traffic accounts for almost all benzene and most of the nitrogen dioxide (NO_2) and carbon monoxide, but only for a small fraction of SO_2 [2]. Fine dust [2], latex particles and black smoke are other emissions related to motor vehicles. Animal and *in vitro* human experiments [3–5] point towards an effect of diesel exhaust on allergic sensitisation. Traffic-related pollutants have been suggested to alter the antigenicity of pollen and might thereby cause increased allergic sensitisation or precipitate symptoms in allergic subjects [6–8].

Most, but not all, paediatric epidemiological studies found a consistent but small effect of long-term exposure to car traffic or its emissions on respiratory symptoms and lung function [2, 9–14]. A large cross-sectional study using pollutant exposure measured on a 1-km^2 grid in Dresden, Germany, found increased cough, bronchitis and nonatopic asthma, but no effect on atopic asthma and allergies [2]. However, due to the German reunification process, pollutant exposure has changed greatly during the lifetime of these children. More doctor-diagnosed asthma was reported in children living within 100 m of a freeway compared with those living farther away [15].

The relationship between allergy and traffic exposure is less consistent. A recent study in Switzerland found increased allergic sensitisation (but no relation to symptoms) in a subgroup of adults living for >10 yrs on a busy road [16]. However, a number of paediatric studies found no increase in allergy with measured traffic exposure [2, 9, 12, 13].

The conflicting results of the epidemiological studies are at least partly attributable to small sample sizes in some studies, the difficulty of separating socioeconomic variables from traffic exposure, and reporting bias for self-reported traffic exposure and symptoms. As the reported effect size is mostly rather small, the sample must be sufficiently large to detect or exclude a possible association. Also, exposure assessment is difficult but critical in such studies.

The purpose of this study was to determine the relationship between vehicle-traffic counts and estimated pollutant levels at the place of residence and reported respiratory symptoms, doctor diagnoses and measured allergic sensitisation and respiratory function in a large random sample of children.

Methods

Study population and study design

An International Study of Asthma and Allergies in Childhood (ISAAC) phase-II cross-sectional survey was performed

For editorial comments see page 913.

in Munich, Germany, with ~1.3 million inhabitants [17]. Random samples of school classes were selected in two age groups: school beginners aged 5–7 yrs and schoolchildren of the fourth grade aged 9–11 yrs. Questionnaires were given to parents, and children underwent skin-prick tests, blood sampling, lung function testing and bronchial challenge if written informed consent had been obtained from the parents. The local ethics committee had approved the study.

Questionnaires

The questionnaires included the ISAAC core questions on symptoms of asthma, allergic rhinitis and atopic eczema, which have been reported in detail elsewhere [18, 19], and were administered between September 1995–December 1996. Current wheeze was defined as wheezing in the last 12 months. Children were defined as having asthma if their parents reported that asthma had been diagnosed at least once or that a doctor had diagnosed asthmatic, or spastic or obstructive bronchitis more than once. Asthma with symptoms in the last 12 months was defined as current asthma. Hay fever and atopic dermatitis were defined by a reported diagnosis from a doctor. The questionnaire included detailed questions about the child's nationality, the family history of atopic diseases, the number of siblings, and several other potential confounding factors, such as environmental tobacco smoke (ETS) exposure and parental education as a marker of socioeconomic status (SES). SES was defined as highest parental school education; high SES being >12 yrs of school education or university. ETS was defined as any current exposure to cigarettes, pipes or cigars in the home.

Skin-prick tests

All children in the 9–11 yrs age group were invited to participate in skin-prick testing, whereas in the younger age group only a random subsample (n=1,875) was selected. The sensitivity to six common aeroallergens (*Dermatophagoides pteronyssinus*, *D. farinae*, tree pollen, grass pollen, *Alternaria tenuis* and cat dander) was assessed using standardised extracts (ALK, Hørsholm, Denmark) and ALK lancets. A positive (histamine 10 $mg \cdot mL^{-1}$) and negative control were added. The weal size after 15 min was defined as the mean of the longest diameter and the length of its perpendicular diameter. Children with a weal reaction ≥ 3 mm after subtraction of the reaction to the negative control, to one or more of the allergens tested, were considered to be atopic.

Blood sampling and laboratory analyses

As with skin-prick testing, all children in the 9–11 yrs age group were asked to provide a blood sample, whereas in the younger age group only a random subsample (n=1,875 in Munich) was selected. Serum was separated by centrifugation, frozen and stored at -70°C before analysis. A screening test (SX1 test; Pharmacia, Uppsala, Sweden) was used to detect specific serum immunoglobulin (Ig)E antibodies to a wide array of aeroallergens (*D. pteronyssinus*, mixed grass pollen, birch pollen, mugwort pollen, cat dander, dog dander and *Cladosporium herbarum*) in one central laboratory at the University of Berlin. Atopic sensitisation was assumed to be present if a level of ≥ 0.7 $kU \cdot L^{-1}$ of specific serum IgE was measured.

Pulmonary function testing and bronchial challenge

Because of the long duration of the bronchial challenge protocol, only a random subsample of the children aged 9–11 yrs was invited to participate (n=2,019). Lung function was measured with a spirometer (MasterScope; Jäger, Würzburg, Germany). The criteria for completion of reproducible and satisfactory spirograms as set by the American Thoracic Society [20] were followed. Airway responsiveness was assessed using a 4.5% hyperosmolar saline challenge [17, 21]. Each subject, whose baseline forced expiratory volume in one second (FEV1) was >75% predicted [22], inhaled the saline solution for periods of increasing duration (0.5, 1, 2, 4, and 8 min). The challenge was stopped after the FEV1 had fallen by $\geq 15\%$ (bronchial hyperresponsiveness positive) or if the total inhalation period of 15.5 min had been completed.

Traffic exposure assessment

Average daily traffic counts were performed by the city administration for all streets with an *a priori* estimated vehicle count of $>4{,}000 \cdot day^{-1}$. This resulted in counts being available for 1,840 street segments (of a total of 19,000). In addition, measurements of yearly average concentrations of traffic-associated pollutants (benzene, soot and NO_2) at 18 heavy traffic sites in the city, and from 16 low-to-medium traffic sites were performed [23]. Traffic counts were measured in 1995; air pollution data from December 1996–February 1998.

The street segments with traffic counts and the address of each child were entered into a computer-based geographical information system (GIS). By setting a distance limit of 50 m around each child's home, the program identified all street segments with traffic counts within this distance, and the sum of their daily traffic counts was used to characterise traffic exposure for this child. This distance limit was used according to published data showing that the effect of car traffic decreases greatly approximately beyond this distance [24].

In a second step, the traffic-count categories of 0–50 m and >50–300 m and other available traffic characteristics (traffic-jam percentage) were used to validate a model predicting average yearly pollutant levels as measured at these 34 monitors. The details of this modelling approach are described in another paper [23]. In short, a model using car-traffic counts and a weighting function, to account for the distance between measurement point and street, together with street characteristics (mainly per cent of time with stop-and-go conditions in the segment), was used to derive pollutant estimates. The parameters of this model were optimised to give the best fit for the available actual pollutant-measurement data at the monitoring sites. The monitoring sites had been selected in a way to include both areas of high and low exposure. This model gave a very good estimate of the measured pollutants at low/medium and high exposure locations (benzene R^2=0.80, soot 0.80, NO_2 0.77). This model was then used to calculate predicted pollution levels at each child's home address as exposure estimate.

Statistical analyses

As the expected effect of traffic was rather small, a high-exposure group was defined for comparison with less-exposed children. For traffic counts, the children with one or more traffic counts at ≤ 50 m from their home (16.3% of all children) were considered at high exposure. This high-exposure group was then divided into tertiles to assess a possible dose/response relationship, and was compared with the rest of the

Table 1. – Participation rates

	All Munich children	German children	German children with test#
Parental questionnaire	6244/7509 (83.2)	4777/6198 (77.1)	3953/6198 (63.8)
Skin-prick test	3422/5705 (60.0)	2577/3710 (69.5)	2233/3710 (60.2)
Serum sample	2569/5705 (45.0)	1895/3710 (51.1)	1656/3710 (44.6)
Bronchial challenge	1140/2019 (56.5)	904/1381 (65.5)	771/1318 (55.8)

Data are presented as n/total n (%). #: plus geographically referenced address, stored in the geographical information system.

study sample (83.7%). The threshold distance was later changed to 100 and 300 m for sensitivity analyses to assess consistency of results across various cut-offs. The children with no traffic count $\leqslant$300 m were considered at very low exposure (22.7% of all children).

For the pollutant levels, as derived from the validated model, the same proportion of children (*i.e.* 16.3%) at the upper end of the exposure distribution as in the 50 m limit analysis of the traffic counts was regarded as highly exposed, and this group was again divided into tertiles for the assessment of dose/response effects.

Multiple logistic regression analyses including known relevant confounding variables (age, sex, ETS exposure, SES, family history of asthma, hay fever or eczema) were used to calculate odds ratios for the influence of traffic exposure on the outcome variables. The rationale to stratify for ETS came from another paper published recently by the present group [25], which showed a strong interaction between ETS and low α_1-antitrypsin serum levels resulting in low lung function among children with both exposures. The authors therefore reasoned that ETS might make the airways more susceptible to other potentially damaging factors, such as pollutant exposure.

Results

The questionnaire was distributed to 7,509 children in Munich (table 1). The proportion of children without German nationality was 23.1% (n=1,431) in Munich. As reported elsewhere, reporting behaviour and socioeconomic variables were profoundly different with regard to ethnic background [26]. Therefore, the analysis presented here was restricted to children with German nationality. Of the eligible German children for whom the questionnaire was returned, skin-prick tests were obtained in 2,577 (69.5%, table 1). Table 2 shows the prevalence of health outcomes for all German children.

Table 2. – Prevalences of health outcomes

Current asthma#	203/3946 (5.1)
Current wheeze#	342/3889 (8.8)
Morning cough#	734/3908 (18.8)
Hay fever#	453/3892 (11.6)
Any skin-test reactivity $\geqslant$3 mm	436/2233 (19.5)
Skin-test reactivity pollen $\geqslant$3 mm	317/2224 (14.3)
Skin-test reactivity against indoor allergens $\geqslant$3 mm	256/2233 (11.5)
Specific IgE against aeroallergens $\geqslant$0.7 kU·mL^{-1}	592/1656 (35.8)
Specific IgE against food allergens $\geqslant$0.7 kU·mL^{-1}	181/679 (26.7)
Bronchial hyperreactivity	159/771 (20.6)

Data are presented as n/total n (%). Ig: immunoglobulin. #: with respective symptoms during the last 12 months.

Table 3. – Outcomes and exposures in children participating and nonparticipating in skin-prick testing (SPT)

	With SPT	Without SPT
Current asthma#	4.4 (3.7–5.4)	6.1 (5.0–7.3)
Current wheeze#	8.2 (7.1–9.4)	9.6 (8.3–11.1)
Morning cough#	18.9 (17.3–20.6)	18.7 (16.9–20.6)
Hay fever#	13.1 (11.8–14.6)	9.7 (8.4–11.2)
No traffic count <50 m	83.4 (81.8–84.9)	84.1 (82.3–85.8)
Low traffic counts¶	5.3 (4.5–6.3)	5.4 (4.4–6.6)
Medium traffic counts+	5.7 (4.8–6.7)	5.4 (4.4–6.5)
High traffic counts§	5.6 (4.7–6.6)	5.2 (4.2–6.3)

Data are presented as % (95% confidence interval). #: with respective symptoms during last 12 months; ¶: 2600–15000 vehicles·day^{-1}; +: 15001–30000 vehicles·day^{-1}; §: >30000 vehicles·day^{-1}, in street segment <50 m away from home.

Substantial participation bias is unlikely since the prevalence of outcomes (except for a slightly higher prevalence of hay fever) and the proportion of car-traffic counts were not statistically different in children participating in skin-prick testing compared with those not participating (table 3). When additional socioeconomic information about individual city areas provided by the administration (percentage unemployed, percentage unemployed youth, percentage immigrants, average family size) were used for cluster analysis, no local effects on outcomes were seen (data not shown).

Traffic counts

Traffic counts in street segments varied from 2,600–148,000 vehicles·day^{-1}. Figure 1 shows the places of residence in relation to street segments. SES and ETS exposure were

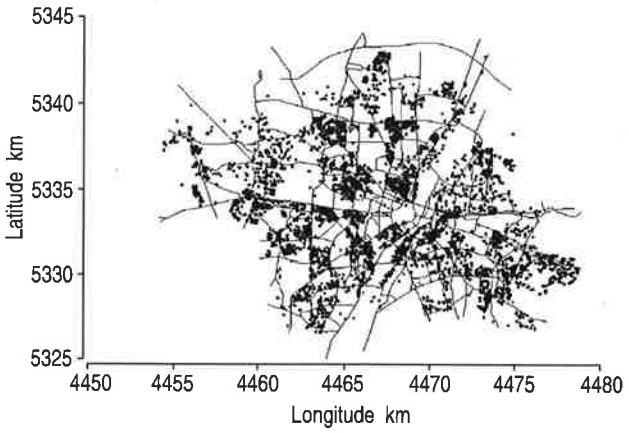

Fig. 1. – Places of residence of children and street segments in Munich.

Table 4.–Respiratory and atopic outcomes in relation to traffic counts in the area of residence

Outcome	Crude reference prevalence % (raw numbers)	Exposure tertile	Crude prevalence % (raw numbers)	Adjusted OR (95% CI)
Asthma	10.4 (318/3071)	Low	9.4 (18/192)	0.902 (0.545–1.493)
		Medium	9.6 (20/208)	0.931 (0.576–1.506)
		High	12.2 (24/197)	1.194 (0.762–1.871)
Current asthma[#]	5.0 (157/3124)	Low	3.1 (6/196)	0.607 (0.264–1.396)
		Medium	5.7 (12/211)	1.177 (0.639–2.171)
		High	8.6 (17/197)	1.790 (1.051–3.048)[§]
Current wheeze[#]	8.6 (266/3085)	Low	7.3 (14/192)	0.848 (0.483–1.488)
		Medium	8.5 (18/211)	1.008 (0.609–1.669)
		High	13.5 (26/193)	1.663 (1.073–2.578)[§]
Cough[¶]	18.0 (559/3097)	Low	18.7 (36/193)	1.049 (0.720–1.528)
		Medium	22.3 (47/211)	1.323 (0.942–1.858)
		High	26.8 (52/194)	1.622 (1.162–2.266)[ƒ]
Hay fever	11.7 (360/3082)	Low	10.9 (21/192)	0.940 (0.585–1.510)
		Medium	12.4 (26/209)	1.061 (0.688–1.638)
		High	13.3 (26/196)	1.171 (0.756–1.814)
Skin-prick test ⩾3 mm	19.4 (341/1762)	Low	17.9 (20/112)	0.846 (0.509–1.405)
		Medium	18.2 (22/121)	0.971 (0.595–1.584)
		High	23.4 (26/111)	1.373 (0.857–2.200)
Pollen	13.9 (243/1754)	Low	14.2 (16/113)	0.961 (0.551–1.677)
		Medium	14.2 (17/120)	1.067 (0.619–1.840)
		High	18.9 (21/111)	1.567 (0.940–2.613)[+]
Specific IgE aeroallergens ⩾0.7 kU·mL^{-1}	36.3 (476/1311)	Low	28.8 (23/80)	0.682 (0.411–1.130)
		Medium	34.7 (34/98)	0.933 (0.600–1.453)
		High	39.5 (32/81)	1.213 (0.755–1.947)

OR: odds ratio; CI: confidence interval; Ig: immunoglobulin; low: 2600–15000 vehicles·day^{-1}; medium: 15001–30000 vehicles·day^{-1}; high: >30000 vehicles·day^{-1} in street segment <50 m away from home. [#]: with respective symptoms during the last 12 months; [¶]: morning cough during the last 12 months. ORs adjusted for age, sex, socioeconomic status, and family history of asthma, hay fever, or eczema. Traffic categories analysed *versus* rest of population (reference). [+]: p=0.05–⩽0.10; [§]: p=0.01–⩽0.05; [ƒ]: p⩽0.01.

linked with traffic exposure. Children living close (⩽50 m) to a busy street (>30,000 cars·day^{-1}) were more often of lower SES (59.3%) compared with the total sample (48.8%) and those (44.3%) with no traffic count ⩽300 m (p<0.0001; Cochran Armitage trend test). When the effect of traffic counts on outcomes was stratified for SES, no effect modification was seen. The same relationship was found for ETS exposure: 51.9% of those living close to a busy street were exposed compared with 40.0% in the total sample and 35.1% in the low traffic areas (p<0.0001).

When traffic counts at ⩽50 m distance were used as an exposure variable and the outcomes were contrasted against the rest of the population (table 4), a significant association was found between total traffic count and cough, current asthma and wheeze, and a dose/response effect was suggested. When stratifying the population into children exposed and not exposed to ETS, an effect modification for ETS was seen. Among children with ETS exposure (table 5), traffic volume was significantly associated with current asthma, a positive skin-prick test and positive skin-prick tests to pollen. A dose/response effect was again observed in this stratum. No significant effects of traffic on lung function and bronchial hyperreactivity were observed. In children without ETS exposure, the effect of traffic was statistically significant only for cough (data not shown).

When children were stratified by skin-prick test positivity, the associations between traffic and outcomes remained significant only for atopic children. This was, however, probably due to the small proportion of children with asthma and asthma symptoms in the nonatopic group leading to very small sample sizes for the highly exposed nonatopic children.

When the distance limit for roads defining high traffic exposure was increased from 50 m up to 100 m, the association of traffic counts with current asthma (in children with ETS exposure with asthma, skin-prick test and sensitisation to pollen) remained significant. For a distance limit of 300 m, the association became less clear but was still significant with cough.

In addition to car-traffic volume, truck-traffic counts (~10% of total counts) were also available. When the latter were used in the analysis, approximately the same associations as with total traffic counts were found with the outcome variables (data not shown).

If the children living in areas with no available traffic count at ⩽300 m distance were analysed separately, a tendency for relatively high prevalences of atopic diseases were found, although NS: 6.3% current asthma (*versus* 4.8% in the rest of the population), 11.9% current hay fever (*versus* 11.7%) and 22.6% had a positive skin-prick test (*versus* 18.7%).

Pollutant concentrations

Estimated exposure to traffic-related pollutants, as calculated from the above-described model for the home of the children, was used as exposure variables in a multivariate logistic regression model. Results for the highest tertiles of high exposure *versus* low exposure are shown in table 6, while the same is presented for children additionally exposed to ETS in table 7.

Cough was associated with soot, benzene and NO_2, current asthma with soot and benzene, and current wheeze with benzene and NO_2, each without a clear dose/response gradient. No effect modification was found for exposure to ETS. No significant associations with lung function and bronchial hyperresponsiveness were detectable for any pollutant (data not shown).

Table 5. – Respiratory and atopic outcomes in relation to traffic counts in the area of residence for children additionally exposed to environmental tobacco smoke

Outcome	Crude reference prevalence % (raw numbers)	Exposure tertile	Crude prevalence % (raw numbers)	Adjusted OR (95% CI)
Asthma	10.8 (126/1169)	Low	4.8 (4/83)	0.438 (0.157–1.222)
		Medium	11.0 (11/100)	1.009 (0.521–1.957)
		High	13.6 (14/103)	1.343 (0.736–2.452)
Current asthma[#]	5.2 (62/1193)	Low	1.2 (1/83)	0.232 (0.032–1.700)
		Medium	5.9 (6/101)	1.130 (0.472–2.706)
		High	9.7 (10/103)	2.047 (1.005–4.171)[§]
Current wheeze[#]	9.1 (107/1178)	Low	4.8 (4/83)	0.523 (0.187–1.461)
		Medium	9.9 (10/101)	1.095 (0.550–2.179)
		High	14.0 (14/100)	1.697 (0.927–3.106)[+]
Cough[¶]	19.1 (226/1186)	Low	20.7 (17/82)	1.177 (0.674–2.054)
		Medium	25.7 (26/101)	1.487 (0.927–2.385)[+]
		High	26.7 (27/101)	1.543 (0.967–2.462)[+]
Hay fever	10.4 (123/1179)	Low	6.1 (5/82)	0.578 (0.227–1.472)
		Medium	16.8 (17/101)	1.678 (0.944–2.981)[+]
		High	15.7 (16/102)	1.739 (0.967–3.126)[+]
Skin-prick test $\geqslant$3 mm	15.8 (110/695)	Low	13.7 (7/51)	0.785 (0.339–1.817)
		Medium	23.8 (15/63)	1.539 (0.812–2.918)
		High	28.8 (15/52)	2.670 (1.353–5.268)[f]
Pollen	11.8 (82/694)	Low	11.5 (6/52)	0.915 (0.372–2.247)
		Medium	19.4 (12/62)	1.662 (0.829–3.331)
		High	25.0 (13/52)	3.255 (1.581–6.699)[f]
Specific IgE aeroallergens $\geqslant$0.7 kU·mL^{-1}	33.1 (164/496)	Low	25.7 (9/35)	0.655 (0.296–1.451)
		Medium	33.3 (16/48)	1.003 (0.524–1.920)
		High	45.0 (18/40)	1.761 (0.897–3.458)[+]

OR: odds ratio; CI: confidence interval; Ig: immunoglobulin; Low: 2600–15000 vehicles·day^{-1}; Medium: 15001–30000 vehicles·day^{-1}; High: >30000 vehicles·day^{-1} in street segment <50 m away from home. [#]: with respective symptoms during the last 12 months. [¶]: morning cough during the last 12 months. ORs adjusted for age, sex, socioeconomic status, and family history of asthma, hay fever, or eczema. Traffic categories analysed *versus* rest of population (reference). [+]: p=0.05–$\leqslant$0.10; [§]: p=0.01–$\leqslant$0.05; [f]: p$\leqslant$0.01.

Table 6. – Respiratory and atopic outcomes in relation to estimated pollutant levels at residence

Outcome	Crude reference prevalence % (raw numbers)	Crude prevalence % (raw numbers)	Adjusted OR (95% CI)
Soot			
Asthma	10.1 (301/2982)	13.2 (26/197)	1.423 (0.920–2.201)
Current asthma	5.0 (152/3031)	8.0 (16/201)	1.763 (1.021–3.044)[¶]
Current wheeze	8.6 (259/2996)	11.2 (22/197)	1.409 (0.883–2.248)
Cough	17.9 (537/3008)	24.1 (48/199)	1.483 (1.055–2.086)[¶]
Hay fever	11.9 (357/2993)	12.8 (25/196)	1.103 (0.708–1.721)
Skin-prick test	20.5 (344/1682)	16.0 (21/131)	0.864 (0.528–1.417)
Pollen	15.2 (255/1675)	12.3 (16/130)	0.904 (0.520–1.573)
Specific IgE aeroallergens	36.8 (463/1258)	37.9 (36/95)	1.124 (0.723–1.747)
Benzene			
Asthma	10.1 (302/2979)	12.4 (25/201)	1.269 (0.816–1.975)
Current asthma	5.1 (153/3029)	9.3 (19/204)	2.045 (1.227–3.407)[+]
Current wheeze	8.8 (263/2992)	13.0 (26/200)	1.646 (1.062–2.552)[¶]
Cough	17.7 (533/3005)	23.4 (47/201)	1.423 (1.010–2.005)[¶]
Hay fever	11.8 (353/2988)	10.9 (22/201)	0.900 (0.565–1.434)
Skin-prick test	20.4 (343/1684)	14.6 (18/123)	0.768 (0.453–1.300)
Pollen	15.0 (251/1678)	9.8 (12/123)	0.688 (0.369–1.283)
Specific IgE aeroallergens	37.4 (469/1255)	34.8 (31/89)	0.983 (0.619–1.563)
NO_2			
Asthma	10.2 (304/2980)	12.1 (24/199)	1.282 (0.817–2.010)
Current asthma	5.1 (154/3029)	7.4 (15/202)	1.655 (0.944–2.901)[#]
Current wheeze	8.8 (263/2992)	12.1 (24/198)	1.579 (1.005–2.482)[¶]
Cough	17.9 (538/3007)	25.5 (51/200)	1.599 (1.144–2.234)[+]
Hay fever	11.8 (352/2990)	12.2 (24/197)	1.038 (0.661–1.630)
Skin-prick test	20.5 (344/1676)	15.4 (20/130)	0.818 (0.495–1.354)
Pollen	15.2 (254/1669)	10.9 (14/129)	0.777 (0.433–1.394)
Specific IgE aeroallergens	37.1 (464/1251)	38.1 (37/97)	1.141 (0.736–1.767)

OR: odds ratio; CI: confidence interval; Ig: immunoglobulin; NO_2: nitrogen dioxide. ORs adjusted for age, sex, socioeconomic status, and family history of asthma, hay fever, or eczema. High exposure tertiles analysed *versus* rest of population. Soot (μg·m^{-3}): 8.07–9.24, >9.24–10.73, >10.73; Benzene (μg·m^{-3}): 4.74–5.31, >5.31–7.27, >7.27; NO_2 (μg·m^{-3}): 42.87–47.15, >47.15–57.44, >57.44. For brevity, ORs (95% CI) and prevalences are only shown for highest traffic exposure tertile for each outcome variable. [#]: p=0.05–$\leqslant$0.10; [¶]: p=0.01–$\leqslant$0.05; [+]: p$\leqslant$0.01.

Table 7. – Respiratory and atopic outcomes in relation to estimated pollutant levels at residence for children additionally exposed to environmental tobacco smoke

Outcome	Crude reference prevalence % (raw number)	Crude prevalence % (raw number)	Adjusted OR (95% CI)
Soot			
Asthma	10.3 (117/1136)	16.7 (16/96)	1.746 (0.978–3.115)#
Current asthma	5.2 (60/1156)	10.1 (10/99)	2.070 (1.013–4.231)¶
Current wheeze	8.9 (102/1144)	13.5 (13/96)	1.582 (0.848–2.950)
Cough	18.9 (218/1151)	27.6 (27/98)	1.668 (1.042–2.670)¶
Hay fever	10.5 (120/1143)	16.3 (16/98)	1.549 (0.862–2.784)
Skin-prick test	17.3 (113/655)	19.7 (13/66)	1.223 (0.629–2.379)
Pollen	13.3 (87/654)	16.7 (11/66)	1.363 (0.669–2.777)
Specific IgE aeroallergens	33.4 (156/467)	34.0 (16/47)	0.980 (0.512–1.874)
Benzene			
Asthma	10.4 (118/1130)	14.6 (15/103)	1.437 (0.797–2.588)
Current asthma	5.2 (60/1150)	11.4 (12/105)	2.407 (1.235–4.692)+
Current wheeze	9.3 (106/1137)	13.7 (14/102)	1.593 (0.871–2.915)
Cough	18.6 (213/1145)	24.3 (25/103)	1.407 (0.873–2.269)
Hay fever	10.5 (119/1137)	15.4 (16/104)	1.432 (0.799–2.565)
Skin-prick test	16.9 (110/651)	19.4 (13/67)	1.274 (0.655–2.479)
Pollen	12.9 (84/651)	13.4 (9/67)	1.109 (0.517–2.377)
Specific IgE aeroallergens	34.3 (158/461)	30.6 (15/49)	0.860 (0.448–1.653)
NO_2			
Asthma	10.3 (117/1131)	15.5 (15/97)	1.563 (0.865–2.826)
Current asthma	5.2 (60/1151)	9.1 (9/99)	1.837 (0.873–3.867)
Current wheeze	9.0 (103/1139)	13.5 (13/96)	1.607 (0.861–2.999)
Cough	18.9 (217/1147)	28.6 (28/98)	1.722 (1.081–2.744)¶
Hay fever	10.3 (117/1138)	16.3 (16/98)	1.538 (0.855–2.766)
Skin-prick test	17.3 (112/649)	21.7 (15/69)	1.442 (0.765–2.718)
Pollen	13.3 (86/648)	17.4 (12/69)	1.487 (0.747–2.963)
Specific IgE aeroallergens	34.0 (157/462)	35.8 (19/53)	1.093 (0.595–2.007)

OR: odds ratio; CI: confidence interval; Ig: immunoglobulin; NO_2: nitrogen dioxide. ORs adjusted for age, sex, socioeconomic status, and family history of asthma, hay fever, or eczema. High exposure tertiles analysed *versus* rest of population. Soot ($\mu g \cdot m^{-3}$): 8.07–9.24, >9.24–10.73, >10.73; Benzene ($\mu g \cdot m^{-3}$): 4.74–5.31, >5.31–7.27, >7.27; NO_2 ($\mu g \cdot m^{-3}$): 42.87–47.15, >47.15–57.44, >57.44. For brevity, ORs (95% CI) and prevalences are only shown for highest traffic exposure tertile for each outcome variable. #: p=0.05–≤0.10; ¶: p=0.01–≤0.05; +: p≤0.01.

Discussion

In this large population-based survey, high vehicle traffic-density close to the home was related to respiratory complaints, such as cough, wheeze and current asthma in children. The authors also found an association between exposure to heavy road traffic and allergic sensitisation for a small subgroup, additionally exposed to ETS. High traffic-related air pollutant exposure was associated with asthma and cough, but not with allergic sensitisation.

Several methodological limitations must be considered before attempting to interpret these findings. The city of Munich provided traffic counts for all street segments with an *a priori* estimate of >4,000 vehicles·day^{-1} only. In this city, most street segments are small side streets and had no counts available. This may have led to some misclassification of traffic exposure. Also, pollutant exposure values at the homes, derived from a validated model, were used to estimate personal exposure rather than direct personal samplers. This approach will only yield an approximation of real exposure.

Children living close to streets with heavy traffic had particular lifestyle characteristics, which make it difficult to differentiate a possible direct effect of car-traffic exposure from these influences. Their families were of a lower SES and their children were more often exposed to ETS. When the analyses were stratified for high/low SES, however, no effect modification for the traffic-related outcomes was seen. No clustering of outcomes was observed with local socioeconomic characteristics of the city areas. Still, a residual confounding effect of SES cannot be excluded, since Munich has no areas with obvious social deprivation. Children living in homes that were least exposed to traffic were at the other end of the socioeconomic spectrum. Atopic diseases have been found more often in affluent families [27, 28]. The beneficial effect of low traffic exposure might actually be obscured, as children from these families can be expected to have higher prevalences of these diseases due to lifestyle factors associated with high SES. Still, these observations must be interpreted with care as the assessment of SES rests on information on parental education and misclassification may have occurred. Interestingly, the study in children living close to a busy freeway in Holland showed an effect on asthma only in children with low-to-medium SES [15].

The advantages of this study are the large number of subjects, the population-based design and the GIS-based assessment of exposure using traffic counts or measurements of air pollutants rather than self-reports. The use of pollution exposure derived from a modelling approach, well validated across high-to-low exposure measurement sites, allows assessment of the plausibility of direct pollutant effects *versus* other traffic-related mechanisms.

Participation or reporting bias is a concern in studies where only subsamples of children take part in some of the measurements (such as skin-prick testing or serum sampling). Participation rates for allergy tests were indeed lower than for the questionnaire (table 1), but as outcomes and traffic exposure categories showed only minor differences between participating and nonparticipating children (table 3), it is

unlikely that bias introduced by this effect can explain these results.

Two interesting observations can be derived from the data presented here. Firstly, high traffic exposure was associated with cough, asthma and wheeze in all children, and with atopic sensitisation in children additionally exposed to ETS. Similar associations with outcomes were seen for traffic-related air pollution levels. The findings regarding asthma are in accordance with those reported for Dutch children living along busy freeways [10, 15]. A survey from Dresden found pollutant effects for bronchitis, nonatopic asthma and cough but not for atopic asthma, allergy and lung function [2]. However, exposure had changed over the lifetime of these children and may have differed during the early years crucial for allergy inception from the exposure measured later on.

A Swiss study reported traffic effects on pollen allergy for a subsample of the adults studied (those living >10 yrs at the same address) [16]. When effects are only found in subsamples in epidemiological studies, the question of multiple comparisons resulting in spurious findings must be considered. Biological plausibility may argue in favour of both the results in the Swiss study and the findings of the study reported here. Recently, ETS exposure was found to be associated with diminished lung function in children with low α_1-antitrypsin levels [25] indicating that ETS exposure together with other detrimental factors may result in overt respiratory damage.

Secondly, when streets >50 m from home were included in the exposure assessment, the traffic effect was diluted. A large study from Italy found effects only for reported lorry traffic at the street of residence, but not in the zone of residence [11]. The same weakening of effects was apparent in this study when mean yearly pollutant levels were used as the exposure variable. No significant effect on allergic sensitisation was seen for pollutant exposure. This could indicate that the observed effects of traffic in streets <50 m from home on atopy are not caused by emissions from vehicles but rather due to residual confounding, *e.g.* socioeconomic characteristics associated with such a place of residence. Although the pollutant exposure assessment gave good results with ~80% predictive power at the measurement stations, misclassification due to the limitations of the modelling approach cannot be excluded. This would equally result in a diminished association between estimated pollutants and outcomes.

A smaller study in 317 children from Düsseldorf, Germany, found exposure to NO_2 as estimated from measured outdoor levels in front of the home (but not the personal sampler derived individual exposure) to be related to skin-prick tests positivity and specific IgE [29]. This could indicate that individual substances may not reflect the mechanism by which the traffic-associated health effects are caused. Alternatively, if traffic exposure at short distance were the cause for the increased prevalence of sensitisation, it would have to be mediated *via* an effect or substance that is less easily dispersed in the surroundings than benzene, soot or NO_2. Larger particles may display such characteristics, and dust containing latex or carbon has been implicated in health effects of traffic [30]. However, a recent study found no increase in latex sensitisation in children living close to busy streets [31].

The less clear association of pollutants compared with traffic counts and lack of dose/response effect suggests that the observed association with traffic may not be mediated directly *via* the measured pollutants. Children living in areas with particularly low traffic exposure also had relatively high atopy prevalence, as well as a higher socioeconomic status. It is also possible that increased outcome prevalences are attributable to differences in lifestyle and/or living conditions related to poverty and low socioeconomic status rather than to traffic exposure itself.

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Living Near a Main Road and the Risk of Wheezing Illness in Children

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The effect of road vehicle traffic pollution on asthma is still not clearly understood. However, any effect is likely to be most marked among those who live within 150 m of a main road, because this is the distance within which concentrations of primary vehicle traffic pollutants are raised above ambient background levels. We have investigated the relation between proximity of the family home to the nearest main road, estimated objectively using geographical information system software, and the risk of wheeze in the past year in a case-control sample of 6,147 primary schoolchildren (age 4 to 11 yr) and a random cross-sectional sample of 3,709 secondary schoolchildren (age 11 to 16 yr) in Nottingham, United Kingdom. Among children living within 150 m of a main road, the risk of wheeze increased with increasing proximity by an odds ratio (OR) of 1.08 (95% confidence interval [CI] 1.00 to 1.16) per 30-m increment in primary schoolchildren, and 1.16 (1.02 to 1.32) in secondary schoolchildren. Most of the increased risk was localized to within 90 m of the roadside. Among primary schoolchildren, effects were stronger in girls than boys ($p_{interaction}$ = 0.02). Living within approximately 90 m of a main road is associated with a proximity-related increase in the risk of wheezing illness in children.

Keywords: asthma; wheezing; children; vehicular emissions

Motor vehicle emissions are a major source of outdoor pollution throughout the world and there is widespread public concern over their effect on asthma, particularly among children. We have previously reported that average traffic activity in the vicinity of schools in Nottingham (1 km^2 surrounding area) is not a major determinant of the risk of asthma in children attending those schools (1), but this may be due to the fact that substantial differences in average traffic flows in 1 km^2 districts do not necessarily translate into equivalent differences in personal exposure to pollution. Data for the main vehicle exhaust pollutants (oxides of nitrogen, carbon monoxide, particulates, and hydrocarbons) indicate that the major contrasts in pollution levels exist near roads, being very high at the roadside and decreasing exponentially with distance from the curbside to reach a plateau by approximately 150 m (2). It therefore follows that increased exposure to vehicle exhaust emissions is likely to occur in those children who live, or spend a large proportion of time, within approximately 150 m of a busy road. Furthermore, we might expect any effect of proximity to a main road on asthma to follow an exposure–response relation within this distance range. Some previous studies have suggested that respiratory morbidity may be increased in children who live within 100 m of a main road (3), or on a busy road relative to a quiet road (4–7), and one study has reported similar findings in adults (8). However, the evidence is not all consistent (9–11), possibly because some of the studies reporting a relationship have relied on potentially biased self-reported measures of exposure (5–7). Also, those studies that have used objective estimates of distance from roads have analyzed these estimates categorized into binary measures, and as a result the nature of the exposure–effect relationship with increasing proximity of residence to road vehicle traffic remains undefined.

We have therefore used objective spatial analysis techniques in a geographical information system (GIS) to investigate the relation between proximity of the home to the nearest main road and wheezing in primary and secondary schoolchildren in Nottingham, United Kingdom, and in particular, to characterize the nature of this relation within the approximate 150 m range in which pollutant levels are increased. Because several previous studies have suggested that the effects of road traffic pollution on respiratory morbidity may be greater in girls than in boys (3, 4, 12, 13), we have also looked for evidence of a difference in the effects of proximity to major roads on the risk of wheezing in girls and boys in the present study.

METHODS

In 1995 and 1996 we surveyed respiratory symptoms in schoolchildren living in and around Nottingham, for a study of local school traffic density effects on asthma (1), using parental questionnaires for primary schoolchildren (age 4 to 11 yr) (14), and self-completion questionnaires in secondary schoolchildren (age 11 to 16 yr) (15). For the present study we used more detailed data collected on respiratory symptoms, lifestyle factors, and residential postcode (exact addresses were not collected) by parental questionnaire in a nested case-control sample of the primary schoolchildren and a random one-in-four subsample of secondary schoolchildren (1). The primary outcome variable in both groups was parent-reported wheeze in the past year.

Distance from the child's home to the nearest main road (motorway, A or B class road) was computed using GIS software (PC ArcInfo, version 3.5; Environmental Research Systems Institute Inc. Redlands, CA). Home postcodes in the U.K. refer to an average of 15 adjacent houses, and we used grid references of 1-m resolution representing the midpoint of each postcode (Ordnance Survey, Southampton, UK) as an estimate of location for each house. We used ArcInfo to link these to a 0.1-m resolution digitized map of all main roads in Nottinghamshire (Meridian digitized road data, version 2.0; Ordnance Survey, Southampton, U.K.), and to compute the shortest distance between each home location and the nearest main road in meters.

The effect of distance from the road on the odds of wheeze was analyzed using logistic regression (SPSS version 7.5; SPSS Inc., Chicago, IL) initially across the full range of distances in quartiles, and then among those living within 150 m of a main road (approximately the lowest quartile of distance) in 30-m intervals. Potential confounding by age, sex, preterm birth, birth weight, maternal age, maternal smoking during pregnancy, parental smoking in infancy, current parental smoking, month of birth, duration of breast-feeding, birth order, and social deprivation was assessed, and odds ratios (OR) adjusted for age, sex, and those variables found to be independently associated with wheeze in the group are presented. Our primary measure of social deprivation was the Carstairs Deprivation Score (16), computed by residential area from 1991 census data, although we also

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explored adjusting for an individual-based measure of deprivation computed from parental occupation. In the secondary schoolchildren we controlled for the age–sex interaction previously reported in this dataset (15). To look for effect modification by sex, a sex–distance interaction term was added to the model.

Our sample sizes provided more than 80% power to detect ORs for the nearest quartile relative to the farthest of 1.25 for primary schoolchildren and 1.4 for secondary schoolchildren, and for those living within 150 m of a main road, 80% power to detect ORs for the closest relative to the most distant 30-m interval of 1.55 for primary groups and 2.05 for secondary groups.

The study was approved by the Nottingham Director of Education and Nottingham City Hospital Ethics Committee.

RESULTS

Primary Schoolchildren

Of the 6,576 primary schoolchildren in the case-control sample (78% of those eligible for inclusion), we were able to georeference 6,147 (93%, Table 1). In this full sample, quartile of distance of the home from a main road was not significantly related to wheeze (OR = 1.02, 0.90, and 0.97 for the first three quartiles relative to the farthest, p = 0.36) and there was no evidence of a trend across the quartiles (p_{trend} = 0.97) either before or after adjustment for potential confounders. However, in the 1,541 children living within 150 m of a main road there was a trend toward an increase in risk of wheeze with increasing proximity to the road, which after adjustment for age, sex, and other risk factors for wheeze in this group (Carstairs Score and preterm birth) was of borderline significance (adjusted OR per 30-m increment = 1.08, 95% confidence interval [CI] 1.00 to 1.16, p_{trend} = 0.06). A plot of adjusted ORs for each distance category demonstrates that most of this effect occurred within 90 m of roadside (Figure 1). There was a significant interaction with sex (p = 0.02), such that the effect of distance was strong in girls (adjusted OR per 30-m increment = 1.17, 95% CI 1.05 to 1.31), but absent in boys (adjusted OR = 0.99, 95% CI 0.89 to 1.10, Figure 2). When adjustment was made for social class based on parental occupation rather than Carstairs Score, the magnitudes of these ORs were mildly reduced (adjusted OR per 30-m increment = 1.14 [95% CI 1.01 to 1.28] in girls and 0.98 [0.87 to 1.09] in boys).

Secondary Schoolchildren

Of the 3,880 secondary schoolchildren in the cross-sectional sample with a completed parental response to the wheeze variable (59% of those selected for inclusion), we were able to georeference 3,709 (96%) (Table 1). Some heterogeneity between the quartiles of distance was identified (OR = 0.93, 1.09, and 0.74 for the first three quartiles relative to the farthest, p = 0.02), but with no obvious trend (p_{trend} = 0.68), either before or after control for potential confounding. However, in those 859 children living within 150 m of a road, there was a positive association between the risk of wheeze and proximity to the roadside, which remained statistically significant after adjustment for other risk factors for wheeze in this group, age, sex, age–sex interaction, Carstairs Score, preterm birth and maternal smoking in infancy (adjusted OR per 30-m increment = 1.16, 95% CI 1.02 to 1.32, p_{trend} = 0.03). The greatest increase in risk occurred within 60 m of the roadside (Figure 3). Slightly stronger effects were seen among secondary school girls than boys (adjusted OR per 30-m increment = 1.19 [95% CI 0.99 to 1.43] for girls and 1.13 [0.94 to 1.36] for boys), but this interaction was not statistically significant (p = 0.65). Substituting social class based on parental occupation in place of Carstairs Score in the model resulted in similar findings (adjusted OR per 30-m increment = 1.14, 95% CI 1.00 to 1.31). Associations of similar magnitude were identified in this cross-sectional group for cough and diagnosed asthma (adjusted OR per 30-m increment = 1.12 [95% CI 0.98 to 1.28] and 1.15 [0.97 to 1.38] respectively), but there was no relation between proximity to roadside and eczema or hay fever.

TABLE 1. CHARACTERISTICS OF PRIMARY AND SECONDARY SCHOOL SAMPLES

	Primary School Case-control	Secondary School Cross-sectional
Number in sample	6,576	3,880
Number (%) georeferenced	6,147 (93%)	3,709 (96%)
Of which:		
number (%) boys	3,204 (52%)	1,805 (49%)
number (%) wheeze in past year	2,483 (40%)	608 (16%)
median distance to main road, m	321	372
interquartile range of distance, m	149–700	163–742
number (%) living ≤ 150 m from main road	1,541 (25%)	859 (23%)
number (%) living ≤ 30 m from main road	214 (3.5%)	125 (3.4%)

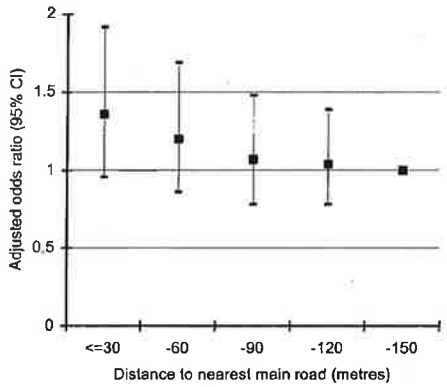

Figure 1. Exposure–response relation between distance of the home from nearest main road and parent-reported wheeze in primary schoolchildren.

DISCUSSION

This study provides evidence that the risk of wheeze in children is increased in relation to proximity to main roads, as might be expected from the profile of traffic pollutant levels close to the road (2), and that this increase in risk applies primarily to those living less than approximately 90 m from the roadside. This suggests that adverse effects of road vehicle pollution are relatively localized, and may explain previous observations by us (1) and others (5, 17) that traffic activity in the wider locality of school or home, in contrast to activity within about 90 m, is not a major determinant of respiratory morbidity.

We used objective estimates of distance between the home and the nearest classified or numbered road, which at the time of the study in the Nottingham area typically carried between 10,000 and 100,000 vehicles per day, as a marker of exposure to the mix of pollutants arising from exhaust emissions. Although this measure of distance is objective and therefore free from reporting bias, it is still an approximate estimate of exposure because individual exposure is also likely to be influenced by the volume and flow of traffic on the road, by the amount of time spent at home, and by the presence of physical obstructions between the road and the home. Also, the postcode

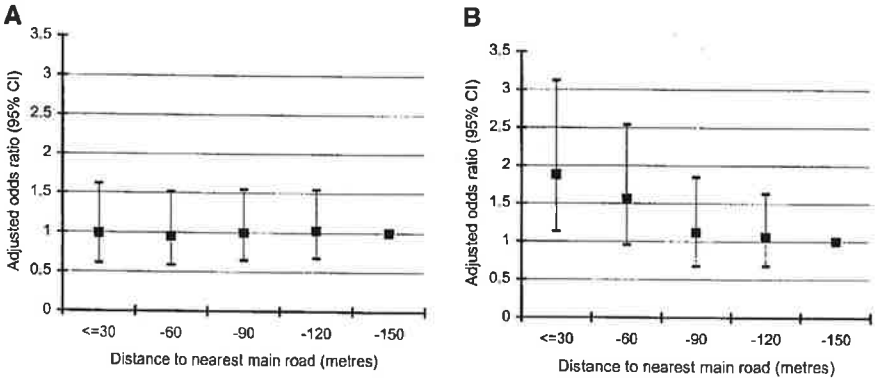

Figure 2. Exposure–repsonse relation between distance of home from nearest main road and parent-reported wheeze in primary (*A*) boys and (*B*) girls.

references we used apply to groups of about 15 adjacent houses and therefore will have resulted in some misclassification. However, this misclassification is not systematic and will therefore only reduce the magnitude of our estimates of effect. We were unable to estimate home location more precisely because we did not have exact home addresses available to us. We chose parent-reported wheeze in the past year as our primary outcome variable to avoid the diagnostic and labeling biases inherent in a diagnosis of asthma, and for consistency between the two school groups, though data on other parent-reported respiratory disease markers available in the secondary schoolchildren provided consistent findings. Although the possibility of response or reporting bias in parents of those living on main roads cannot be ruled out, parents were not aware of the hypothesis being tested, and such bias is also unlikely to explain the clear difference in effect between the sexes in primary schoolchildren.

Two previous studies have shown associations between objective measures of proximity to main roads and respiratory symptoms consistent with our findings in children (3, 4), as has one study in adults (8). Other studies have failed to find an effect on treated asthma (9), or hospital admission for asthma (10, 11), but these used larger distances to define a binary measure of proximity to a main road. Oosterlee and coworkers reported a 50% increase in wheeze in the past year among Dutch children living on a busy street relative to living on a quiet street (4), and another study of Dutch children reported a doubling of wheeze prevalence in children living within 100 m of a freeway (3), but no effect on lung function (12). Both studies reported larger estimates for girls than boys, and a similar predominance in girls has been reported for associations between truck traffic density and lung function (12), nitrogen dioxide exposure and wheezing bronchitis (13), and passive exposure to tobacco smoke and bronchial responsiveness (18). In the light of these previous observations, we looked for differences in effect between sexes and found significant evidence of a greater effect in primary, but not secondary girls. Why girls appear more susceptible to the impact of pollution than boys is unclear.

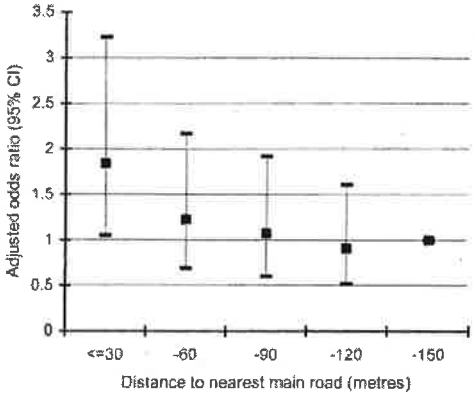

Figure 3. Exposure–response relation between distance of home from nearest main road and parent-reported wheeze in secondary schoolchildren.

The consistency of our findings between the two independent datasets, and the exposure–response association within the distance over which concentrations of the main primary pollutants arising from vehicle exhaust emissions are known to be increased, support a causal effect of exposure to road traffic pollution on wheezing illness in children. If true, this has important health implications for the minority of the population (approximately 12% in the Nottingham area) who live within about 90 m of a main road. Further work is now required to determine which specific pollutant or pollutants are likely to be responsible, why girls appear to be more susceptible than boys, and the extent to which risks of cardiovascular and other pollution-related disorders are also increased in relation to this exposure in children and adults.

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Air Pollution from Traffic at the Residence of Children with Cancer

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The hypothesis that exposure to traffic-related air pollution increases the risk of developing cancer during childhood was investigated. The authors enrolled 1,989 children reported to the Danish Cancer Registry with a diagnosis of leukemia, tumor of the central nervous system, or malignant lymphoma during 1968–1991 and 5,506 control children selected at random from the entire childhood population. The residential histories of the children were traced from 9 months before birth until the time of diagnosis of the cases and a similar period for the controls. For each of the 18,440 identified addresses, information on traffic and the configuration of streets and buildings was collected. Average concentrations of benzene and nitrogen dioxide (indicators of traffic-related air pollution) were calculated for the relevant period, and exposures to air pollution during pregnancy and during childhood were calculated separately. The risks of leukemia, central nervous system tumors, and all selected cancers combined were not linked to exposure to benzene or nitrogen dioxide during either period. The risk of lymphomas increased by 25% (p for trend = 0.06) and 51% (p for trend = 0.05) for a doubling of the concentration of benzene and nitrogen dioxide, respectively, during the pregnancy. The association was restricted to Hodgkin's disease. *Am J Epidemiol* 2001;153:433–43.

air pollution; benzene; child; Hodgkin disease; neoplasms; nitrogen dioxide

In many countries, road traffic is the major source of ambient air pollution in urban areas, where most people live. Air pollution from traffic is a complex mixture of many chemicals, of which several are known or suspected carcinogens (1). The evidence for the carcinogenicity of traffic exhaust derives mainly from experiments in animals and studies of occupationally exposed persons. In 1989, however, a case-control study performed in Denver, Colorado, showed elevated risks of cancer among children living near streets with high traffic density (2), and two studies from Sweden (3) and the United Kingdom (4) indicated an elevated risk of cancer among children living at addresses with high levels of ambient air pollution. These results have raised great public concern. The shortcomings of these three studies include limited sample sizes and crude methods for assessing exposure based on only part of the residential history of the children. Large studies with improved methods are required to address the hypothesis and the public concern properly.

The present study was designed as a case-control study to test the hypothesis that air pollution from traffic causes cancer in childhood. The level and extent of past exposure to exhaust fumes at the residences of children with the most common types of cancer were evaluated and compared with those of a population-based, random sample of children without cancer. The exposure assessment method was validated against measurements of air pollution.

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MATERIALS AND METHODS

Study population

The cases of cancer in this study were those of children born after December 31, 1959, who received a diagnosis of leukemia, tumor of the central nervous system, or malignant lymphoma when they were under the age of 15 years, during the period from January 1, 1968, to December 31, 1991. They were identified from the files of the Danish Cancer Registry, which also provided the name and personal identification number. This number, which is unique to every Danish citizen, incorporates sex and date of birth and permits accurate linkage of information among registers. The nationwide cancer registration system, set up in 1942, has been described and evaluated previously (5). The register also includes histologically benign tumors of the brain and intracranial meninges. Children born outside Denmark were excluded, leaving a total of 1,989 children with cancer for the study (table 1).

Control children were drawn from the files of the Danish Central Population Registry and likewise identified by name and personal identification number. They had to have been born in Denmark and, at the time of the diagnosis of the corresponding case, to have been alive without a diagnosis of cancer and residing in Denmark. Two, three, and five controls matched by sex, age (exact), and calendar time (within

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TABLE 1. Cases of childhood cancer diagnosed in Denmark during 1968–1991 among children born in 1960 or later and matched controls

Diagnostic group* and morphologic subtype	Cases (no.)	Controls (no.)	Total (no.)	Cases: controls
Leukemia	986	1,972	2,958	1:2
Acute lymphocytic	731			
Acute nonlymphocytic	127			
Other/unknown subtypes	128			
Central nervous system tumors	740	2,220	2,960	1:3
Ependymoma	79			
Astrocytoma	263			
Medulloblastoma	148			
Other/unknown subtypes	250			
Malignant lymphoma	263	1,314	1,577	1:5†
Hodgkin's disease	84			
Non-Hodgkin's lymphoma	179			
All types combined	1,989	5,506	7,495	

* Based on the *International Classification of Diseases for Oncology* (ICD-O).
† The data check revealed a control child who had died 3 years before diagnosis of the corresponding case; this control was excluded, so that one case of lymphoma had only four controls.

1 year) were chosen for each case of leukemia, central nervous system tumor, and malignant lymphoma, respectively, for a total of 5,506 children included as controls (table 1).

Residential history

Information on the parents of case children and control children and on the number of the index child in the sibship (birth order) was obtained from the files of the Central Population Registry. The residential histories of each family were ascertained retrospectively from the date of diagnosis of cancer or a similar date for the matched control children (referred to in the following as "date of diagnosis") to 9 months before the child's birth. Addresses were provided jointly by the Central Population Registry and the 275 local population registries of Denmark. Each location was identified according to county, municipality, town, postal code, street, building number, floor, and side; the dates of moving in and leaving the address were noted. The families were not approached directly. Altogether, we identified 18,440 addresses at which the 7,495 pregnant mothers and children had lived. The residential history was incomplete for 301 families (4 percent); no information at all was available for 98 mothers during the pregnancy (20 cases, 78 controls) and for nine children during childhood (one case, eight controls).

Exposure assessment

Benzene and nitrogen dioxide (NO_2) are commonly used markers of traffic-related air pollution and were used as such in this study. The average concentrations of benzene and NO_2 at the front door of each dwelling during the period that the families occupied the address were assessed by use of a modified version of the Operational Street Pollution Model (6–12). Briefly, the model can be used to calculate air pollution levels from information on the exact configuration of the street in front of the address (the width of the street, the height of the buildings and their distance from the street, and street sections with no buildings), the traffic density on the street (vehicles per day), the proportion of vehicles weighing more than 3,500 kg, the average traffic speed (km/h), and the presence of other streets within 50 m with a traffic density higher than that on the index street, in combination with information on emission factors (g/km) for the Danish car fleet, meteorologic variables (wind speed, temperature, and solar radiation), and background levels of air pollution.

The data on the street configuration, the traffic pattern, and the degree of urban development for each of the 18,440 addresses occupied by the 7,495 study subjects were collected for the relevant period by use of a registration scheme designed for this purpose (12). Staff from each of the 275 municipal highway departments of Denmark filled in a registration form for each address in their municipality, without knowledge of the case or control status of the children. All of the 18,440 schemes were filled in and returned. Meteorologic data, obtained from Copenhagen Airport, were considered to be representative of Denmark as a whole. The geographic distribution and temporal variation in background air pollution levels were estimated from measurements made during 1994–1995 at four sites in different parts of Denmark and from information on changes in air pollution emissions in Denmark since 1960 (13).

Potential confounding factors

The degree of urban development was calculated as the average category of all residences of the child, weighted by the length of each residential period. The categories were as follows: 1, rural area; 2, <2,000; 3, 2,000–19,999; 4, 20,000–39,999; 5, 40,000–79,999; 6, 80,000–149,999; and 7,

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≥150,000 inhabitants in the town. The type of residence was scored according to the proportion of time the child had lived in a one-family house (villa, row house, farmhouse, etc.) or an apartment. The address at birth was used to allocate each child to one of eight geographic regions of Denmark. Trained employees of the Danish electric utility companies investigated each address for proximity to 50- to 400-kV overhead lines, underground cables, or transformer stations during the relevant residential period, and the average electromagnetic field strength at the address was calculated (14). Moreover, we considered the mother's age and the birth order of the child as potential confounding factors. The degree of urban development and the type of residence were considered for the pregnancy and the childhood period separately, whereas exposure to an electromagnetic field was assessed for the two periods combined.

Statistical analyses

The risk of childhood cancer was analyzed according to the traffic density and air pollution level at the residence during the pregnancy and during childhood. Exposure of the mothers to benzene and NO_2 was summarized as "exposure level × time" for each address occupied from the presumed date of conception until the date of birth of the child. The residential history of 98 mothers was unknown for the entire pregnancy, leaving 7,397 children (99 percent) available for these analyses.

Cumulated exposure during childhood was calculated from the date of birth until 12 months before the date of diagnosis, with an additional requirement of at least 6 months of follow-up of the exposure of the child. Thus, 697 children (cases and matched controls) in whom cancer had been diagnosed before they reached 18 months of age were not included in the analyses of childhood exposure, and no information on exposure was available for nine of the remaining children, leaving 6,789 children (91 percent) available for these analyses. The time period for which exposure was calculated was exactly equal for a case and the matched controls.

If a pregnant mother or a child had lived at an unidentified address during part of the relevant period, that address was assigned the exposure level that corresponded to the time-weighted average at the known addresses.

Because of the nested case-control design of the study, with equal follow-up time for a case and the matched controls, the rate ratios (15) (henceforth denoted relative risks) for childhood cancer were estimated by conditional logistic regression analysis with the "Phreg" procedure of SAS (16). Four categories of exposure to air pollution were defined, with the 50th, 90th, and 99th percentiles as cutoff points. Exposure was log-transformed in the trend analyses, such that an excessive influence of a few extremely high values was avoided, and a doubling of the exposure corresponded to a fixed relative risk independently of the exposure level. Trend tests for residential traffic density were done similarly.

Validation of exposure assessment

We estimated how many of the children allocated to each category of exposure could be expected to have been correctly classified. In a separate validation study, we included 204 children living in Copenhagen or in rural areas outside Copenhagen and measured the NO_2 concentration outside the front door of each dwelling during 6 months (11) and the personal exposure to NO_2 of each child during 1 week (17). Following exactly the same procedures as in the case-control study, we collected information about streets, buildings, and traffic and calculated the concentrations of NO_2 at each residence. Thus, the validation study provided information about the relation between 1) the calculated and measured half-year average concentration of NO_2 at the front door and 2) the calculated 1-week average concentration of NO_2 at the front door and simultaneously measured personal exposure.

For every calculated average concentration of NO_2 at the front door in the case-control study during the pregnancy, we estimated the probability of falling into different categories of measured concentrations; that is, we fitted a linear relation, with a slope fixed at 1, between the log-transformed calculated and measured concentrations of NO_2 and used this line and the estimated standard deviation around the line to predict the probability. The probabilities for each individual were accumulated into expected numbers of children within each category, thus providing the expected number of misclassified children in each category of exposure in the case-control study. Cutoff points among the different categories of measured exposure were defined to obtain the same expected numbers of children in each category as in the corresponding category of the case-control study. The statistical method for estimating the distribution of the measured concentrations has been described by Secher et al. (18). The same procedure was used for personal exposures, except that the relation between personal exposure and the calculated front-door concentration was estimated by standard linear regression.

RESULTS

Cases and controls were similar with regard to the mother's age, birth order, residential region at the time of birth, degree of urban development, the proportion of time they had lived in a one-family house, and exposure to an electromagnetic field, although a slightly smaller proportion of case than control children was born in the Copenhagen area and a slightly larger proportion of case children had lived in a one-family house (table 2).

Traffic density

No increased risk of leukemia, central nervous system tumors, lymphomas, or these diagnoses combined (denoted "all cancers") was associated with exposure to high traffic density during pregnancy or childhood in comparison with exposure to less than 500 vehicles per day, even when the density was more than 10,000 vehicles per day at the residence. The trend analyses showed no significant results (table 3).

Air pollution

The predicted exposures to the two different indicators of traffic-related air pollution were correlated; the corre-

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TABLE 2. Characteristics of 1,989 children with cancer (cases) and 5,506 control children, Denmark, 1968–1991

Characteristic	Cases		Controls	
	No.	%	No.	%
Mother's age at birth of the child (years)				
<20	154	7.7	427	7.8
20–24	674	33.9	1,918	34.8
25–29	678	34.1	1,904	34.6
30–34	349	17.5	906	16.5
≥35	125	6.5	346	6.3
Missing value	9	0.5	5	0.1
Birth order of child				
1	871	43.8	2,459	44.7
2	734	36.9	1,981	36.0
3	268	13.5	777	14.1
≥4	106	5.3	280	5.1
Missing value	10	0.5	9	0.2
Residential region at birth				
Copenhagen	211	10.6	639	11.6
Suburbs of Copenhagen	227	11.4	700	12.7
Other parts of Sealand	365	18.4	1,004	18.2
Bornholm	21	1.1	56	1.0
Funen	180	9.0	451	8.2
East Jutland	330	16.6	893	16.2
West Jutland	255	12.8	649	11.8
North Jutland	380	19.1	1,034	18.8
Missing value	20	1.0	80	1.5
Urban development by category*,†				
≤1.0–<1.5	254	12.8	680	12.4
1.5–<2.5	368	18.5	1,031	18.7
2.5–<3.5	525	26.4	1,495	27.2
3.5–<4.5	274	13.8	814	14.8
4.5–<5.5	211	10.6	593	10.8
5.5–<6.5	148	7.4	319	5.8
6.5–≤7.0	208	10.5	566	10.3
Missing value	1	0.1	8	0.1
Proportion of time† (%) lived in a one-family house‡				
0	285	14.3	787	14.3
0–100	569	28.6	1,699	30.9
100	1,134	57.0	3,012	54.7
Missing value	1	0.1	8	0.1
Electromagnetic field at residence (μT)†,§				
<0.1	1,975	99.3	5,467	99.3
0.1–<0.4	6	0.3	21	0.4
≥0.4	7	0.4	10	0.2
Missing value	1	0.1	8	0.1

* Time-weighted average of the categories of urban development for all addresses. The categories were as follows: 1, no town (rural areas); 2, <2,000; 3, 2,000–19,999; 4, 20,000–39,999; 5, 40,000–79,999; 6, 80,000–149,999; and 7, ≥150,000 inhabitants.

† During the period from the time of conception to the time of diagnosis.

‡ In contrast to an apartment.

§ Highest level at any address.

lation coefficient (Pearson's) was 0.71 for the pregnancy (n = 7,379) and 0.83 for the childhood period (n = 6,789).

The results showed no increased risk of developing "all cancers" in association with the three higher categories of exposure when the lowest category was used as reference

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TABLE 3. Crude relative risks (RRs) and 95% confidence intervals (CIs) for childhood cancers associated with residential traffic density, Denmark, 1968–1991

Traffic density* (vehicles per day)	All cancers†		Leukemias		CNS tumors		Lymphomas	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Pregnancy period								
<500	1.0		1.0		1.0		1.0	
500–<5,000	1.0	0.9, 1.1	0.9	0.8, 1.0	1.0	0.9, 1.2	1.0	0.8, 1.4
5,000–<10,000	0.9	0.7, 1.2	0.8	0.6, 1.2	0.7	0.5, 1.2	1.7	1.0, 2.8
≥10,000	0.7	0.5, 1.1	0.8	0.5, 1.3	0.6	0.3, 1.1	1.2	0.5, 3.0
Trend‡	0.99	0.97, 1.01	0.98	0.95, 1.01	0.99	0.95, 1.02	1.03	0.98, 1.09
No. of cases/no. of controls§	1,969/5,378¶		975/1,934		733/2,165		261/1,279	
Childhood period								
<500	1.0		1.0		1.0		1.0	
500–<5,000	0.9	0.8, 1.0	0.9	0.8, 1.1	0.9	0.7, 1.0	0.9	0.7, 1.2
5,000–<10,000	0.8	0.6, 1.1	0.8	0.5, 1.2	0.6	0.4, 1.1	1.5	0.8, 3.0
≥10,000	1.0	0.7, 1.6	1.1	0.6, 2.2	0.9	0.4, 1.8	1.3	0.4, 4.8
Trend‡	0.99	0.96, 1.01	0.99	0.96, 1.02	0.98	0.94, 1.01	1.00	0.94, 1.06
No. of cases/no. of controls	1,796/4,990		883/1,765		666/1,995		247/1,230	

* Time-weighted average for the addresses occupied during the pregnancy and childhood, respectively.
† Leukemias, central nervous system (CNS) tumors, and lymphomas considered together.
‡ In the trend analyses, traffic density was entered as a continuous variable, disregarding the four exposure groups. The estimates show the relative risk corresponding to a doubling of the traffic density.
§ Numbers of case and control children used in the analyses. The numbers are marginally lower than the numbers of available children, because the controls matched to cases with missing values for traffic density could not be used in the analyses.
¶ When case and control children were considered together, 60% were exposed to less than 500, 33% to 500–<5,000, 5% to 5,000–<10,000, and 2% to 10,000 vehicles or more per day.

(table 4). No effect was seen even in the highest categories of exposure to both pollutants, and this result was the same for both exposure periods. The trend analyses indicated no effect of air pollution on the risk of "all cancers." The unadjusted (crude) and adjusted relative risks differed very little. Similarly, the risks of developing the two major types of childhood cancer, leukemias and central nervous system tumors, were not increased at the higher categories of exposure, and no significant trends were found, no matter which pollutant or exposure period was considered. Actually, a weak, nonsignificant tendency for an inverse association between exposure and the risk of these two types of cancer was seen in most cases.

The results indicated an association between exposure in utero and the risk of developing lymphomas (table 4): The relative risks increased with increasing category of exposure to both pollutants. The exception was the highest category of exposure to benzene; however, the confidence interval was wide for this relative risk estimate. The exposure-response pattern was confirmed in the trend analyses, which showed a 25 percent (p for trend = 0.06) and a 51 percent (p for trend = 0.05) increase in risk of doubling the concentrations of benzene and NO_2, respectively. The association between the risk of lymphomas and exposure during childhood was less consistent. The highest category of exposure to benzene was associated with a decreased risk, but the opposite was observed for exposure to NO_2. Correspondingly, the trend estimates indicated effects in opposite directions for the two markers of traffic-related air pollution but were not significantly different from the neutral value (both p for trend > 0.80) (table 4).

Analyses by morphologic subtype showed that the increased risk of lymphomas in association with exposure to benzene and NO_2 in utero was restricted to Hodgkin's disease. In the adjusted analyses, the highest categories of exposure to benzene and NO_2 resulted in 4.3 and 6.7 times higher risks of Hodgkin's disease than the reference category (table 5). The trend analyses showed an 84 percent (p for trend = 0.005) and a 147 percent (p for trend = 0.02) increase in risk of doubling the concentrations of benzene and NO_2, respectively. Separate analyses for children below and above the median age at diagnosis (11.7 years) showed that the association was restricted to children who received their diagnosis at younger ages (p for trend = 0.001 for benzene and 0.009 for NO_2; data not shown). Unadjusted analyses showed weaker associations of only borderline significance; the difference between the crude and adjusted risk estimates (table 5) was caused primarily by adjustment for degree of urban development. There was no significant relation between the risk of Hodgkin's disease and exposure during childhood. Moreover, the results showed no significantly increased risk of non-Hodgkin's lymphoma or any of the morphologic subtypes of leukemia (acute lymphocytic leukemia, acute nonlymphocytic leukemia) or of central nervous system tumors (ependymoma, astrocytoma, medulloblastoma) in association with exposure to benzene or NO_2 during any of the exposure periods (data not shown). An analysis restricted to children in whom acute lymphocytic leukemia was diagnosed when they were below age 5 (the childhood peak) and the matched control children showed no increase in risk in association with exposure to air pollution.

All of the analyses of the associations with air pollution were repeated after exclusion of children whose residential history was not fully identified. This had only minor effects on the results.

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TABLE 4. Crude and adjusted* relative risks (RRs) and 95% confidence intervals (CIs) for childhood cancers associated with air pollution at the residence, Denmark, 1968–1991

Category of exposure (cutoff points in 1,000 ppb-days)†	All cancers‡			Leukemias			CNS tumors			Lymphomas		
	Crude RR	Adjusted RR	Adjusted 95% CI	Crude RR	Adjusted RR	Adjusted 95% CI	Crude RR	Adjusted RR	Adjusted 95% CI	Crude RR	Adjusted RR	Adjusted 95% CI
Pregnancy period												
Benzene												
<0.5	1.0	1.0		1.0	1.0		1.0	1.0		1.0	1.0	
0.5–<1.3	1.0	1.0	0.9, 1.2	1.0	1.1	0.9, 1.3	0.9	0.9	0.7, 1.1	1.2	1.3	0.9, 1.9
1.3–<3.5	1.0	1.0	0.8, 1.3	0.8	0.9	0.6, 1.3	0.9	0.8	0.5, 1.2	2.0	2.2	1.2, 3.9
≥3.5	0.6	0.6	0.3, 1.2	0.7	0.8	0.4, 1.9	0.4	0.4	0.1, 1.3	1.0	1.2	0.2, 5.9
Trend§	0.96	0.97	0.88, 1.06	0.90	0.95	0.82, 1.09	0.95	0.91	0.78, 1.06	1.19	1.25	0.99, 1.58
NO_2												
<1.5	1.0	1.0		1.0	1.0		1.0	1.0		1.0	1.0	
1.5–<3.0	1.0	1.0	0.9, 1.2	1.0	1.1	0.8, 1.3	0.9	1.0	0.7, 1.2	1.2	1.4	0.9, 2.1
3.0–<5.3	0.9	1.1	0.8, 1.5	0.8	1.2	0.8, 2.0	1.0	1.0	0.6, 1.5	1.3	1.8	0.8, 3.9
≥5.3	0.9	1.1	0.6, 2.0	0.5	0.8	0.3, 2.2	1.0	1.0	0.4, 2.5	2.2	3.1	0.8, 12.4
Trend	0.96	1.04	0.88, 1.22	0.90	1.01	0.79, 1.30	0.95	0.96	0.74, 1.25	1.19	1.51	0.99, 2.30
No. of cases/no. of controls¶		1,931/5,267			955/1,891			718/2,115			258/1,261	
Childhood period												
Benzene												
<3.8	1.0	1.0		1.0	1.0		1.0	1.0		1.0	1.0	
3.8–<10.4	0.9	0.9	0.8, 1.1	0.7	0.8	0.6, 1.0	1.1	1.2	0.9, 1.6	1.1	1.0	0.6, 1.7
10.4–<27.0	0.9	1.0	0.7, 1.3	0.8	0.9	0.6, 1.5	1.0	1.1	0.7, 1.8	1.1	0.8	0.4, 1.8
≥27.0	0.5	0.5	0.2, 1.0	0.3	0.4	0.1, 1.6	0.6	0.6	0.2, 1.7	0.6	0.4	0.1, 2.0
Trend	0.93	0.94	0.83, 1.05	0.90	0.96	0.81, 1.14	0.92	0.91	0.76, 1.10	1.06	0.96	0.71, 1.30
NO_2												
<11.5	1.0	1.0		1.0	1.0		1.0	1.0		1.0	1.0	
11.5–<29.4	1.0	1.1	0.9, 1.3	0.8	0.9	0.7, 1.3	1.0	1.1	0.8, 1.5	1.5	1.7	1.0, 3.0
29.4–<57.8	0.9	1.1	0.8, 1.5	1.0	1.3	0.8, 2.3	0.8	0.8	0.5, 1.5	1.5	1.8	0.7, 4.3
≥57.8	1.0	1.2	0.6, 2.3	0.3	0.4	0.1, 1.3	1.0	1.0	0.3, 3.1	3.7	4.7	1.2, 17.6
Trend	0.93	0.95	0.78, 1.16	0.91	1.00	0.75, 1.35	0.90	0.91	0.67, 1.23	1.11	1.04	0.62, 1.74
No. of cases/no. of controls		1,772/4,851			870/1,721			658/1,939			244/1,191	

* Adjusted for urban development, geographic region, type of residence, electromagnetic fields, mother's age, and birth order.

† We used the 50th, 90th, and 99th percentiles for the total study group as cutoff points, which for the pregnancy period corresponded to mean concentrations of 1.8, 4.6, and 12.9 ppb of benzene and 5.5, 11.0, and 19.4 ppb of NO_2. The ranges of mean concentrations were 0.6–29.9 ppb of benzene and 1.5–28.1 ppb of NO_2. Benzene: 1 ppb = 3.2 $\mu g/m^3$; NO_2: 1 ppb = 1.9 $\mu g/m^3$.

‡ Leukemias, central nervous system (CNS) tumors, and lymphomas considered together.

§ In the trend analyses, air pollution was entered as a continuous variable, disregarding the four exposure groups. The estimates show the relative risk corresponding to a doubling of the concentration of air pollution.

¶ Numbers of case and control children used in the adjusted analyses. The numbers are marginally lower than the numbers of available children, because of missing values in any of the included variables and because the analyses are based on at least one case and one control within each matched pair.

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TABLE 5. Crude and adjusted* relative risks (RRs) and 95% confidence intervals (CIs) for Hodgkin's disease associated with air pollution at the residence during pregnancy, Denmark, 1968–1991

Category of exposure (cutoff points in 1,000 ppb-days)	Cases (no.)	Controls (no.)	Crude		Adjusted	
			RR	95% CI	RR	95% CI
Benzene						
<0.5	34	194	1.0		1.0	
0.5–<1.3	30	160	1.1	0.6, 1.8	1.7	0.8, 3.8
≥1.3	19	53†	2.2	1.1, 4.3	4.3	1.5, 12.4
Trend‡	83	407†	1.31	1.01, 1.68	1.84	1.21, 2.80
NO_2						
1.5	40	217†	1.0		1.0	
1.5–<3.0	31	153	1.1	0.7, 2.0	1.5	0.7, 3.2
≥3.0	12	37	1.9	0.9, 4.0	6.7	1.7, 26.0
Trend‡	83	407†	1.30	0.91, 1.84	2.47	1.17, 5.25

* Adjusted for urban development, geographic region, type of residence, electromagnetic fields, mother's age, and birth order.

† Because of one control child with no information on mother's age and birth order, this number was reduced by one in the adjusted analyses.

‡ In the trend analyses, air pollution was entered as a continuous variable, disregarding the three exposure groups. The estimates show the relative risks corresponding to a doubling of the concentration of air pollution.

Validation of exposure assessment

Figure 1 (top) shows the distribution in the case-control study of the log-transformed calculated average concentrations of NO_2 at the front door during the pregnancy period. Figure 1 (bottom) refers to the validation study and shows the *calculated* half-year average concentrations of NO_2 at the front door plotted against the corresponding *measured* concentrations. For a given calculated value, the measured value has a 95 percent probability of being within the 95 percent prediction limits shown as dashed lines. The area between the prediction limits is divided into triangles (hatched) and hexagons (the remainder area) by the vertical and horizontal dotted lines, which indicate the cutoff points among the exposure groups. The area of the triangles, representing the misclassified observations, is smaller than that of the hexagons, representing the correctly classified observations. Table 6 shows the expected numbers of correctly classified and misclassified children in the case-control study under the assumption that the measured concentration at the front door is the exposure that is truly relevant to the hypothesis: 89 percent of the children categorized in the case-control study as having low exposure were expected to have been correctly categorized, 11 percent to be in the middle category, and none to be in the high or extreme category. Similarly, 83 percent, 80 percent, and 62 percent were expected to have been correctly classified in the middle, high, and extreme categories, respectively. Misclassification occurred only between adjacent categories.

Table 7 shows that there would be more misclassification in the case-control study if the measured personal exposure to NO_2 were assumed to be the truly relevant exposure, but misclassification was found mainly between adjacent categories.

DISCUSSION

We found no support for the hypothesis that traffic-related air pollution at the residence of children causes leukemias or central nervous system tumors, which are the two major diagnostic groups in children. The risk of lymphomas increased with increasing concentrations of ambient air pollution at the residence at the time the child was in utero, but the increase was restricted to Hodgkin's disease diagnosed before the age of 12 years.

The main finding of no effect conflicts with the results of previous studies. In a study in Denver, Colorado (2), traffic density at the residence at the time of diagnosis was used as a surrogate for exposure. Eight percent of cases and 25 percent of controls did not respond, leaving 328 cases and 262 controls for the analyses. When exposure to more than 500 vehicles per day was compared with less exposure, the odds ratios were 1.7 for all cancers combined, 2.1 for leukemia, 1.7 for central nervous system tumors, and 0.7 for lymphomas. In a contrast of exposure to more than 10,000 and fewer than 500 vehicles per day, the odds ratios were 3.1 for all cancers combined and 4.7 for leukemia. The results for leukemia, central nervous system tumors, and all cancers combined were above the upper confidence limits found in our study (table 3). A Swedish group (3) calculated the peak concentrations of NO_2 (99th percentiles of 1-hour means) at the residences of 142 case children and 550 control children selected from a cohort of children with potential exposure to an electromagnetic field; only the exposure to NO_2 at the latest address was considered. A comparison of children who lived at addresses with peak air pollution levels above the 75th percentile with those below the 50th percentile resulted in nonsignificantly elevated relative risks of 2.7, 2.7, and 5.1 for all cancers combined, leukemia, and central nervous system tumors. A study in West Midlands, United Kingdom (4), found an incidence ratio of 1.16 based on 24

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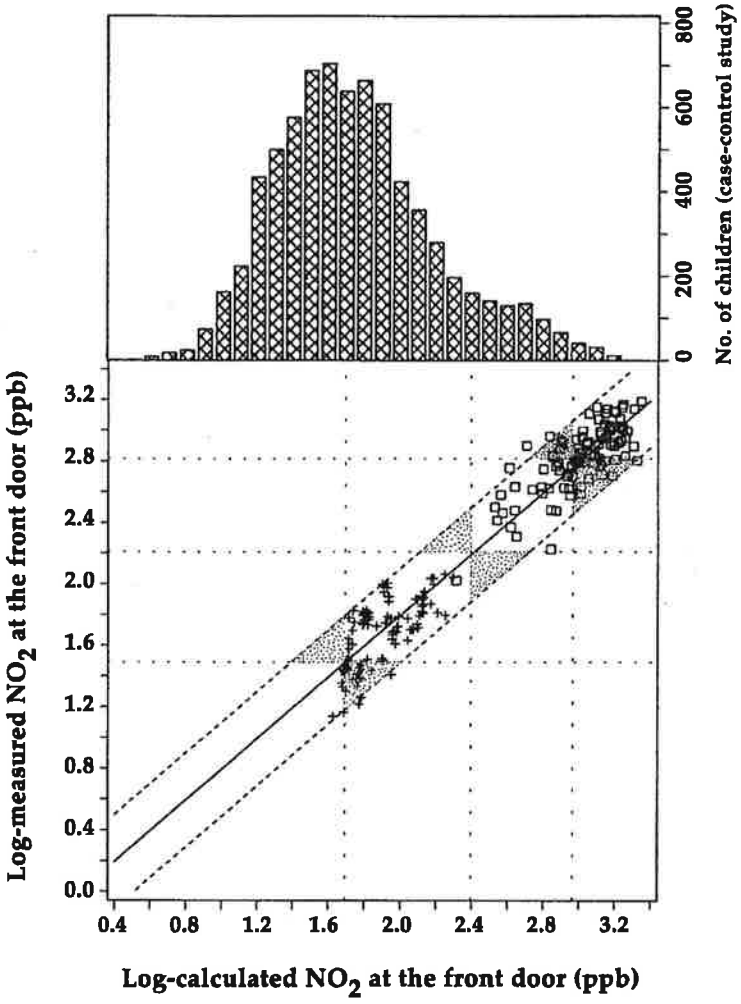

FIGURE 1. Distribution of exposure to NO_2 in the case-control study (top) and calculated half-year average concentrations at the front door plotted against measured concentrations in the validation study (bottom), Denmark, 1968–1991. Rural areas are indicated by pluses and Copenhagen by squares, and the estimated line with 95% prediction limits (dashed lines) is drawn. The vertical dotted lines refer to the cutoff points among the exposure groups in the case-control study, and the horizontal dotted lines refer to the same percentiles in the estimated distribution of measured NO_2 at the front door.

observed and 20.7 expected cases of leukemia among children living within 100 m of a main road. The exposure was assessed for the address at the time of the diagnosis. We designed our investigation to address the limitations of these studies, in particular the size of the study population, the selection procedures, and the exposure assessment.

TABLE 6. Expected misclassification among the four categories of exposure if calculated concentrations of NO_2 are used as the surrogate for measured concentrations at the front door, Denmark, 1968–1991

Distribution of calculated NO_2* in the case-control study	No.	%	Distribution of NO_2 measured at the front door†							
			Low‡		Middle		High		Extreme	
			No.	%	No.	%	No.	%	No.	%
Low‡	3,703	100	3,297	89	406	11	0	0	0	0
Middle	2,955	100	406	14	2,444	83	106	4	0	0
High	665	100	0	0	106	16	532	80	28	4
Extreme	74	100	0	0	0	0	28	38	46	62

* During the pregnancy period.
† Refers to the distribution estimated from the relation between calculated and measured concentrations (figure 1).
‡ Each of the four categories included the same number of children as the corresponding category in the case-control study.

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TABLE 7. Expected misclassification among the four categories of exposure if calculated concentrations of NO_2 at the front door are used as the surrogate for measured personal exposure, Denmark, 1968–1991

Distribution of calculated NO_2* in the case-control study	No.	%	Distribution of measured personal exposure to NO_2†							
			Low‡		Middle		High		Extreme	
			No.	%	No.	%	No.	%	No.	%
Low‡	3,703	100	2,741	74	933	25	29	1	0	0
Middle	2,955	100	941	32	1,717	58	289	10	8	0
High	665	100	21	3	294	44	304	46	46	7
Extreme	74	100	0	0	12	16	42	57	20	27

* During the pregnancy period.
† Refers to the distribution estimated from the relation between calculated and measured concentrations (figure 1).
‡ Each of the four categories included the same number of children as the corresponding category in the case-control study.

The selection of cancer cases and control children from reliable, population-based registries makes selection bias in this study unlikely. Differential misclassification of exposure is also unlikely, because the data were provided by persons who were unaware of the identity or health status of the children and because the exposure assessment was based on all of the addresses occupied by the children, avoiding bias due to differential mobility of cases and controls.

The degree of nondifferential misclassification was estimated from the results of a validation study. Marshall et al. (19) showed that nondifferential misclassification between adjacent categories of exposure, as expected in this study, can lead to risk estimates biased toward the neutral value; however, a true effect would be reduced only moderately by a 30 percent nondifferential misclassification of each of four exposure categories to each adjacent category. Moreover, the validation study showed that the misclassification or error (the difference between measured and calculated concentrations of NO_2) was almost independent of the calculated concentration, as might be expected when exposure is predicted from a model (20), and the error variance was constant. Thus, the data correspond to a "Berkson error model" (21); trend estimates derived from exposure data encumbered with a "Berkson error" and constant error variance can be expected to be unbiased (22, 23). The measured values of NO_2 in the validation study were approximately 0.81 times the calculated values. This has no implication for the trend or relative risk estimates, but the cutoff points between exposure groups (tables 4 and 5) should be multiplied by 0.81 to correspond to concentrations measured by the methods used in the validation study.

The validation study showed that greater misclassification of exposure occurred when measured personal exposure to NO_2, rather than measured concentration at the front door, was considered to be the truly relevant exposure. This difference is due mainly to individual time-activity patterns combined with contributions to personal exposure from indoor sources and children's exposure to ambient air pollution at locations other than the residence. The relevance of the two types of measurement depends on the question to be answered. The major concern of families with small children, in relation to the hypothesis of this study, is whether their children are at risk of cancer because they live on a street with dense traffic. In this case, the measured concentration at the front door is the relevant choice, and the lower degree of misclassification would be relevant. Moreover, concentrations of NO_2 at the front door reflect the concentrations of other traffic-related air pollutants as well. The higher degree of misclassification when personal measurements are considered to be the truly relevant exposure would pertain if the results of this study were used to evaluate whether exposure to NO_2 (regardless of the source) causes cancer in children. As different air pollutants have different indoor sources, personal exposure to NO_2 cannot be considered to indicate personal exposure to other (traffic-related) air pollutants by default. If a (group of) pollutant(s) present in traffic exhaust causes childhood cancer but traffic exhaust was not the main source of the exposure of children to this pollutant, it would be difficult to detect the effect by the methods used in this study. In that situation, however, the effect of overlooking a risk due partly to traffic would be limited exactly because traffic contributed only little to the exposure; in contrast, if traffic made a large contribution to the exposure of children to a relevant pollutant, one would have a better chance of detecting the effect by using the concentration at the front door as the exposure surrogate.

We have hitherto focused on the validity of calculated concentrations of NO_2, but the levels of benzene were also addressed in the validation study, although fewer and only 1-week measurements were included. Despite a less precise measurement method for benzene (24), simultaneous 1-week measurements of benzene and NO_2 at the same 76 locations in Copenhagen showed that calculated concentrations were slightly more correlated with measured concentrations for benzene than for NO_2 (11), indicating a similar or better performance of the Operational Street Pollution Model when predicting concentrations of benzene in streets. Sources other than traffic at the residence may contribute significantly to children's personal exposure to benzene (24, 25), and use of the residential front-door concentration as a surrogate for personal exposure to benzene would probably imply a high degree of misclassification.

Altogether, the validation study indicated a low degree of misclassification of traffic-related air pollutants and a Berkson type of error if calculated levels are used as surrogate for measured levels outside the residence. However,

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because of different sources (other than traffic) of the personal exposure to different pollutants present in traffic exhaust, the results for misclassification of personal exposure to NO_2 cannot be generalized, and the degree of misclassification of the personal exposure to other pollutants is unpredictable without validation for each pollutant.

Few risk factors have been consistently linked to the risks of leukemia, central nervous system tumors, and lymphomas in children; those that have are inherited genetic alterations, ionizing radiation, Epstein-Barr virus infection, age, and sex (26). In our study, age, sex, and calendar period were excluded as confounders by matching, and it is difficult to imagine that inherited genetic alterations or exposure to diagnostic or therapeutic x-rays is associated with air pollution. Gamma-radiation from natural sources and indoor concentrations of radon vary by geographic region and type of house, but these were adjusted for in the analyses. Epstein-Barr virus is a cause of Burkitt's lymphoma in children, and these tumors constituted 20 percent of the non-Hodgkin's lymphomas. If exposure to Epstein-Barr virus is associated with air pollution at a residence, the most likely link would be urban development (many contacts) and not the number of vehicles passing by the address; urban development was adjusted for in the analyses. Since the degree of urban development and the concentration of ambient air pollution correlate, adjustment for urban development in the analyses might unduly remove a true effect of air pollution; however, the results of the unadjusted analyses excluded that possibility. The proposed risk factors that were not adjusted for in this study include medication, maternal smoking, parental occupation, pets, and immunizations. However, the evidence that these factors are indeed risk factors of childhood cancer is conflicting, and any increase in risk would be weak; moreover, strong correlations between these factors and traffic-related air pollution at residences are unlikely.

Given the low potential for selection bias, information bias, and confounding from known or proposed risk factors not adjusted for in the analyses and given the expected degree of nondifferential misclassification, the most reasonable interpretation of the results of our study is that no major risk of leukemia, central nervous system tumors, or all of the selected cancers combined is associated with traffic-related air pollution at the residence at the levels found in Denmark. The concentrations of benzene estimated in this study were similar to those found outdoors in the United States (25). We are confident in this interpretation of no major risk as regards traffic-related air pollution at the residence, whereas an interpretation of no effect of personal exposure is problematic.

A causal interpretation of the association between the risk of Hodgkin's disease and exposure in utero is supported by the strength of the association, the exposure-response pattern, and previous findings of different risk factors of Hodgkin's disease in young and older children (27–30). Moreover, immunologic disorders may be involved in the etiology of Hodgkin's disease (31, 32), and a number of studies indicate that exposure to ambient air pollution affects the human immune system (33–36). Arguments against a causal interpretation are the absence of a known, plausible, specific biologic mechanism and the unexpected nature of the finding. Moreover, the finding was of only borderline significance in univariate models and may be a chance finding due to multiple testing.

In summary, traffic-related air pollution at the residence does not appear to cause leukemias, central nervous system tumors, or non-Hodgkin's lymphomas in children. The finding of a higher risk of Hodgkin's disease among children whose mothers had lived at residences with high outdoor levels of air pollution from traffic during the pregnancy might reflect a causal relation, although the evidence allows no firm conclusion at present.

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Traffic Air Pollution and Mortality Rate Advancement Periods

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Chronic exposure to air pollution is associated with increased mortality rates. The impact of air pollution relative to other causes of death in a population is of public health importance and has not been well established. In this study, the rate advancement periods associated with traffic pollution exposures were estimated. Study subjects underwent pulmonary function testing at a clinic in Hamilton, Ontario, Canada, between 1985 and 1999. Cox regression was used to model mortality from all natural causes during 1992–2001 in relation to lung function, body mass index, a diagnosis of chronic pulmonary disease, chronic ischemic heart disease, or diabetes mellitus, household income, and residence within 50 m of a major urban road or within 100 m of a highway. Subjects living close to a major road had an increased risk of mortality (relative risk = 1.18, 95% confidence interval: 1.02, 1.38). The mortality rate advancement period associated with residence near a major road was 2.5 years (95% confidence interval: 0.2, 4.8). By comparison, the rate advancement periods attributable to chronic pulmonary disease, chronic ischemic heart disease, and diabetes were 3.4 years, 3.1 years, and 4.4 years, respectively.

air pollution; automobiles; mortality; urbanization; vehicles

Abbreviations: CI, confidence interval; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; ICD-9, *International Classification of Diseases*, Ninth Revision.

There is increasing evidence that long-term exposure to the particulate and gaseous air pollution common to many metropolitan areas is an important risk factor for mortality. The strongest evidence comes from an updated study of mortality in approximately 550,000 adults who were enrolled in the American Cancer Society's Cancer Prevention Study II. During 16 years of follow-up, Pope et al. (1) found that fine particulate- and sulfur oxide-related pollution was associated with all-cause, lung cancer, and cardiopulmonary mortality. In a Dutch study, Hoek et al. (2) reported that proximity to major roads produced a larger association with cardiopulmonary mortality than did urban background concentrations of air pollution.

Although these studies and the earlier Six Cities Study (3) demonstrated a relation between air pollution and mortality rates, the impact of air pollution relative to other causes of death in a population has not been well established. This relation is of public health importance. Pope et al. (1) compared the mortality effect of fine particulate air pollution with that of cigarette smoking and obesity. They concluded that the risk associated with exposure to particulate air pollution was much smaller than the risk of cigarette smoking but was comparable to the estimated effect of being moderately overweight.

One measure of the impact of a risk factor on mortality is the "rate advancement period" developed by Brenner et al. (4). The rate advancement period estimates the impact of a risk factor on the *timing* of death; that is, the risk is phrased in terms of a premature mortality rate among exposed persons. In this study, we estimated the rate advancement period associated with intraurban traffic pollution exposures in a cohort of subjects in the city of Hamilton, Ontario, Canada.

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MATERIALS AND METHODS

Study subjects and mortality follow-up

The study subjects were residents of the Hamilton metropolitan area who underwent pulmonary function testing between 1985 and 1999 at a Hamilton clinic. These included persons referred for specialist assessment and persons referred for spirometric testing only. Information available included the subject's Ontario Health Insurance Number and postal code, the subject's body mass index (weight (kg)/height $(m)^2$), and the results of pulmonary function tests, including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1). Predicted FVC and FEV_1 were computed using the equations of Hankinson et al. (5). Smoking histories were not recorded.

The province of Ontario provides universal health insurance for all residents. A family-based Medicare number was replaced with a personal Health Insurance Number in 1992. This Health Insurance Number was used to link subjects to Ontario Health Insurance Plan physician billing files for 1992–1999.

Subjects entered mortality follow-up on January 1, 1992, or the date of their first clinic visit, whichever was later. To ensure that subjects without a visit after January 1, 1992, were still alive at the start of follow-up, we excluded subjects without an Ontario Health Insurance Plan transaction after 1991. Mortality was ascertained by linkage to the Ontario Mortality Registry for the period 1992–2001. Because there were only 27 deaths among younger subjects, this study was restricted to subjects aged 40 years or older. Subjects diagnosed with cancer, either before or within 45 days of the initial visit, were excluded. This left 5,228 subjects (950 total deaths, 923 nonaccidental) available for analysis. This study was approved by the Research Ethics Board at St. Joseph's Healthcare, Hamilton.

Underlying medical diagnoses

We used the Ontario Health Insurance Plan linkage to classify underlying respiratory disease status, utilizing diagnostic codes submitted to the Ontario Health Insurance Plan by specialist physicians. Diagnoses included chronic obstructive pulmonary disease (*International Classification of Diseases*, Ninth Revision (ICD-9), codes 491, 492, and 496: 2,035 subjects, 431 deaths), asthma (ICD-9 code 493: 604 subjects, 28 deaths), bronchiectasis (ICD-9 code 494: 141 subjects, 31 deaths), asbestosis (ICD-9 code 501: 39 subjects, four deaths), silicosis (ICD-9 code 502: 24 subjects, three deaths), and postinflammatory pulmonary fibrosis (ICD-9 code 515: 240 subjects, 75 deaths). Subjects with diagnoses of both asthma and chronic obstructive pulmonary disease were classified as having chronic obstructive pulmonary disease.

The Ontario Health Insurance Plan billing file and the Ontario hospital discharge database were also searched for diagnoses of diabetes mellitus (ICD-9 code 250) and chronic ischemic heart disease (ICD-9 codes 412–414). Subjects were classified as having these disorders if the diagnosis had been made in two or more claims submissions by a general practitioner, in one claim submission by a specialist, or in any hospitalization. This algorithm has been validated for diagnosis of diabetes in Ontario (6).

Income and road traffic indicators

Postal codes were used to estimate household income on the scale of the enumeration area, the smallest unit in the Canadian census. They were also used to assign residential addresses to road traffic buffers. Canadian six-character postal codes are localized to small areas in urban regions, and the domain included may be as small as a single apartment building or city block. The ArcView 3.2 geographic information system (ESRI, Inc., Redlands, California) was used with the Statistics Canada 1999 postal code conversion file and the Census of 1996 to estimate mean household income in the subjects' neighborhoods by overlay of enumeration area data. The geographic information system was also used to map subjects' residences with respect to major sources of road traffic pollution. We used the same indicator of traffic-related pollution as Hoek et al. (2). Subjects' postal codes were mapped with respect to buffers of 50 m around major urban roads and 100 m from highways (road/highway buffers).

Modeling of mortality relative risk

The Cox proportional hazards model (7) was used to model the risk of mortality from all natural causes. We tested for deviations from the proportional hazards assumption by introducing interactions of time with all covariates. To account for possible geographic "clustering" of mortality, we grouped subjects among census tracts (n = 120 tracts; mean number of subjects per census tract = 59 (range, 1–224)), and we adjusted estimates of variance for clustering in census tracts. Calculations were performed with Stata statistical software (8).

Rate advancement periods

The effect of traffic-related pollution with respect to the time dimension of premature mortality was assessed using Brenner et al.'s (4) "rate advancement period," which estimates how much older a reference population would have to be to experience the same "attrition rate" as subjects residing within a road buffer. The rate advancement period is derived from a linear model of the form $\log(\text{hazard}) = b_1 \times \text{exposure} + b_2 \times \text{age} + \text{covariates}$. From this model, a point estimate of the rate advancement period is b_1/b_2 per unit increase in exposure. A formula for the variance is given in the paper by Brenner et al. (4).

Additional deaths per year

Rates of mortality from all natural causes are dependent on age. To compute the "additional deaths per year" attributable to traffic pollutant exposure, we added the rate advancement period to chronologic age and performed interpolation in Ontario life tables. We subtracted the mortality rate at a given reference age from the rate at the "rate-advanced" age to obtain the number of additional all-natural-cause deaths

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TABLE 1. Characteristics of subjects exposed to traffic air pollution and control subjects in relation to distance of residence from major roads and highways, Hamilton, Ontario, Canada, 1992–2001

Variable	Control subjects (>100 m from a highway *and* >50 m from a major road) (n = 4,280)				Exposed subjects (within a road/highway buffer (<50 m from a major road or <100 m from a highway)) (n = 948)			
	No.	%	Median	IQR*	No.	%	Median	IQR
Male gender	1,905	45			401	42		
Age (years)			60.4	49.8–70.0			63.6	51.7–72.4
Annual household income (thousands of Canadian dollars)			48.3	36.5–61.1			39.1	30.7–52.2
Forced vital capacity (% predicted)			84	70–97			81	67–94
Forced expiratory volume in 1 second (% predicted)			79	55–96			73	52–91
Body mass index†			27.8	24.4–31.6			27.6	24.4–31.6
Diagnosed with asthma	471	11			85	9		
Diagnosed with chronic pulmonary disease (except asthma)	1,753	41			432	46		
Diagnosed with chronic ischemic heart disease	1,372	32			318	34		
Diagnosed with diabetes mellitus	695	16			145	15		
Nonaccidental death	706				217			

* IQR, interquartile range (25th–75th percentile).
† Weight (kg)/height (m)^2.

per 1,000 subjects per year, adjusted for clinical and disease variables.

RESULTS

Study subjects and pollutants

The distribution of subjects by road buffer residence classification is shown in table 1, in conjunction with mean values for clinical variables. Approximately half of the subjects in this cohort of patients from a lung function laboratory did not have a chronic respiratory disease diagnosed by a specialist.

Mortality in relation to clinical variables, disease status, household income, and residence near major roads

Table 2 shows the results from Cox models for all-natural-cause mortality in relation to physiologic and disease variables, income, and residence within a traffic pollution buffer. To explore risk modification by respiratory disease status, we conducted analyses separately for subjects with and without diagnoses of chronic pulmonary disease. The proportional hazards hypothesis was not rejected for any of the models.

As has been observed in other studies, mortality rates were lower among subjects with higher FVC and FEV_1 (percent predicted) (9); there was a quadratic relation between mortality rates and body mass index (10); and mortality rates were lower in households with higher incomes (11). Subjects residing within traffic pollution buffers had elevated mortality rates regardless of whether they had been diagnosed with chronic pulmonary disease (excluding asthma). A test for effect modification by chronic pulmonary disease did not give a significant result ($p = 0.77$). The final column of table 2 shows that after adjustment for clinical variables and chronic disease diagnoses, the relative risk for all-natural-cause mortality among subjects residing within a road buffer was 1.18 (95 percent confidence interval (CI): 1.02, 1.38).

Rate advancement periods

We used the formulae in the paper by Brenner et al. (4) to compute the rate advancement periods and their variances. The formula for the rate advancement period requires a correct statement of the dependency between log(hazard) and age. The hypothesis of linearity between log(hazard) and age was not rejected after examination of the martingale residuals (7). Table 3 shows the mortality rate advancement period attributable to residence within a traffic pollution buffer. Rate advancement periods attributable to diagnoses of chronic disease are presented for comparison.

The rate advancement period associated with traffic pollution was 2.5 years (95 percent CI: 0.2, 4.8). By way of comparison, the rate advancement periods attributable to chronic pulmonary diseases other than asthma, to chronic ischemic heart disease, and to diabetes were 3.4 years, 3.1 years, and 4.4 years, respectively. Thus, the "aging effect"

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TABLE 2. Relative risk of mortality from all natural causes (n = 923) during follow-up in relation to clinical measures, chronic disease diagnoses, household income, and pollutant exposures, Hamilton, Ontario, Canada, 1992–2001

Variable	Diagnosed with CPD*		Not diagnosed with CPD		All subjects	
	RR*	95% CI*	RR	95% CI	RR	95% CI
Female gender	0.62	0.52, 0.74	0.28	0.20, 0.41	0.52	0.44, 0.61
Age (per year above 40)	1.06	1.05, 1.08	1.07	1.06, 1.08	1.07	1.06, 1.08
Forced vital capacity (% predicted—per 1%)	0.987	0.980, 0.993	0.991	0.981, 1.001	0.986	0.980, 0.992
Forced expiratory volume in 1 second (% predicted—per 1%)	0.993	0.987, 0.999	0.985	0.977, 0.995	0.993	0.988, 0.998
Body mass index† (per unit)	0.93	0.91, 0.95	0.95	0.92, 0.99	0.94	0.92, 0.95
Body mass index squared	1.003	1.001, 1.004	1.001	0.999, 1.003	1.002	1.001, 1.003
CPD					1.26	1.05, 1.49
Asthma			0.68	0.48, 0.96	0.67	0.48, 0.94
Chronic ischemic heart disease	1.12	0.97, 1.30	1.55	1.20, 2.01	1.23	1.06, 1.42
Diabetes mellitus	1.36	1.13, 1.62	1.32	0.97, 1.78	1.34	1.13, 1.59
Log(household income)	0.88	0.72, 1.07	0.74	0.48, 1.14	0.84	0.69, 1.03
Residence within a road/highway buffer	1.18	0.98, 1.41	1.27	0.92, 1.75	1.18	1.02, 1.38

* CPD, chronic pulmonary disease (excluding asthma); RR, relative risk; CI, confidence interval.
† Weight (kg)/height $(m)^2$.

attributable to traffic pollution was only slightly lower than the aging effect associated with these chronic diseases.

By interpolation in Ontario life tables, we estimated the number of deaths attributable to an "aging effect" of 2.5 years. The estimates were 0.4 additional deaths/1,000/year among subjects aged 40 years, 1.6 deaths/1,000/year among subjects aged 50 years, 4.4 deaths/1,000/year among subjects aged 60 years, and 10.9 deaths/1,000/year among subjects aged 70 years.

DISCUSSION

We have confirmed Hoek et al.'s observation (2) that rates of mortality from all natural causes vary in association with residential proximity to traffic pollution. The importance of pollution with respect to mortality and other health outcomes in a population is meaningful for cost-benefit analyses of interventions designed to lower pollution exposures. There is a consensus that these assessments should not rely on the results of time-series studies but rather should be based on long-term follow-up in cohort studies (12, 13). Our cohort comprised subjects who underwent lung function testing. We found that there was no risk modification by respiratory disease and that the relative risk associated with residential proximity to traffic was the same for subjects with and without physician-diagnosed chronic respiratory disease.

TABLE 3. Rate advancement period for mortality from all natural causes in relation to residence close to a major road and common chronic diseases, Hamilton, Ontario, Canada, 1992–2001

Risk factor	RAP*,† (years)	95% CI*
Residence within a road/highway buffer	2.5	0.2, 4.8
Diagnosis of chronic pulmonary disease (excluding asthma)	3.4	0.8, 6.0
Diagnosis of chronic ischemic heart disease	3.1	0.8, 5.4
Diagnosis of diabetes mellitus	4.4	1.8, 7.0

* RAP, rate advancement period; CI, confidence interval.
† The rate advancement period is the number of years older the comparison subjects would have to be in order to have the same attrition rates as subjects with the indicated risk factor exposure.

Use of the rate advancement period provides one view of the impact of increased mortality rates. Since interpretation of the rate advancement period is conditional on the absence of competing causes of death (4), we restricted our analysis to mortality from all natural causes and did not consider specific underlying causes, such as cardiopulmonary diseases, which are most causally linked to air pollutant exposures (1). The use of all-natural-cause mortality provides a global assessment of mortality impact. We found that the "aging effect" attributable to traffic pollution exposure was 2.5 years, which is similar to the rate advancement period attributable to common chronic diseases. We emphasize that the rate advancement period does not equate directly with "years of life lost" but rather indicates the advancement of mortality *rates*. The extent of life shortening will depend on the ages of the subjects.

A weakness of our study is that we have, as yet, no measurements of pollutant levels within the road buffers. In North American cities, pollution is generated by gasoline-powered cars and diesel-fueled buses and trucks. The

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burning of fossil fuels creates primary pollutants, including carbon dioxide, carbon monoxide, oxides of nitrogen, sulfur dioxide, hydrocarbons, and airborne particulates. It has been estimated that particulate emission from diesel engines, per distance traveled, is over 100 times higher than that from gasoline engines fitted with catalytic converters (14). Dutch investigators (15) reported that daytime concentrations of particulate matter less than 10 μm in aerodynamic diameter were, on average, 30 percent higher near busy roads than at background locations, differing by 7–13 $\mu g/m^3$. Researchers in another Dutch study reported that indoor and outdoor particulate concentrations were higher at homes located on high-traffic-intensity streets than at homes on low-traffic streets (16). The mean indoor concentration of particulate matter less than 2.5 μm in aerodynamic diameter was 27 $\mu g/m^3$ in high-traffic homes and 12 $\mu g/m^3$ in low-traffic homes. In Roxbury, Massachusetts (17), concentrations of fine particulate matter and particle-bound polycyclic aromatic hydrocarbons were higher on roads reported to have heavy bus traffic. In the South Bronx (New York City), sidewalk measures of diesel exhaust particulates were correlated with the count of large trucks (18).

As in the other studies of air pollution cohorts, we did not have a measure of residential mobility during the follow-up period, so misclassification of traffic exposure was possible. Similar to several of the other air pollution cohorts, our cohort was not a random sample of the population. The cohort in the Six Cities Study (3) was a random sample of 8,111 White adults. Participants in the American Cancer Society study (1) were friends, neighbors, or acquaintances of American Cancer Society volunteers. In the Dutch study by Hoek et al. (2), only 36 percent of potential subjects completed the baseline questionnaire.

Our subjects underwent lung function testing at an academic clinic. Although only half of the subjects were diagnosed with respiratory disease, they presumably had respiratory symptoms such as cough and shortness of breath prior to referral. Although our cohort contained a greater proportion of persons with respiratory illness than is found in the general population, we believe that our results can be generalized because of the availability of clinical diagnoses. To our knowledge, this was the first cohort study with access to physicians' diagnoses of underlying disease status. Thus, we were able to adjust for diagnoses of chronic respiratory and pulmonary diseases and diabetes. We found no effect modification by underlying respiratory disease status; and our adjusted estimate of the all-natural-cause mortality risk associated with residential exposure to traffic was 1.18 (95 percent CI: 1.02, 1.38), similar to the Dutch estimate of 1.35 (95 percent CI: 0.93, 1.95) (2).

In conclusion, we found an association between residential proximity to traffic and mortality. The mortality rate advancement attributable to traffic pollution was similar to that associated with chronic respiratory and pulmonary diseases and diabetes. This suggests that decreasing pollutant exposures may have a substantial public health impact.

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Health Effects of Diesel Exhaust

A fact sheet by
Cal/EPA's Office of Environmental Health Hazard Assessment and
The American Lung Association of California.

AMERICAN LUNG ASSOCIATION. of California

Diesel fuel is widely used throughout our society. It powers trucks that deliver products to our communities, buses that carry us to school and work, agricultural equipment that plants and harvests our food, and backup generators that can provide electricity during emergencies. It is also used for many other applications. Diesel engines have historically been more versatile and cheaper to run than gasoline engines or other sources of power. Unfortunately, the exhaust from these engines contains substances that can pose a risk to human health.

In 1998, the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) completed a comprehensive health assessment of diesel exhaust. This assessment formed the basis for a decision by the California Air Resources Board (ARB) to formally identify particles in diesel exhaust as a toxic air contaminant that may pose a threat to human health. The American Lung Association of California (ALAC) and its 15 local associations work to prevent lung disease and promote lung health. Since 1904, the American Lung Association has been fighting lung disease through education, community service, advocacy and research.

This fact sheet by OEHHA and ALAC provides information on health hazards associated with diesel exhaust.

Diesel exhaust contains more than 40 toxic air contaminants

What is diesel exhaust?

Diesel exhaust is produced when an engine burns diesel fuel. It is a complex mixture of thousands of gases and fine particles (commonly known as soot) that contains more than 40 toxic air contaminants. These include many known or suspected cancer-causing substances, such as benzene, arsenic and formaldehyde. It also contains other harmful pollutants, including nitrogen oxides (a component of urban smog).

How are people exposed to diesel exhaust?

Diesel exhaust particles and gases are suspended in the air, so exposure to this pollutant occurs whenever a person breathes air that contains these substances. The prevalence of diesel-powered engines makes it almost impossible to avoid exposure to diesel exhaust or its byproducts, regardless of whether you live in a rural or urban setting. However, people living and working in urban and industrial areas are more likely to be exposed to this pollutant. Those spending time on or near roads and freeways, truck loading and unloading operations, operating diesel-powered machinery or

working near diesel equipment face exposure to higher levels of diesel exhaust and face higher health risks.

What are the health effects of diesel exhaust?

As we breathe, the toxic gases and small particles of diesel exhaust are drawn into the lungs. The microscopic particles in diesel exhaust are less than one-fifth the thickness of a human hair and are small enough to penetrate deep into the lungs, where they contribute to a range of health problems.

Diesel exhaust and many individual substances contained in it (including arsenic, benzene, formaldehyde and nickel) have the potential to contribute to mutations in cells that can lead to cancer. In fact, long-term exposure to diesel exhaust particles poses the highest cancer risk of any toxic air contaminant evaluated by OEHHA. ARB estimates that about 70 percent of the cancer risk that the average Californian faces from breathing toxic air pollutants stems from diesel exhaust particles.

Diesel exhaust increases the risk of cancer…

In its comprehensive assessment of diesel exhaust, OEHHA analyzed more than 30 studies of people who worked around diesel equipment, including truck drivers, railroad workers and equipment operators. The studies showed these workers were more likely to develop lung cancer than workers who were not exposed to diesel emissions. These studies provide strong evidence that long-term occupational exposure to diesel exhaust increases the risk of lung cancer. Using information from OEHHA's assessment, ARB estimates that diesel-particle levels measured in California's air in 2000 could cause 540 "excess" cancers (beyond what would occur if there were no diesel particles in the air) in a population of 1 million people over a 70-year lifetime. Other researchers and scientific organizations, including the National Institute for Occupational Safety and Health, have calculated cancer risks from diesel exhaust that are similar to those developed by OEHHA and ARB.

Exposure to diesel exhaust can have immediate health effects. Diesel exhaust can irritate the eyes, nose, throat and lungs, and it can cause coughs, headaches, light-headedness and nausea. In studies with human volunteers, diesel exhaust particles made people with allergies more susceptible to the materials to which they are allergic, such as dust and pollen. Exposure to diesel exhaust also causes inflammation in the lungs, which may aggravate chronic respiratory symptoms and increase the frequency or intensity of asthma attacks.

… And it can cause coughs and aggravate asthma

Diesel engines are a major source of fine-particle pollution. The elderly and people with emphysema, asthma, and chronic heart and lung disease are especially sensitive to fine-particle pollution. Numerous studies have linked elevated particle levels in the air to increased hospital admissions, emergency room visits, asthma attacks and premature deaths among those suffering from respiratory problems. Because children's lungs and respiratory systems are still developing, they are also more susceptible than healthy adults to fine particles. Exposure to fine particles is associated with increased frequency of childhood illnesses and can also reduce lung function in children.

Like all fuel-burning equipment, diesel engines produce nitrogen oxides, a common air pollutant in California. Nitrogen oxides can damage lung tissue, lower the body's resistance to respiratory infection and worsen chronic lung diseases, such as asthma. They also react with other pollutants in the atmosphere to form ozone, a major component of smog.

What is being done to reduce the health risks from diesel exhaust?

Improvements to diesel fuel and diesel engines have already reduced emissions of some of the pollutants associated with diesel exhaust. However, diesel exhaust is still one of the most widespread and toxic substances in California's air.

ARB's Diesel Risk Reduction Plan, when fully implemented, will result in a 75 percent reduction in particle emissions from diesel equipment by 2010 (compared to 2000 levels), and an 85 percent reduction by 2020. The plan calls for the use of cleaner-burning diesel fuel, retrofitting of existing engines with particle-trapping filters, and the use in new diesel engines of advanced technologies that produce nearly 90 percent fewer particle emissions, as well as the use of alternative fuels.

Diesel exhaust contributes to smog and fine-particle pollution

The use of other fuels, such as natural gas, propane and electricity offer alternatives to diesel fuel. All of them produce fewer polluting emissions than current formulations of diesel fuel. As a result of ARB and local air-quality regulations, public transit agencies throughout California are using increasing numbers of passenger buses that operate with alternative fuels or retrofitted equipment.

For further information

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The energy challenge facing California is real. Every Californian needs to take immediate action to reduce energy consumption. For a list of simple ways you can reduce demand and cut your energy costs, see OEHHA's web site at www.oehha.ca.gov/public_info.html.

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ARTICLE

The Diesel Exhaust in Miners Study: A Nested Case–Control Study of Lung Cancer and Diesel Exhaust

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Background Most studies of the association between diesel exhaust exposure and lung cancer suggest a modest, but consistent, increased risk. However, to our knowledge, no study to date has had quantitative data on historical diesel exposure coupled with adequate sample size to evaluate the exposure–response relationship between diesel exhaust and lung cancer. Our purpose was to evaluate the relationship between quantitative estimates of exposure to diesel exhaust and lung cancer mortality after adjustment for smoking and other potential confounders.

Methods We conducted a nested case–control study in a cohort of 12315 workers in eight non-metal mining facilities, which included 198 lung cancer deaths and 562 incidence density–sampled control subjects. For each case subject, we selected up to four control subjects, individually matched on mining facility, sex, race/ethnicity, and birth year (within 5 years), from all workers who were alive before the day the case subject died. We estimated diesel exhaust exposure, represented by respirable elemental carbon (REC), by job and year, for each subject, based on an extensive retrospective exposure assessment at each mining facility. We conducted both categorical and continuous regression analyses adjusted for cigarette smoking and other potential confounding variables (eg, history of employment in high-risk occupations for lung cancer and a history of respiratory disease) to estimate odds ratios (ORs) and 95% confidence intervals (CIs). Analyses were both unlagged and lagged to exclude recent exposure such as that occurring in the 15 years directly before the date of death (case subjects)/reference date (control subjects). All statistical tests were two-sided.

Results We observed statistically significant increasing trends in lung cancer risk with increasing cumulative REC and average REC intensity. Cumulative REC, lagged 15 years, yielded a statistically significant positive gradient in lung cancer risk overall (P_{trend} = .001); among heavily exposed workers (ie, above the median of the top quartile [REC ≥ 1005 μg/m³-y]), risk was approximately three times greater (OR = 3.20, 95% CI = 1.33 to 7.69) than that among workers in the lowest quartile of exposure. Among never smokers, odd ratios were 1.0, 1.47 (95% CI = 0.29 to 7.50), and 7.30 (95% CI = 1.46 to 36.57) for workers with 15-year lagged cumulative REC tertiles of less than 8, 8 to less than 304, and 304 μg/m³-y or more, respectively. We also observed an interaction between smoking and 15-year lagged cumulative REC ($P_{interaction}$ = .086) such that the effect of each of these exposures was attenuated in the presence of high levels of the other.

Conclusion Our findings provide further evidence that diesel exhaust exposure may cause lung cancer in humans and may represent a potential public health burden.

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The question of whether diesel exhaust causes lung cancer in humans has been investigated in many studies since the 1980s. In 1989, the International Agency for Research on Cancer (IARC) classified diesel exhaust as a "probable" carcinogen (IARC classification: Group 2A) based on "sufficient" experimental evidence and "limited" evidence of carcinogenicity in humans (1). Two meta-analyses (2,3) of epidemiological studies have estimated the summary relative risk for lung cancer for those ever occupationally exposed to diesel exhaust as 1.33 (95% confidence interval [CI] = 1.24 to 1.44) (2) and 1.47 (95% CI = 1.29 to 1.67) (3), based on more than 35 studies. A pooled analysis (4) of 13 304 case subjects and 16 282 control subjects from 11 lung cancer case–control studies in Europe and Canada yielded an odds ratio (OR) of 1.31 (95% CI = 1.19 to 1.43) for subjects in the highest vs lowest quartile of cumulative diesel exposure based on a job exposure matrix (4). Although these meta-analyses (2,3) and the pooled analysis (4) suggest a modest but consistent effect, the excesses are in a range that could be explained by confounding (5), particularly from

CONTEXTS AND CAVEATS

Prior knowledge

Most previous studies have found a modest association between the risk of lung cancer and exposure to diesel exhaust (DE). However, these studies typically have inferred DE exposure from job title in the absence of quantitative data on historical DE exposures.

Study design

A nested case–control study of lung cancer and DE in a cohort of 12315 workers in eight non-metal mining facilities included 198 lung cancer deaths and 562 control subjects. The case–control study evaluated the exposure–response relationship between DE and lung cancer mortality after adjustment for cigarette smoking and other potential confounding factors that were unavailable in the cohort study.

Contribution

The results showed a strong and consistent relationship between quantitative exposure to DE and increased risk of dying from lung cancer. Among heavily exposed workers, the risk of dying from lung cancer was approximately three times greater than that among workers in the lowest quartile of exposure.

Implication

Exposure to DE may cause lung cancer in mine workers.

Limitations

Data on smoking and other potential confounders were derived mainly from next-of-kin interviews. Retrospective assessment of DE exposure may result in some misclassification, leading to imprecision in exposure estimates.

From the Editors

smoking. Alternatively, these excesses may be underestimates of risk due to inadequate latent period for the development of lung cancer in some studies or misclassification of exposure because most epidemiological studies inferred diesel exhaust exposure from job title in the absence of any additional information on level of diesel exposure. In-depth studies of truck drivers (6,7) and railroad workers (8), two occupational groups with light to moderate exposure to diesel exhaust, have found nearly a doubling of lung cancer risk among long-term workers. Retrospective exposure assessments in these studies, however, were hampered by limited historical industrial hygiene measurements. In fact, few studies have based estimates of lung cancer risk on quantitative estimates of exposure to diesel exhaust (8–11). Only one study of German potash miners reported results based on quantitative estimates of historical exposures that included industrial hygiene measurements but was based on only 61 lung cancer deaths (11). To our knowledge, no study to date has had quantitative data on historical diesel exposure coupled with adequate sample size to evaluate the exposure–response relationship for diesel exhaust and lung cancer with adjustment for potential confounding from cigarette smoking and other risk factors for lung cancer.

We conducted a cohort mortality study among workers employed at eight underground non-metal mining facilities (12) and a companion case–control study of lung cancer nested in this cohort to evaluate the risk of lung cancer from exposure to diesel exhaust (The Diesel Exhaust in Miners Study [DEMS]). The purpose of the case–control study reported in this article was to further evaluate the exposure–response relationship between diesel exhaust and lung cancer mortality after adjustment for cigarette smoking and other potential confounding factors that were unavailable in the cohort study.

Materials and Methods

Cohort Design and Follow Up

Eight non-metal mining facilities (three potash, three trona, one limestone, and one salt [halite]) were selected from all US non-metal mining facilities with at least 50 employees who were considered to have had high air levels of diesel exhaust underground but low levels of potential occupational confounders (ie, radon, silica, asbestos) (12). Eligible subjects included all workers who were ever employed in a blue-collar job for at least 1 year after introduction of diesel equipment into the mining facility (year of introduction: 1947–1967 across the eight facilities) until the end of follow-up on December 31, 1997. The cohort consisted of 12315 workers with a total of 278041 person-years of follow-up. More detailed information on the cohort can be found in the accompanying article on the cohort study (12).

Case Subject Definition and Identification

Vital status of each cohort member was ascertained through December 31, 1997, by linkage with the National Death Index Plus (NDI Plus) (http://www.cdc.gov/nchs/ndi.htm) and Social Security Administration mortality files. Cause of death information was obtained from NDI Plus or from death certificates (for deaths occurring before the introduction of NDI Plus). A total of 217 deaths were identified with lung cancer (*International Classification of Diseases-O* code 162) specified as either the underlying or contributing cause on the death certificate. We attempted to retrieve pathology reports and diagnostic slides for all case subjects, which proved to be challenging because 85% of the case subjects had died more than 10 years before we contacted the hospital. After repeated attempts, we successfully obtained pathology reports or slides for 70 of the 170 case subjects for whom we obtained consent to access medical records. When the pathology report or diagnostic slides were available, the diagnosis of lung cancer was confirmed through review by an expert pathologist (W. D. Travis), which resulted in the exclusion of one case subject as "unlikely" to have had lung cancer. Of the 217 eligible case subjects identified, we interviewed 213 (98.1%) of their next of kin.

Control Subject Selection for the Nested Case–Control Study

Based on incidence density sampling, we selected up to four control subjects for each lung cancer case subject by random sampling from all members of the study cohort who were alive before the day the case subject died. With this design, all cohort members were eligible to serve as control subjects for more than one case subject, and case subjects before death were eligible to serve as control subjects for other case subjects who died earlier (23 control subjects went on to become case subjects at a later point in time).

Control subjects were individually matched to each case subject on mining facility, sex, race/ethnicity (ie, white, African American, American Indian, Hispanic), and birth year (within 5 years). In the analysis, estimates of diesel exposure and potential confounders (eg, cigarette smoking, employment in other high-risk occupations for lung cancer, and history of nonmalignant respiratory disease) for each control subject were truncated at the date of death of the matched case subject. We identified 650 eligible control subjects and interviewed 611 (94.0%) of them or their next of kin (if the control subject was deceased or too ill for interview). Of the next of kin who were interviewed, 55% were adult children, 31% were spouses or former spouses, 6% were siblings, and 8% were other relatives (with the exception of two friends/co-workers).

The Interview

Living control subjects (n = 222) and next of kin of lung cancer case subjects (n = 198) and ill or deceased control subjects (n = 340) were interviewed using a computer-assisted telephone interview (as explained below, an additional 15 case subjects and 49 control subjects were excluded from analysis). The interview was designed to collect information about the subject's demographics, smoking history (both active and passive), lifetime occupational history, medical history, family medical history, and usual adult diet. We obtained information on all jobs held for 12 months or longer since the age of 16. For each job held at a study mining facility, we collected information on the use of respiratory protective equipment (eg, respirators and masks) and the mining facility location where each subject spent most of his or her time (surface or underground) to supplement information obtained from the subject's company employment record. We also collected information about all jobs held before and after employment at the study mining facilities, including whether the subjects operated or worked near diesel engines.

We compared data obtained from next of kin of deceased control subjects to those obtained from direct interviews with living control subjects for several key variables (eg, cigarette smoking, history of employment in a high-risk occupation for lung cancer, and history of nonmalignant respiratory disease). In general, data obtained from next of kin were similar to those obtained from directly interviewed control subjects. For cigarette smoking, the percentages of direct vs next-of-kin interviews by smoking category were as follows: never smoker, 27% vs 28%; occasional smoker, 3% vs 2%; former smoker of less than one pack per day, 17% vs 17%; former smoker of one to less than two packs per day, 31% vs 24%; former smoker of two or more packs per day, 11% vs 6%; current smoker of less than one pack per day, 1% vs 3%; current smoker of one to less than two packs per day, 9% vs 14%; and current smoker of two or more packs per day, 1% vs 6%, respectively. Living control subjects and next of kin of dead control subjects reported similar proportions of "ever smokers" (73% and 72%, respectively). As expected, deceased control subjects had a slightly higher proportion of current smokers of one or more packs per day than living control subjects (20% and 10%, respectively). This observation is consistent with the reported cause of death; 80% of control subjects who were current smokers of one or more packs per day died of a smoking-related cause compared with 60% of control subjects who never smoked.

This study was approved by the Institutional Review Boards of the National Cancer Institute, the National Institute for Occupational Safety and Health (NIOSH), and Westat, Inc. All interviewees provided verbal informed consent before the interview, and next of kin of case subjects provided written consent to obtain medical records and pathology materials.

Diesel Exhaust Exposure Assessment

The eight facilities in the study had both underground (ore extraction) and surface (ore processing) operations. Underground workers were exposed to diesel exhaust primarily from ore extraction, haulage, and personnel transport vehicles. Surface workers generally had little to no contact with diesel equipment, although some had low levels of diesel exposure from the operation of heavy equipment or diesel trucks or because they worked near diesel equipment.

Respirable elemental carbon (REC), a component of diesel exhaust, is considered the best index of diesel exhaust in underground mining (13). The methods we used to develop quantitative estimates of historical exposure to REC at each mining facility have been described in detail (14–18). Briefly, the exposure assessors (P. A. Stewart, R. Vermeulen, J. B. Coble) developed location- and job title–specific estimates, by year, back to the year of the introduction of diesel equipment in each facility, blinded to mortality outcomes. The estimates were based on measurements from 1998 to 2001 DEMS industrial hygiene surveys at each working mining facility, past Mine Safety and Health Administration enforcement surveys, other measurement data, and information from company records and interviews with long-term workers. The same REC estimates were used to develop quantitative estimates of average intensity and cumulative REC exposure for subjects in both this and the cohort study (12).

A small percentage of subjects in the nested case–control study worked at more than one study facility (ie, 5.9% worked at two facilities and 0.7% worked at three). For these workers, their exposure metrics were based on diesel exposure at all relevant study facilities. Control subjects working in more than one facility were matched to case subjects on the facility where the control subject worked the longest. In facility-specific analyses, workers at multiple facilities were assigned to the facility where they worked the longest.

Statistical Analysis

The effect of diesel exhaust exposure on risk of dying of lung cancer was quantified by the odds ratio. Odds ratios and 95% confidence intervals were estimated by conditional logistic regression. Quartile and tertile cut points for exposure metrics were chosen to achieve approximately equal numbers of case subjects in each category. In all tables, statistical models included a term for exposure (ie, quartiles of average REC intensity [$\mu g/m^3$], cumulative REC exposure [$\mu g/m^3$-y], or duration of exposure [years]). Final models also included terms for potential confounding factors. These included a variable that combined cigarette smoking status and smoking intensity with location worked because initial analyses indicated that the risk of lung cancer from cigarette smoking was different for surface and underground workers (ie, smoking status [never, former, current], by smoking intensity [unknown or

occasional smoker, <1, 1 to <2, ≥2 packs per day], by location [surface only, ever underground]). Former smoker was defined as a case subject who had stopped smoking more than 2 years before their date of death and a control subject who had stopped smoking more than 2 years before the matched case subject's date of death. We included intensity smoked rather than duration smoked or pack-years in our final models; however, results were similar when either of these metrics was used to control for smoking (data not shown). The addition of a variable representing the interaction of location worked and smoking to models statistically significantly improved analogous models that included smoking without location (range of P values for the likelihood ratio test = .011–.064 for average REC intensity and cumulative REC, unlagged and lagged). The final models also included two other potential confounders: employment in a high-risk occupation for lung cancer for at least 10 years (ie, miner outside the study mining facilities, truck driver, welder, machinery mechanic, painter) and history of nonmalignant respiratory disease diagnosed at least 5 years before death/reference date (ie, primarily pneumoconiosis, emphysema, chronic obstructive pulmonary disease, silicosis, tuberculosis but excluding asthma, pneumonia, and bronchitis because the latter three diseases were not associated with lung cancer in our study). Other potential confounders [ie, duration of cigar smoking; frequency of pipe smoking; environmental tobacco smoke; family history of lung cancer in a first-degree relative; education; body mass index based on usual adult weight and height; leisure time physical activity; diet; estimated cumulative exposure to radon, asbestos, silica, polycyclic aromatic hydrocarbons (PAHs) from non-diesel sources, and respirable dust in the study facility based on air measurement and other data (14)] were evaluated but not included in the final models because they had little or no impact on odds ratios (ie, inclusion of these factors in the final models changed point estimates for diesel exposure by ≤10%). Exposure levels to other possible confounding exposures in these facilities, such as arsenic, nickel, and cadmium, were not estimated because of very low levels and generally non-detectable measurement results (14).

To test for trend, a Wald test was performed, treating the median value for each level of the categorical exposure variable among the control subjects as continuous in the model. To test for interaction between two risk factors, we added a cross-product term to the logistic model and conducted a likelihood ratio test between the model with and without the cross-product term. All statistical tests were two-sided.

We explored quantitative patterns in odds ratios for both continuous average REC intensity and continuous cumulative REC exposure, denoted by d, by fitting various standard models for occupational epidemiological data, including a log-linear model, $OR(d) = \exp(\beta d)$; a power model, $OR(d) = d^{\beta}$; a linear model, $OR(d) = 1 + \beta d$; and a linear-exponential model, $OR(d) = 1 + \beta d \exp(\gamma d)$. All models were adjusted for the same set of potential confounding factors as described above. We fitted models over the full range of exposure and, for comparative purposes, over a restricted range of lower exposure levels. We compared deviances (a measure of model fit) with the null model that omitted REC exposure, in which larger changes in deviance denoted greater improvements in fit (Supplementary Table 1, available online).

For average REC intensity and cumulative REC exposure, we evaluated lag intervals by excluding exposure occurring 0, 3, . . . , 25 years (by 2-year intervals) before the death/reference date and compared changes in model deviance to a model that omitted REC exposure. The optimal lag interval (ie, the largest improvement in model fit) occurred for a lag between 13–17 years for average REC intensity and 15 years for cumulative REC exposure (Supplementary Figure 1, available online). For consistency, we used a 15-year lag for both exposure metrics in the final analyses.

Of the 213 lung cancer case subjects and 611 control subjects interviewed for study, subjects were excluded for the following reasons: one case subject was identified as "unlikely" to have had lung cancer based on review of pathology material; 10 case subjects did not have any eligible control subjects (because of race/ethnicity for nine nonwhite or Hispanic case subjects and age for one case subject who was 88 years old); 39 control subjects were incorrectly matched on race/ethnicity based on more accurate information obtained during interview; four case subjects and five control subjects were found ineligible for inclusion in the cohort based on a final review of company work histories by NIOSH (12); and five control subjects were not suitable matches to any case subject because the original matched case subject was found to be ineligible for study. The final analytic dataset included 198 case subjects and 562 control subjects (666 control subjects for analytical purposes because some cohort members served as control subjects for more than one case subject). This analytical dataset was predominantly male, with only two female case subjects and eight female control subjects.

Results

Odds ratios for potential confounders (except cigarette smoking) and lung cancer risk are shown in Table 1. A statistically significant increased risk of lung cancer was observed for workers employed at least 10 years in occupations at high-risk for lung cancer (OR = 1.75, 95% CI = 1.06 to 2.91) (Table 1) and those with a history of nonmalignant respiratory disease for at least 5 years before death/reference date (OR = 2.15, 95% CI = 1.21 to 3.82) (Table 1). The elevated risk among those with nonmalignant respiratory disease less than 5 years before death may have been reflective of the early stages of lung cancer. Statistically nonsignificant increased risks were observed for workers who had a family history of lung cancer, smoked cigars for 10 or more years, lived with two or more smokers, exercised less than once per day, and had a vocational school education. Statistically nonsignificant decreased risks were observed among workers who were overweight or obese and who smoked at least 10 pipefuls of tobacco per week (Table 1).

Several non-diesel exposures present at very low levels (ie, levels not typically associated with risk in epidemiological studies) at the study mining facilities were not statistically significantly related to lung cancer risk in our study (Table 1). Levels of radon underground at the study mines were low (ie, arithmetic mean ≤0.02 Working Levels). The odds ratio for workers in the top quartile of cumulative radon exposure was 1.32 (95% CI = 0.76 to 2.29), and workers in quartiles 2 or 3 had little or no increased risk (Table 1). No consistent trend in risk with increasing cumulative radon exposure was apparent (P_{trend} = .220). Little or no

Table 1. Odds ratios (ORs) and 95% confidence intervals (CIs) by potential risk factors for lung cancer*

Potential risk factor	Case subjects	Control subjects	OR (95% CI)
Employment in other high-risk occupations, †‡			
No	100	365	1.0 (referent)
0 to <5y	24	90	0.90 (0.52 to 1.55)
5 to <10y	6	53	0.49 (0.19 to 1.21)
≥10y	39	68	1.75 (1.06 to 2.91)
Unknown	29	90	1.14 (0.67 to 1.92)
History of respiratory disease†§			
No	86	473	1.0 (referent)
<5 y before death/reference date	26	16	5.97 (2.93 to 12.19)
≥5 y before death/reference date	28	58	2.15 (1.21 to 3.82)
Unknown	58	119	2.94 (1.87 to 4.63)
Family history of lung cancer†			
No	136	532	1.0 (referent)
Yes	35	78	1.58 (0.97 to 2.57)
Unknown	27	56	1.65 (0.96 to 2.83)
Cigar smoking duration, y†			
Nonsmoker of cigars	176	564	1.0 (referent)
<10	8	42	0.81 (0.36 to 1.86)
10 to <20	5	16	1.46 (0.49 to 4.39)
≥20	3	14	1.67 (0.42 to 6.73)
Unknown	6	30	0.64 (0.24 to 1.67)
Pipe smoking, no. of pipefuls per week)‖			
Nonsmoker of pipes	153	487	1.0 (referent)
<10	11	39	0.89 (0.41 to 1.95)
10 to <20	6	24	0.66 (0.25 to 1.77)
≥20	5	35	0.50 (0.18 to 1.38)
Unknown	23	81	0.90 (0.52 to 1.57)
Number of smokers living in participant's childhood/adult home†			
0 smokers	28	164	1.0 (referent)
1 smoker	75	201	1.99 (1.20 to 3.30)
≥2 smokers	70	230	1.43 (0.84 to 2.44)
Unknown	25	71	1.30 (0.67 to 2.52)
Body mass index (kg/m^2)†			
<18.5 (underweight)	0	6	
18.5 to <25.0 (normal weight = referent)	105	285	1.0 (referent)
25.0 to <30.0 (overweight)	71	268	0.75 (0.51 to 1.11)
≥30.0 (obese)	14	59	0.73 (0.36 to 1.45)
Unknown	8	48	0.52 (0.23 to 1.19)
Physical activity†			
Exercise ≥1/d	23	110	1.0 (referent)
Exercise <1/d	162	515	1.46 (0.87 to 2.45)
Unknown	13	41	1.65 (0.70 to 3.89)
Education†			
Any college	22	88	1.0 (referent)
Vocational school	14	35	1.49 (0.63 to 3.52)
High school/GED	48	176	0.94 (0.51 to 1.72)
Less than high school	100	325	1.09 (0.61 to 1.98)
Unknown	14	42	1.40 (0.62 to 3.18)
Radon, quartiles (Working Level Months)¶#**			
No exposure	74	254	1.0 (referent)
>0 to <0.6	31	117	0.73 (0.43 to 1.25)
0.6 to <1.9	31	123	0.86 (0.51 to 1.45)
1.9 to <3.0	31	80	1.08 (0.63 to 1.84)
≥3.0	31	92	1.32 (0.76 to 2.29)
Asbestos, quartiles†¶††			
No exposure	122	402	1.0 (referent)
>0 to <1.1	19	40	1.12 (0.59 to 2.10)
1.1 to <5.9	19	92	0.73 (0.41 to 1.29)
5.9 to <13.7	19	73	0.81 (0.44 to 1.48)
≥13.7	19	59	1.08 (0.59 to 2.01)

(Table continues)

Table 1 (Continued).

Potential risk factor	Case subjects	Control subjects	OR (95% CI)
Silica, quartiles†¶††			
No exposure	48	169	1.0 (referent)
>0 to <4.6	37	111	0.68 (0.25 to 1.90)
4.6 to <12.6	37	155	0.56 (0.19 to 1.61)
12.6 to <20.5	38	86	1.07 (0.37 to 3.14)
≥20.5	38	145	0.78 (0.26 to 2.32)
PAHs from non-diesel sources, quartiles†¶‡‡			
No exposure	120	398	1.0 (referent)
>0 to <1.2	19	49	1.03 (0.55 to 1.91)
1.2 to <5.1	20	74	0.94 (0.53 to 1.68)
5.1 to <12.3	19	81	0.87 (0.48 to 1.57)
≥12.3	20	64	1.06 (0.59 to 1.92)
Cumulative respirable dust, quartiles, mg/m^3-y*¶§§			
0 to <5.66	49	142	1.0 (referent)
5.66 to <14.08	50	184	0.91 (0.52 to 1.57)
14.08 to <29.54	49	194	0.86 (0.49 to 1.52)
≥29.54	50	146	1.31 (0.70 to 2.46)

* *P* values based on two-sided Wald test for linear trend; PAH = polycyclic hydrocarbon; WL = Working Level; WLM = Working Level Months.

† Adjusted for smoking status/mine location combination (surface work only/never smoker, surface work only/unknown/occasional smoker, surface work only/former smoker/<1 pack per day, surface work only/former smoker/1 to <2 packs per day, surface work only/former smoker/≥2 packs per day, surface work only/current smoker/<1 packs per day, surface work only/current smoker/1 to <2 packs per day, surface work only/current smoker/≥2 packs per day, ever underground work/never smoker, ever underground work/unknown/occasional smoker, ever underground work/former smoker/<1 pack per day, ever underground work/former smoker/1 to <2 packs per day, ever underground work/former smoker/≥ 2 packs per day, ever underground work/current smoker/<1 pack per day, ever underground work/current smoker/1 to <2 packs per day, ever underground work/current smoker/≥2 packs per day).

‡ Other high-risk occupations for lung cancer (ie, miner who worked outside the study mines, truck driver, welder, machinery mechanic, painter).

§ History of respiratory disease excluding asthma, pneumonia, and bronchitis.

‖ Adjusted for cigarette smoking and education.

¶ Pertains only to exposures at study mines.

Quartiles of cumulative radon exposure derived from estimated levels in WL multiplied by months at each job, summed across jobs. Thus, exposure to radon is expressed in units of WLM. One WL = 130000 MeV alpha energy per liter of air, and one WLM is equivalent to 1 WL exposure for 170 hours.

** Adjusted for smoking status: unknown, never smoker, occasional smoker, former smoker/<1 pack per day, former smoker/1 to <2 packs per day, former smoker/≥2 packs per day, current smoker/<1 pack per day, current smoker/1 to <2 packs per day, current smoker/≥2 packs per day.

†† Quartiles of cumulative exposure derived from intensity scores (0–3) multiplied by years at each job, summed across jobs.

‡‡ Quartiles of cumulative exposure derived from the presence or absence of non-diesel PAHs based on job title tasks (0,1) multiplied by years at each job, summed across jobs.

§§ Respirable dust in milligrams per cubic meter multiplied by years of exposure.

increased risk was observed for possible exposure to asbestos, silica, and PAHs from non-diesel sources, which was consistent with the low measured mean air levels of these potential confounding variables (Table 1) (14). Workers in the top quartile of cumulative respirable dust exposure had an elevated risk (OR = 1.31, 95% CI = 0.70 to 2.46), but workers in quartiles 2 or 3 had no increased risk (Table 1). Factors with statistically nonsignificant increased or decreased risks had little or no confounding effect on estimates of risk from diesel exposure (ie, changed point estimates by ≤10%) and were not included in the final models.

Table 2 shows the effect of cigarette smoking overall and cross-classified by location of employment (ie, surface only and ever underground). Overall, for both surface-only and ever underground workers combined, the risk of lung cancer was statistically significantly associated with smoking status (never, former, current smoker) and smoking intensity (former smoker of ≥2 packs per day vs never smoker: OR = 5.40, 95% CI = 2.23 to 13.06; current smoker of ≥2 packs per day vs never smoker: OR = 12.41, 95% CI = 5.57 to 27.66) (Table 2). We also observed an interaction between cigarette smoking and location of employment, after adjustment for cumulative REC, lagged 15 years ($P_{interaction}$ = .082). The lung cancer risks associated with moderate (1 to <2 packs per day) and heavy smoking (≥2 packs per day) were higher among workers who only worked at the surface than among those who ever worked underground for both current and former smokers. For example, the odds ratio for current smokers of one to less than two packs per day who worked only at the surface was 13.34 (95% CI = 4.50 to 39.53) compared with an OR of 4.51 (95% CI = 1.50 to 13.58) for those who ever worked underground (Table 2). Because the effect of smoking appeared to be diminished among underground workers compared with that among surface workers, we included the cross classification of location of employment, smoking status, and smoking intensity in all models used to estimate lung cancer risk by diesel exposure (Tables 1, 3, and 7; Figure 1), unless noted otherwise. It is also noteworthy that among never smokers, underground and surface-only workers had similar risks after adjustment for 15-year lagged cumulative REC (OR = 0.90; 95% CI = 0.26 to 3.09) (Table 2), suggesting that the risk experienced by surface-only workers was mainly due to smoking.

Table 2. Odds ratios (ORs) and 95% confidence intervals (CIs) for smoking status/smoking intensity by location of employment*

Smoking status/smoking intensity (packs per day)	OR (95% CI), No. of case subjects/No. of control subjects		
	Surface only†, average REC intensity (0–8 μg/m³ REC)	Ever underground†, average REC intensity (1–423 μg/m³ REC)	All subjects‡
Never smoker	1.0 (referent), 5/87	0.90 (0.26 to 3.09), 9/91	1.0 (referent), 14/178
Former, <1	1.36 (0.24 to 7.59), 2/31	2.51(0.78 to 8.11), 17/62	2.87 (1.30 to 6.33), 19/93
Former, 1 to <2	6.66 (2.07 to 21.50), 14/40	1.97 (0.61 to 6.37), 16/68	3.56 (1.72 to 7.40), 30/108
Former, ≥2	16.30 (3.55 to 74.82), 6/7	2.70 (0.72 to 10.12), 9/29	5.40 (2.23 to 13.06), 15/36
Current, <1	5.22 (1.16 to 23.39), 4/15	5.71 (1.63 to 20.01), 12/21	5.91 (2.47 to 14.10), 16/36
Current, 1 to <2	13.34 (4.50 to 39.53), 26/41	4.51 (1.50 to 13.58), 32/78	7.36 (3.71 to 14.57), 58/119
Current, ≥2	26.60 (7.14 to 99.08), 12/9	7.13 (2.12 to 23.99), 17/27	12.41 (5.57 to 27.66), 29/36
Unknown§	2.86 (0.71 to 11.64), 5/24	2.65 (0.76 to 9.24), 12/36	3.10 (1.33 to 7.26), 17/60

* REC = respirable elemental carbon.

† ORs relative to never smokers who worked only surface jobs, adjusted for cumulative REC, lagged 15 years (quartiles: 0 to <3 μg/m³-y; 3 to <72 μg/m³-y, 72 to <536 μg/m³-y, ≥536 μg/m³-y), history of respiratory disease 5 or more years before date of death/reference date, and history of a high-risk job for lung cancer for at least 10 years. *P* value for interaction between smoking status and location of employment based on likelihood ratio test = .082.

‡ ORs for intensity smoked relative to never smokers, adjusted for cumulative REC, lagged 15 years (quartiles: 0 to <3 μg/m³-y; 3 to <72 μg/m³-y, 72 to <536 μg/m³-y, ≥536 μg/m³-y), location of employment (surface only, ever underground), history of respiratory disease 5 or more years before date of death/reference date, and history of a high-risk job for lung cancer for at least 10 years.

§ Unknown includes subjects with unknown smoking status, and subjects considered occasional smokers, who smoked at least 100 cigarettes during their lifetimes, but never smoked regularly (≥1 cigarette per day for at least 6 months).

Trends in risk with increasing levels of diesel exposure are either statistically significant or of borderline significance ($P_{trend} \leq .08$) for all metrics (both unlagged and lagged) (Table 3). The strongest gradient in risk was seen for 15-year lagged cumulative REC ($P_{trend} = .001$). The odds ratio for workers in the top quartile of 15-year lagged cumulative REC exposure (ie, ≥536 μg/m³-y) was 2.83 (95% CI = 1.28 to 6.26) compared with workers in the lowest quartile. When the top exposure quartile was split at the median (ie, 1005 μg/m³-y), the risk continued to rise (P_{trend} over all five exposure levels = .002); odds ratios were 2.53 (95% CI = 1.06 to 6.04) and 3.20 (95% CI = 1.33 to 7.69) for workers in the top quartile with cumulative REC exposures below and above the median of the quartile, respectively.

We observed a statistically significant gradient in risk with increasing number of years exposed to diesel exhaust among all workers ($P_{trend} = .043$), although an elevated odds ratio occurred only in the highest duration category. The odds ratio for workers exposed to diesel exhaust for 15 or more years was 2.09 (95% CI = 0.89 to 4.90) compared with surface workers with negligible or bystander exposure (Table 3).

We also examined risk among all subjects who ever worked underground (Table 4) and among those who worked only at the surface (Table 5). Among underground workers, we observed statistically significant trends in risk with increasing average REC intensity, unlagged ($P_{trend} = .01$) and lagged 15 years ($P_{trend} = .001$), and with increasing cumulative REC, lagged 15 years ($P_{trend} = .004$) (Table 4). Among surface workers, in contrast, no consistent positive gradient in risk with increasing diesel exposure was apparent (Table 5), probably due to the small number of subjects (53 case subjects and 100 control subjects) and the low levels of diesel exposure experienced by surface workers. Because of the increased precision gained by estimating odds ratios based on all subjects, our primary estimates of risk are based on surface and underground workers combined (Table 3).

We stratified the combined results (Table 3) on whether the subject had self-reported diesel exhaust exposure from a job outside the study mining facility (eg, ever employed as a long-haul truck driver) (data not shown). No systematic differences in risk were apparent among subjects with or without occupational diesel exposure outside the study facility ($P_{interaction}$ between cumulative REC, lagged 15 years, and outside occupational diesel exhaust exposure = .222).

Use of protective equipment did not appear to modify the observed associations between diesel exhaust exposure and lung cancer. However, most information on protective equipment use was obtained from next-of-kin interviews, resulting in a large number of workers with unknown data (59 case subjects and 129 control subjects). Subjects who reported having used protective equipment appeared to experience risks similar to the estimates for all workers combined (Table 3). For example, among workers who used protective equipment, odds ratios for 15-year lagged cumulative REC exposures of less than 3 μg/m³-y, 3 to less than 72 μg/m³-y, 72 to less than 536 μg/m³-y, and 536 μg/m³-y or more were 1.0 (referent), 0.31 (95% CI = 0.04 to 2.23; 16 case subjects and 42 control subjects), 1.76 (95% CI = 0.11 to 27.91; 10 case subjects and 23 control subjects), and 3.66 (95% CI = 0.26 to 52.09; 20 case subjects and 31 control subjects), respectively.

Figure 1 shows category-specific odd ratios (square symbol), with confidence intervals omitted for clarity, and fitted odds ratios for 15-year lagged average REC intensity and cumulative REC using various continuous models. To provide additional points for graphing the exposure–response curve based on categorical data (Figure 1), we expanded the number of cut points (cut points for average REC intensity, lagged 15 years: <2, 2 to <4, 4 to <8, 8 to <16, 16 to <32, 32 to <64, 64 to <128, 128 to <256, and ≥256 μg/m³; cut points for cumulative REC, lagged 15 years, were similarly defined but multiplied by a factor of 10 to account for duration of exposure: <20, 20 to <40, 40 to <80, 80 to <160, 160 to <320, 320

Table 3. Odds ratios (ORs) and 95% confidence intervals (CIs) for average and cumulative REC and total duration REC exposure*

Exposure metric	Case subjects	Control subjects	OR (95% CI)	P_{trend}
Average REC intensity, quartiles, unlagged, μg/m³				
0 to <1	49†	166	1.0 (referent)	.025
1 to <32	50	207	1.03 (0.50 to 2.09)	
32 to <98	49	145	1.88 (0.76 to 4.66)	
≥98	50	148	2.40 (0.89 to 6.47)	
Quartiles, lagged 15 y, μg/m³				
0 to <1	47†	190	1.0 (referent)	.062
1 to <6	52	187	1.11 (0.59 to 2.07)	
6 to <57	49	141	1.90 (0.90 to 3.99)	
≥57	50	148	2.28 (1.07 to 4.87)	
Cumulative REC, quartiles, unlagged, μg/m³-y				
0 to <19	49	151	1.0 (referent)	.083
19 to <246	50	214	0.87 (0.48 to 1.59)	
246 to <964	49	147	1.50 (0.67 to 3.36)	
≥964	50	154	1.75 (0.77 to 3.97)	
Quartiles, lagged 15 y, μg/m³-y				
0 to <3	49	158	1.0 (referent)	.001
3 to <72	50	228	0.74 (0.40 to 1.38)	
72 to <536	49	157	1.54 (0.74 to 3.20)	
≥536	50	123	2.83 (1.28 to 6.26)	
Duration of REC exposure, y				
Unexposed‡	48	165	1.0 (referent)	.043
0 to <5	51	169	1.16 (0.53 to 2.55)	
5 to <10	20	95	0.88 (0.38 to 2.03)	
10 to <15	31	107	0.93 (0.39 to 2.21)	
≥15	48	130	2.09 (0.89 to 4.90)	

* *P* values based on two-sided Wald test for linear trend; adjusted for smoking status/mine location combination (surface work only/never smoker, surface work only/unknown/occasional smoker, surface work only/former smoker/<1 pack per day, surface work only/former smoker/1 to <2 packs per day, surface work only/former smoker/≥2 packs per day, surface work only /current smoker/<1 pack per day, surface work only/current smoker/1 to <2 packs per day, surface work only/current smoker/≥2 packs per day, ever underground work/never smoker, ever underground work/unknown/occasional smoker, ever underground work/former smoker/<1 pack per day, ever underground work/former smoker/1 to <2 packs per day, ever underground work/former smoker/≥2 packs per day, ever underground work/current smoker/<1 pack per day, ever underground work/current smoker/1 to <2 packs per day, ever underground work/current smoker/≥2 packs per day); history of respiratory disease 5 or more years before date of death/reference date; and history of a high-risk job for lung cancer for at least 10 years. REC = respirable elemental carbon.

† The number of case subjects in the referent group for the 15-year lagged average REC analysis is 2 fewer than that in the unlagged analysis because rounded cut points are presented. The unrounded cut points are <0.86 and <1.37 μg/m³, respectively.

‡ Unexposed includes all subjects who worked surface jobs with either negligible or bystander exposure to REC, regardless of duration.

to <640, 640 to <1280, 1280 to <2560, and ≥2560 μg/m³-y; Supplementary Table 2, available online). Odds ratios increased with 15-year lagged average REC intensity and leveled off above 20–80 μg/m³ (Figure 1, A for the full range and Figure 1, B for average REC intensity under 128 μg/m³). For the full range, the odds ratio pattern was best explained by a one-parameter power model (deviance = 5.3, $P = .022$), whereas for the restricted range, the power and linear models were comparable (deviance = 2.8, $P = .092$ and deviance = 3.2, $P = .075$, respectively). A similar increasing pattern of odds ratios was observed for cumulative REC exposure, lagged 15 years (Figure 1, C for the full range and Figure 1, D for cumulative REC under 1280 μg/m³-y), with a leveling off of risk for exposures above 1,000 μg/m³-y and perhaps a decline in risk among the most heavily exposed workers. The two-parameter linear-exponential model (dotted line) was the best fitting model for the full range (relative to the null model, deviance = 12.2, $P = .002$) (Figure 1, C); for the restricted range, the best models were the one-parameter linear model (dashed-dotted line) (deviance = 15.6, $P < .001$) and the two-parameter linear-exponential model (dotted line) (deviance = 16.0, $P < .001$) (Figure 1, D) (Supplementary Table 1, available online). We carried out similar model comparisons using the unlagged exposure metrics (Supplementary Table 3, available online). However, our evaluation of optimal lag intervals (Supplementary Figure 1, available online) suggested that the unlagged approach led to exposure misclassification because recent exposures may not have had sufficient time to contribute to lung cancer risk and thus resulted in generally poorer fit of the various models.

The combined effect of diesel exposure and intensity of cigarette smoking is shown in Table 6. Among the 14 case subjects and 178 control subjects who never smoked, odds ratios by tertile of cumulative REC, lagged 15 years, were: 1.0 (referent), OR = 1.47 (95% CI = 0.29 to 7.50), and OR = 7.30 (95% CI = 1.46 to 36.57). Risk also increased with increasing level of diesel exposure among smokers of less than one and one to less than two packs per day. In contrast, risk decreased with increasing levels of diesel exposure among smokers of at least two packs per day. Similarly, risk associated with smoking intensity was modified by diesel exposure. Among workers in the lowest tertile of cumulative REC, lagged 15 years, smokers of at least two packs per day had a risk 27 times that of nonsmokers, whereas among those in the highest tertile of cumulative REC, heavy smokers had about 2.5-fold the risk of nonsmokers. The $P_{interaction}$ between level of diesel exposure and cigarette smoking was .086.

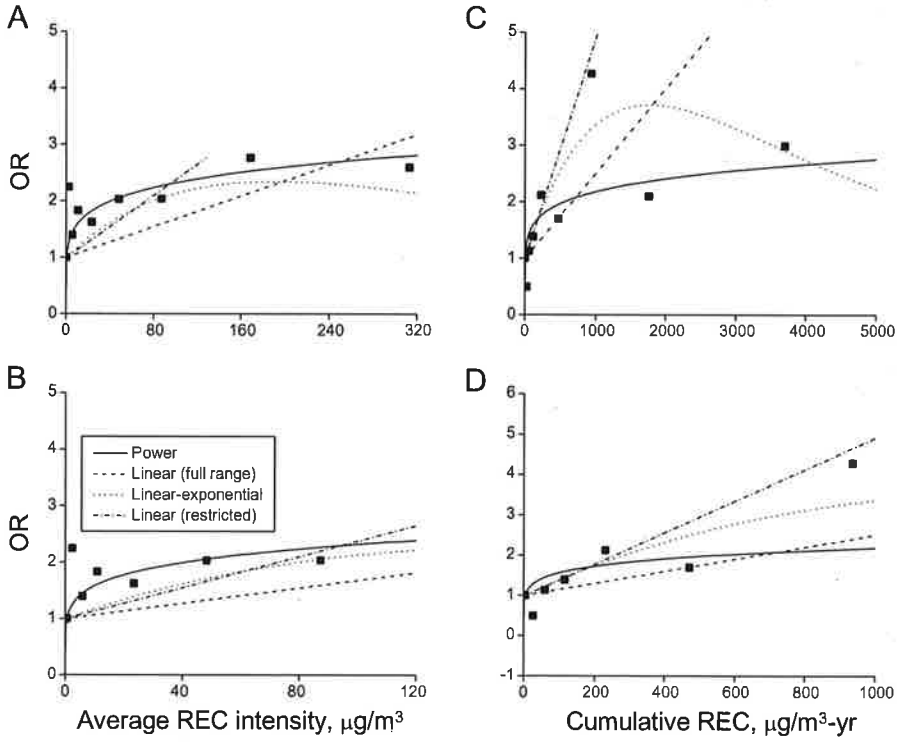

Figure 1. Odds ratios (ORs) (**solid squares**) for lung cancer by expanded categories of average respirable elemental carbon (REC) intensity and cumulative REC (Supplementary Table 2, available online). **A**) Average REC intensity, full range; **B**) Average REC intensity, less than 128 µg/m³; **C**) Cumulative REC exposure, full range; **D**) Cumulative REC exposure, less than 1280 µg/m³-y. ORs located at the mean exposure within category. Models for OR by continuous exposure (d) include a power model, $OR(d) = d^{\beta}$ (**solid line**); a linear model, $OR(d) = 1 + \beta\, d$ (**dashed line** for the full range and **dashed-dotted line** for the restricted range); and a linear-exponential model, $OR(d) = 1 + \beta\, d \exp(\gamma\, d)$ (**dotted line**). Exposure variables were based on a 15-year lag. Confidence intervals were omitted for clarity. The log-linear model was excluded because it did not fit the data well.

We evaluated lung cancer risk by quantitative level of diesel exposure for each type of mining facility (Table 7). Too few workers were employed in the one salt and the one limestone mining facility to estimate risk for these types separately. For workers in both potash and trona mining facilities, risk tended to increase with increasing levels of average REC intensity and cumulative REC exposure. Trends were more consistent among potash miners, perhaps reflecting more stability in odds ratios resulting from twice as many case subjects in the potash as in the trona facilities (Table 7).

Discussion

This case–control study nested within a cohort of miners showed a strong and consistent relation between quantitative exposure to diesel exhaust and increased risk of dying of lung cancer. To our knowledge, this is the first report of a statistically significant exposure–response relationship for diesel exposure and lung cancer based on quantitative estimates of historical diesel exposure with adjustment for smoking and other potential confounders. We observed increasing trends in risk with increasing exposure to diesel exhaust for both average REC intensity and cumulative REC exposure, unlagged and lagged 15 years, with the strongest gradient in risk with cumulative REC, lagged 15 years. We further observed a gradient of increasing risk within the top quartile of 15-year lagged cumulative REC exposure for workers below and above the median of the quartile. The associations between diesel exposure and lung cancer were apparent for workers employed in either the potash or trona facilities (too few workers were employed in the one salt and one limestone mine to estimate risk separately). The consistency of findings for both potash and trona facilities is noteworthy because smoking was prohibited in the trona facilities but not in potash or the other facilities in the study. Reports by next of kin or study subjects of workers' use of protective equipment within the study mining facilities and workers' additional occupational exposure to diesel exhaust outside the study facilities had little or no impact on our findings.

These positive findings are consistent with those of the cohort analysis of underground workers in the same study population (12). However, estimates of risk for underground workers in the case–control analysis were somewhat higher than those based on the cohort analysis. For example, the odds ratios by quartile of the 15-year lagged cumulative REC exposure in the case–control analysis were 1.0, 2.11, 3.48, and 5.90 (for cohort cut points, <108, 108 to <445, 445 to <946, and ≥946 µg/m³-y, respectively), compared with hazard ratios of 1.0, 1.50, 2.17, and 2.21 from the cohort analysis (12). The lower point estimates from the cohort analysis may be partly due to negative confounding from cigarette smoking because current smoking was inversely related to diesel exposure in underground workers (36% and 21% current smokers in lowest vs highest cumulative REC tertile, respectively). Odds ratios for underground workers in the case–control analysis using the same cohort cut points dropped to 1.0, 1.94, 2.42, and 3.75, respectively, when smoking was removed from the model.

The continuous models suggest a steep slope at the low end of the exposure–response curve followed by a leveling, or perhaps even a decline, in risk among the most heavily exposed workers. A plateauing of exposure–response curves has been reported in studies of other occupational exposures and cancer risk (19). Possible biological explanations for a plateauing effect include saturation of metabolic activation and enhanced detoxification or greater DNA repair efficiency at higher exposure levels.

Table 4. Odds ratios (ORs) and 95% confidence intervals (CIs) for average and cumulative REC and total duration REC exposure for subjects who ever worked underground jobs*

Exposure metric	Case subjects†	Control subjects†	OR (95% CI)	P_{trend}
Average REC intensity, quartiles, unlagged, μg/m³				
0 to <39	29	89	1.0 (referent)	.010
39 to <71	29	57	1.91 (0.91 to 4.01)	
71 to <147	29	66	2.38 (1.04 to 5.44)	
≥147	29	52	3.69 (1.40 to 9.70)	
Quartiles, lagged 15 y, μg/m³				
0 to <8	29	81	1.0 (referent)	.001
8 to <49	29	73	1.04 (0.45 to 2.43)	
49 to <104	29	58	2.19 (0.87 to 5.53)	
≥104	29	52	5.43 (1.92 to 15.31)	
Cumulative REC, quartiles, unlagged, μg/m³-y				
0 to <298	29	81	1.0 (referent)	.123
298 to <675	29	63	1.45 (0.68 to 3.11)	
675 to <1465	29	57	1.81 (0.84 to 3.89)	
≥1465	29	63	1.93 (0.90 to 4.15)	
Quartiles, lagged 15 y, μg/m³-y				
0 to <81	29	92	1.0 (referent)	.004
81 to <325	29	52	2.46 (1.01 to 6.01)	
325 to <878	29	69	2.41 (1.00 to 5.82)	
≥878	29	51	5.10 (1.88 to 13.87)	
Duration of REC exposure, y				
<5	37	92	1.0 (referent)	.062
5 to <10	14	39	1.18 (0.52 to 2.68)	
10 to <15	25	60	0.84 (0.39 to 1.82)	
≥15	40	73	2.08 (1.01 to 4.27)	

* *P* values based on two-sided Wald test for linear trend. Adjusted for smoking status (never smoker, unknown/occasional smoker, former smoker/<1 pack per day, former smoker/1 to <2 packs per day, former smoker/≥2 packs per day, current smoker/<1 pack per day, current smoker/1 to <2 packs per day, current smoker/ ≥2 packs per day); history of respiratory disease 5 or more years before date of death/reference date; and history of a high-risk job for lung cancer for at least 10 years. REC = respirable elemental carbon.

† Eight case subjects and 148 control subjects were excluded because they no longer belonged to a complete matched set after analysis was restricted to underground workers.

Alternatively, nondifferential misclassification of diesel exposure may be greater at higher exposures, obscuring further increases in risk.

We observed an increased lung cancer risk associated with diesel exposure as was seen among German potash miners (11), as well as among other diesel-exposed occupational groups including truck drivers (6,7), railroad workers (8,20), dockworkers (9), and bus garage workers (10). The German potash miners study (11) found elevated risk with increasing estimated cumulative total carbon exposure (another surrogate for diesel exposure), although the trend was not statistically significant. Relative risks were 1.0, 1.13, 2.47, 1.50, and 2.28 for exposure quintiles (ie, <1.29, 2.04, 2.73, 3.90, >3.90 mg/m³-y, respectively) (11). Some differences between the German study (11) and this study are that this study is considerably larger (US miners: 198 lung cancer deaths out of a total of 278041 person-years; German miners: 61 lung cancer deaths out of 152557 person-years), and the US miners had a longer latent period for the development of lung cancer than the German miners because diesel technology was introduced earlier in the US study mines (1947–1967) than in the German mines (1969). Finally, in this study, an intensive effort was undertaken to characterize diesel exposure levels over time by incorporating changes in size of the diesel equipment, numbers of equipment, and air flow rates exhausted from the mines based on information collected from the facilities. Our information indicated that these factors varied considerably over time (14). In the German study, the investigators relied on reports from local engineers and industrial hygienists that working conditions were constant over past years. However, in contrast to this study, no past industrial hygiene measurements were available to confirm this assumption.

We observed an attenuation of the effect of cigarette smoking among study subjects who were exposed to high levels of diesel exhaust as estimated by REC (Table 6). This finding mirrors a recent observation from a study in Xuanwei, China (21), where lung cancer rates are high because of unvented indoor burning of coal for heating and cooking in homes (22). The effect of tobacco on lung cancer risk in that study was weak in the presence of heavy indoor exposures from smoky coal but became stronger with installation of venting, which greatly diminished smoky coal air concentrations (21,22). Little is known about the effect of the interaction between cigarette smoking and diesel exhaust exposure on lung cancer risk. If our observation of attenuation of the smoking effect in the presence of high levels of diesel exhaust is confirmed, several possible mechanistic explanations are apparent. First, at high levels of diesel exhaust exposure, PAHs, nitro-PAHs, and related compounds could compete with the metabolic activation of PAHs in tobacco smoke, leading to enzyme saturation. For example, PAHs in complex mixtures have been shown to have less than additive genotoxic effects at higher exposure levels (23). Second, constituents of diesel exhaust may suppress enzymes that

Table 5. Odds ratios (ORs) and 95% confidence intervals (CIs) for average and cumulative REC and total duration REC exposure for subjects who worked only surface jobs*

Exposure metric	Case subjects†	Control subjects†	OR (95% CI)	P_{trend}
Average REC intensity, quartiles, unlagged, μg/m³				
0 to <0.86	13	24	1.0 (referent)	.983
0.86 to <0.95	13	21	1.29 (0.18 to 9.33)	
0.95 to <1.9	13	19	7.24 (0.23 to 228.53)	
≥1.9	14	36	3.28 (0.09 to 123.50)	
Quartiles, lagged 15 y, μg/m³				
0 to <0.6	13	38	1.0 (referent)	.659
0.6 to <0.9	13	17	4.38 (0.56 to 34.24)	
0.9 to <1.4	13	12	5.67 (0.77 to 42.06)	
≥1.4	14	33	1.31 (0.14 to 12.01)	
Cumulative REC, quartiles, unlagged, μg/m³-y				
0 to <6.5	13	17	1.0 (referent)	.294
6.5 to <12.5	13	27	0.78 (0.18 to 3.43)	
12.5 to <22.5	13	23	0.60 (0.14 to 2.53)	
≥22.5	14	33	0.40 (0.07 to 2.40)	
Quartiles, lagged 15 y, μg/m³-y				
0 to <0.7	13	29	1.0 (referent)	.117
0.7 to <4.4	13	9	3.98 (0.69 to 23.02)	
4.4 to <14.3	13	32	0.76 (0.12 to 4.98)	
≥14.3	14	30	0.42 (0.05 to 3.59)	
Duration REC exposure, y				
Unexposed‡	34	61	1.0 (referent)	.152
0 to <5	10	17	1.44 (0.26 to 8.17)	
5 to <10	5	12	0.74 (0.10 to 5.21)	
10 to <15	3	3	0.55 (0.05 to 6.17)	
≥15	1	7	0.22 (0.01 to 3.67)	

* *P* values based on two-sided Wald test for linear trend. Adjusted for smoking status (never smoker, unknown/occasional smoker, former smoker/<1 pack per day, former smoker/1 to <2 packs per day, former smoker/≥2 packs per day, current smoker/<1 pack per day, current smoker/1 to <2 packs per day, current smoker/≥2 packs per day); history of respiratory disease 5 or more years before date of death/reference date; and history of a high-risk job for lung cancer for at least 10 years. REC = respirable elemental carbon.

† Twenty-one case subjects and 154 control subjects were excluded because they no longer belonged to a complete matched set after analysis was restricted to surface workers.

‡ Unexposed includes subjects who worked surface jobs with either negligible or bystander exposure to REC.

activate or induce enzymes that detoxify carcinogens in tobacco smoke. For example, diesel exhaust particles have been shown to reduce activity of CYP2B1, which plays a role in the activation of certain tobacco-specific nitrosamines (24). Also, diesel particulate matter has been shown to reduce the initiation of skin tumors in Sencar mice treated with the potent PAH dibenzo[a,l]pyrene, possibly through inhibition of enzymes that carry out its metabolic activation (25).

We also observed a weakening of the diesel exhaust effect among heavy smokers (ie, smokers of at least two packs per day), which is necessarily implied by the observation of a weakening of the effect of smoking at least two packs per day among workers heavily exposed to diesel exhaust. It has previously been reported that coal dust burden in the lungs of coal miners is reduced among smokers, which may be attributable to increased coal dust clearance (26), and it is possible that diesel exhaust particulate deposition may be reduced in the lungs of smokers by a similar process. Although little experimental evidence is available to date to support and explain effect modification of diesel exposure by smoking, it is theoretically possible by one or more of the mechanisms described above.

Table 6. Odds ratios (ORs) and 95% confidence intervals (CIs) for cumulative REC lagged 15 years crossed with smoking intensity*

Smoking intensity (packs per day)	Cumulative REC lagged 15 years OR (95% CI), No. of case subjects/No. of control subjects		
	Tertile 1, 0 to < 8 μg/m³-y	Tertile 2, 8 to < 304 μg/m³-y	Tertile 3, ≥304 μg/m³-y
Never smoker	1.0 (referent), 3/59	1.47 (0.29 to 7.50), 4/74	7.30 (1.46 to 36.57), 7/45
<1	6.25 (1.42 to 27.60), 10/41	7.42 (1.62 to 34.00), 10/49	16.35 (3.45 to 77.63), 15/39
1 to <2	10.16 (2.55 to 40.53), 29/78	11.58 (2.87 to 46.71), 32/86	20.42 (4.52 to 92.36), 27/63
≥2	26.79 (6.15 to 116.63), 19/22	22.17 (4.84 to 101.65), 15/22	17.38 (3.48 to 86.73), 10/28
Unknown†	4.13 (0.74 to 23.22), 4/25	3.79 (0.64 to 22.41), 4/23	27.85 (5.03 to 154.31), 9/12

* Adjusted for history of respiratory disease 5 or more years before date of death/reference date, history of a high-risk job for lung cancer for at least 10 years, and mine location (surface-only vs any underground work). *P* value for interaction between smoking intensity and cumulative REC lagged 15 years = .086. REC = respirable elemental carbon.

† Unknown includes subjects with unknown smoking status, and subjects considered occasional smokers, who smoked at least 100 cigarettes during their lifetimes, but never smoked regularly (≥1 cigarette per day for at least 6 months).

Table 7. Odds ratios (ORs) and 95% confidence intervals (CIs) for average and cumulative REC lagged 15 years, by mining facility type*

Exposure by mine type	Case subjects	Control subjects	OR (95% CI)	P_{trend}
Potash				
Average REC intensity, lagged 15 years, quartiles, μg/m³				
0 to <1	25	95	1.0 (referent)	.058
1 to <6	20	51	1.16 (0.49 to 2.76)	
6 to <57	30	105	2.05 (0.70 to 6.01)	
≥57	27	85	3.01 (0.98-9.25)	
Cumulative REC, lagged 15 years, quartiles, μg/m³-y				
0 to <3	19	60	1.0 (referent)	.006
3 to <72	30	103	1.64 (0.67 to 3.98)	
72 to <536	25	105	2.50 (0.86 to 7.24)	
≥536	28	68	5.53 (1.68 to 18.21)	
Trona				
Average REC intensity, lagged 15 years, quartiles, μg/m³				
0 to <1	17	70	1.0 (referent)	.105
1 to <6	18	64	2.32 (0.52 to 10.40)	
6 to <57	2	6	1.71 (0.12 to 23.66)	
≥57	14	34	5.95 (0.92 to 38.37)	
Cumulative REC, lagged 15 years, quartiles, μg/m³-y				
0 to <3	24	72	1.0 (referent)	.062
3 to <72	11	64	0.23 (0.06 to 0.91)	
72 to <536	7	17	0.95 (0.16 to 5.72)	
≥536	9	21	2.38 (0.44 to 13.00)	

* *P* values based on two-sided Wald test for linear trend. Adjusted for smoking status/mine location combination (surface work only/never smoker, surface work only/unknown/occasional smoker, surface work only/former smoker/<1 pack per day, surface work only/former smoker/1 to <2 packs per day, surface work only/former smoker/≥2 packs per day, surface work only/current smoker/<1 pack per day, surface work only/current smoker/1 to <2 packs per day, surface work only/current smoker/≥2 packs per day, ever underground work/never smoker, ever underground work/unknown/occasional smoker, ever underground work/former smoker/<1 pack per day, ever underground work/former smoker/1 to <2 packs per day, ever underground work/former smoker/≥2 packs per day, ever underground work/current smoker/<1 pack per day, ever underground work/current smoker/1 to <2 packs per day, ever underground work/current smoker/≥2 packs per day); history of respiratory disease 5 or more years before date of death/reference date; and history of a high-risk job for lung cancer for at least 10 years. REC = respirable elemental carbon.

If the observed interaction between smoking and diesel exhaust represents a real effect, then the generalizability of our estimates of risk for diesel exposure to other populations depends not only on the level of exposure to diesel exhaust but also on the distribution of smoking status and intensity in the population. For example, estimates of lung cancer risk in a population of never smokers with diesel exposures similar to those of the miners in this study would be 1.0, 1.47, and 7.30 for individuals with cumulative REC, lagged 15 years, of less than 8 μg/m³-y, 8 to less than 304 μg/m³-y, and 304 μg/m³-y or more, respectively. In contrast, the overall study population, which included 29% never smokers, had lower odds ratios of 1.0, 1.12, and 2.40 for the same tertiles of cumulative REC exposure, lagged 15 years, respectively (data not shown). In fact, the proportion of never smokers in this study population is substantially lower than the 51% reported for the US population of men aged 18 years or older (27), suggesting that diesel-related estimates of lung cancer risk in the US population may be higher than the overall risk estimates reported here because the proportion of never smokers in the US population is higher than in this study cohort.

Our study has several major strengths including its relatively large size, which provided adequate statistical power to detect a statistically significant exposure–response relationship, adequate latent period for the development of lung cancer, detailed exposure assessment that enabled us to evaluate risk based on quantitative historical exposure to REC, subjects with a wide range of diesel exposure and with underground workers experiencing exposure levels considerably higher than that of other occupationally exposed groups in previous studies, a high interview participation rate for both case subjects and control subjects, and the ability to control for confounding from smoking and other lung cancer risk factors. Two main limitations are also apparent. First, the data on smoking and other potential confounders were derived mainly from next-of-kin interviews. Although a comparison of confounder data derived directly from living and from next of kin for deceased control subjects revealed comparability of responses, we cannot completely rule out the possibility of residual confounding. Second, as in most epidemiological studies of cancer that rely on retrospective exposure assessment, estimates of diesel exposure in this study undoubtedly had some imprecision despite considerable effort to minimize misclassification. This imprecision is likely to result in nondifferential misclassification of exposure, which would tend to bias the estimates of risk toward the null (28). Thus, the true estimates of lung cancer risk associated with diesel exhaust may, in fact, be higher than those reported here.

In sum, our results provide further evidence supporting a causal effect of diesel exhaust exposure on lung cancer mortality in humans. We observed a statistically significant exposure–response relationship after we adjusted for possible confounding from smoking and other established and hypothesized lung cancer risk factors. The exposure–response curve showed a steep increase in risk with increasing exposure at low-to-moderate levels followed by a plateauing or perhaps a decline in risk among heavily exposed subjects.

Our findings are important not only for miners but also for the 1.4 million American workers and the 3 million European workers exposed to diesel exhaust (29), and for urban populations worldwide. Some of the higher average elemental carbon levels reported in cities include Los Angeles (4.0 μg/m³) (30), the Bronx (a borough in New York City) (6.6 μg/m³) (31), nine urban sites in China (8.3 μg/m³) (32), Mexico City (5.8 μg/m³) (33), and Estarreja, Portugal (11.8 μg/m³) (34). Environmental exposure to average elemental carbon levels in the 2-6 μg/m³ range over a lifetime as would be experienced in highly polluted cities approximates cumulative exposures experienced by underground miners with low exposures in our study. Because such workers had at least a 50% increased lung cancer risk, our results suggest that the high air concentrations of elemental carbon reported in some urban areas may confer increased risk of lung cancer. Thus, if the diesel exhaust/lung cancer relation is causal, the public health burden of the carcinogenicity of inhaled diesel exhaust in workers and in populations of urban areas with high levels of diesel exposure may be substantial.

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Traffic, Susceptibility, and Childhood Asthma

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Results from studies of traffic and childhood asthma have been inconsistent, but there has been little systematic evaluation of susceptible subgroups. In this study, we examined the relationship of local traffic-related exposure and asthma and wheeze in southern California school children (5–7 years of age). Lifetime history of doctor-diagnosed asthma and prevalent asthma and wheeze were evaluated by questionnaire. Parental history of asthma and child's history of allergic symptoms, sex, and early-life exposure (residence at the same home since 2 years of age) were examined as susceptibility factors. Residential exposure was assessed by proximity to a major road and by modeling exposure to local traffic-related pollutants. Residence within 75 m of a major road was associated with an increased risk of lifetime asthma [odds ratio (OR) = 1.29; 95% confidence interval (CI), 1.01–1.86], prevalent asthma (OR = 1.50; 95% CI, 1.16–1.95), and wheeze (OR = 1.40; 95% CI, 1.09–1.78). Susceptibility increased in long-term residents with no parental history of asthma for lifetime asthma (OR = 1.85; 95% CI, 1.11–3.09), prevalent asthma (OR = 2.46; 95% CI, 0.48–4.09), and recent wheeze (OR = 2.74; 95% CI, 1.71–4.39). The higher risk of asthma near a major road decreased to background rates at 150–200 m from the road. In children with a parental history of asthma and in children moving to the residence after 2 years of age, there was no increased risk associated with exposure. Effect of residential proximity to roadways was also larger in girls. A similar pattern of effects was observed with traffic-modeled exposure. These results indicate that residence near a major road is associated with asthma. The reason for larger effects in those with no parental history of asthma merits further investigation. *Key words:* **air pollution, asthma, child, epidemiology, traffic.** *Environ Health Perspect* **114:766–772 (2006). doi:10.1289/ehp.8594 available via** *http://dx.doi.org/* **[Online 16 February 2006]**

Childhood asthma prevalence and incidence have been associated with local variation in traffic patterns within communities in many (Brauer et al. 2002; Gauderman et al. 2005; Nicolai et al. 2003; van Vliet et al. 1997; Venn et al. 2000; Zmirou et al. 2004) but not all (English et al. 1999; Waldron et al. 1995; Wjst et al. 1993) studies that have examined the impact of local traffic or traffic-related air pollutants near children's homes. However, many studies did not evaluate exposure at early age, which may be an important determinant of risk from traffic-related pollution (Zmirou et al. 2004) and which might vary depending on residential stability of study participants. The duration of residence at the same home might also be expected to increase any risk of asthma associated with traffic-related exposure. Other characteristics that might make children more susceptible to this exposure include parental history of asthma and childhood allergy, which are strong risk factors for asthma (London et al. 2001; Peden 2000). A recent study found larger associations of traffic with asthma in children without a parent with asthma (Gordian et al. 2005), and we have previously found that children with incident asthma associated with ozone were less likely to have a parental history of asthma than were other children with asthma (McConnell et al. 2002). Susceptibility to second-hand tobacco smoke exposure, another environmental combustion product, and traffic-related pollutants has been found to vary by atopy in some studies that have examined this relationship (Janssen et al. 2003; Kershaw 1987; Palmieri et al. 1990; Strachan and Cook 1998; Strachan et al. 1996a, 1996b; Zmirou et al. 2004). Some evidence also suggests that girls may be more susceptible than boys to traffic-related exposure (Oosterlee et al. 1996; Pershagen et al. 1995; Shima et al. 2003; van Vliet et al. 1997; Venn et al. 2001).

Concentrations of pollutants in fresh vehicular exhaust are high near roadways but decline markedly within 150–300 m (Gilbert et al. 2005; Zhu et al. 2002). Accurate assessment of this large but very local variation in exposure may be important to identify health hazards. One promising approach has been to estimate residential distance to a major roadway. This can be done with relatively little error in measurement, using geographic information systems and accurately located roadways. Some studies have found increased asthma prevalence in children living within 100 m of a major road, and there is evidence that the risk increases dramatically within 75 m (van Vliet et al. 1997; Venn et al. 2001).

In this population-based study, we examined characteristics that might increase childhood susceptibility to the effects of traffic-related air pollution in a new cohort in the southern California Children's Health Study, an ongoing longitudinal evaluation of air pollution and respiratory health (Künzli et al. 2003). We evaluated whether parental history of asthma and child age at exposure, symptoms of allergy, and sex influenced susceptibility to the risk of childhood asthma and wheeze associated with exposure to traffic.

Materials and Methods

Population. A new cohort was recruited in 2003 from schools in 13 southern California communities (shown in Figure 1). Nine communities were the same as in the original Children's Health Study cohorts, and four were new. All students present in 2003 in all kindergarten and first grade classrooms (5–7 years of age) in participating schools were given a questionnaire and informed consent to take home for parents to complete. Informed consent, approved for this study by the University of Southern California Institutional Review Board, was obtained, and questionnaires were completed and returned for 5,341 (65%) of 8,193 eligible children.

Assessment of exposure to traffic-related pollutants. We estimated distance of each participant's residence to the nearest major road, including freeways, other highways, and arterial roads. Participant residence addresses were standardized, and their locations were geocoded to 13 m perpendicular to the side of the adjacent road, using the Tele Atlas Multinet road network data (Tele Atlas Inc.,

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Menlo Park, CA). Distance to the nearest major road was estimated using ArcGIS software (version 8.3; Environmental Systems Research Institute Inc., Redlands, CA). Each direction of travel was represented as a separate roadway, and the shortest distance was estimated from the residence to the middle of the nearest side of the freeway or major road. We included in the analysis only children with addresses that could be accurately geocoded. Specifically, only residential addresses for which the Tele Atlas geocoding software assigned its highest-quality match code were included. These addresses are located on the correct side of the street with their relative position between cross-streets determined by linear interpolation of residence number between the nearest intersections.

Residential distance to a major road was categorized as < 75 m, 75–150 m, > 150–300 m, and > 300 m, based on results of previous studies showing markedly increased exposure and risk of asthma within 75 m of large roadways, which decreased to background levels by 150–300 m (Gilbert et al. 2005; van Vliet et al. 1997; Venn et al. 2001; Zhu et al. 2002). We also estimated residential exposure to fresh traffic-modeled pollutants from local freeway and nonfreeway sources, accounting for traffic volume, wind speed, and direction in each community, using a line source dispersion model, as described in the accompanying online supplemental material (http://www.ehponline.org/members/2006/8594/suppl.pdf).

Health outcomes and other questionnaire information. We classified lifetime asthma based on a questionnaire response to the question "Has a doctor ever diagnosed this child as having asthma?" Current wheeze was defined to include children with any wheezing in the previous 12 months [International Study of Asthma and Allergies in Children (ISAAC) Steering Committee 1998]. Prevalent asthma was defined as the reported use of controller medications for asthma (inhaled corticosteroids, leukotriene inhibitors, cromolyn sodium, or long-acting beta agonists) in the previous year or lifetime asthma with any wheeze in the previous year. In addition, children without a physician's diagnosis who had severe wheeze in the previous 12 months were included as prevalent asthmatics to identify asthma undiagnosed because of poor access to medical care. Severe wheeze included four or more attacks of wheeze, one or more nights per week of wheeze, or wheeze with shortness of breath so severe as to interfere with speech (ISAAC Steering Committee 1998).

We collected personal and family covariates and housing characteristics by questionnaire, including child's race and date of birth and the language in which the questionnaire was completed (Spanish or English). Potentially susceptible groups were identified based on child's sex, allergic characteristics defined as a history of hay fever or a problem with sneezing or runny or blocked nose when the child did not have a cold, parental history of asthma, and residence (exposure) in the current home since 2 years of age or earlier. Information on potentially confounding exposures or characteristics included maternal smoking while pregnant with the child, current second-hand tobacco smoke exposure, family income and responding parent's education, current coverage of the child by a health insurance plan, and housing characteristics, which included pets inside the home (dog, cat, bird, other furry or hairy pets, or other pets), cockroaches, rats or mice, carpeting, water damage or mold or mildew in the home since the child lived there, use of an air conditioner, second-hand tobacco smoke, and a combustion source for nitrogen dioxide in the home (a gas oven or stove or heating unit with a pilot light).

Statistical analysis. The odds ratio (OR) for each distance category was estimated with residences further than 300 m as the reference group, using logistic regression. All models were adjusted for the child's age, sex, race, community, and language of questionnaire completion. To assess the effect of long-term and early-life exposure, some analyses were stratified into children living since 2 years of age or younger at the same residence and those moving to the current residence at a later age. Confounding was evaluated by assessing whether the coefficient of the log OR for exposure changed by > 10% after adding an additional covariate to this basic model. We assessed effect modification by parental history of asthma and the child's history of allergic symptoms and sex by modeling the interaction of the potential effect modifier with exposure category (or with traffic-modeled exposure, as described in the online supplemental material) and by examining the effects of exposure by strata.

We also fitted logistic additive models (Hastie and Tibshirani 1990) to assess the functional relationship between childhood asthma and proximity to major roads. These models used the smoothing spline with 3 degrees of freedom for the continuous distance from major road and used the same adjustment variables as in the linear logistic models described above.

Significance was defined as two-sided $p < 0.05$ for all analyses. The logistic additive models were fitted using the S-plus programming language (Venables et al. 2002). All other analyses were performed using the Statistical Analysis System (SAS version 9.0; SAS Institute Inc., Cary, NC).

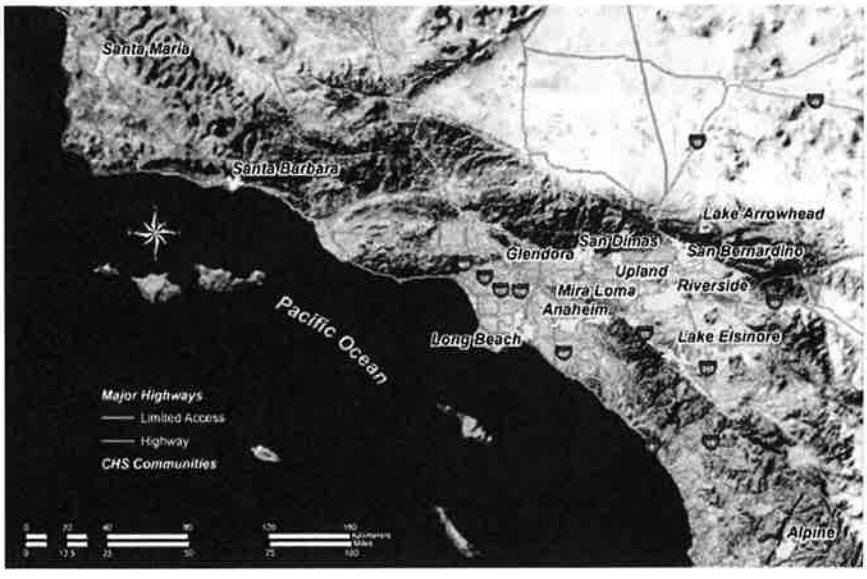

Figure 1. Location of study communities.

Results

Of the 5,341 children completing a questionnaire and informed consent, 4,762 had an address that could be accurately matched and geocoded. Among these, there were 650 reports of ever physician-diagnosed asthma (14%); 577 cases of prevalent asthma (13%) based on current severe symptoms, use of controller medications, or lifetime asthma with current wheeze; and 682 children with current wheeze during the previous year (15%). Although there was some overlap of these phenotypes, 38% of children with lifetime asthma had no current wheeze, 16% with prevalent asthma had no current wheeze (based primarily on use of controller medications in prevalent asthma), and 17% of prevalent asthma

cases had no lifetime reported doctor diagnosis of asthma. The mean (± SD) age was 6.5 ± 0.68 years. The frequency of other characteristics of children, parents, and households is shown in Table 1. Most children were Hispanic, and almost one-quarter of parents completed a questionnaire in Spanish. Eighteen percent of parents reported that annual household income was < $15,000, and 22% had less than a high school education. Forty-two percent of children had lived at the same address since 2 years of age or younger.

The mean (± SD) distance from the child's residence to a major road was 418 ± 519 m (median, 254 m; range, 0.02–7,516 m). (Error in precisely locating homes and roadways accounted for distances less than the 13-m offset from the street used in geocoding residences.) Most residences (56.6%) were within 300 m of a major road: 25.2% were between 150 and 300 m, 16.4% between 75 and 150 m, and 15% within 75 m.

Table 1. Demographic characteristics and potential confounders or susceptibility factors.

Characteristic	No. (%)[a]
Child	
Sex	
Male	2,425 (51)
Female	2,295 (49)
Race	
North American Indian	44 (0.93)
Asian	170 (3.6)
Black	197 (4.2)
Hispanic white	2,617 (55)
Non-Hispanic white	1,682 (35)
Other	32 (0.67)
Health insurance	3,985 (88)
Long-term residence	1,856 (42)
Allergy	1,834 (44)
In utero tobacco smoke	360 (7.9)
Parent	
Spanish questionnaire	1,091 (23)
Family income	
< $7,500	279 (6.9)
$7,500–14,999	456 (11)
$15,000–29,999	692 (17)
$30,000–49,999	709 (18)
$50,000–74,999	726 (18)
$75,000–99,999	535 (13)
≥ $100,000	639 (16)
Parental education	
< 12th grade	982 (22)
Grade 12	880 (20)
Some post-high school	1,681 (38)
Four years of college	512 (11)
Some postgraduate	417 (9.3)
Parental asthma	965 (23)
Home	
Any pet	2,479 (54)
Dog	1,337 (29)
Cat	841 (18)
Bird	462 (10)
Cockroach	487 (11)
Mice	366 (8.1)
Rats	189 (4.2)
NO_2 source	3,358 (72)
Air conditioner	2,763 (60)
Carpeting	4,230 (92)
Water damage	653 (14)
Mold or mildew	1,068 (25)
Second-hand smoke	794 (18)

[a]Total (% of total) with each characteristic; denominator varies due to missing values or "don't know" responses.

The risk of asthma-related outcomes was associated with residential distance to a major road (Table 2). Compared with those living at least 300 m from a major road, there were increased risks for all three outcomes among children within 75 m. For both prevalent asthma and current wheeze, there was increasing risk with decreasing residential distance to the roadway. Among long-term residents (living since 2 years of age at the same home), risk was increased only among those living within 75 m of a major road, and the ORs were slightly larger than the corresponding ORs in the entire population. Confounding by housing characteristics or other covariates from Table 1 was assessed among long-term residents, and the effect of living within 75 m of a major road was not substantially changed.

We examined interactions of exposure with the susceptibility factors in the sample restricted to long-term residents, because exposure in this group was more likely to have been accurately assigned for the period during which asthma developed than for children moving later. Parental asthma modified the effect of living within 75 m of a major road (Table 3). There were almost 2-fold (lifetime asthma) to almost 3-fold increased risks (current wheeze) associated with this exposure, but only among those children without a parental history of asthma. The interaction of parental history with residential proximity within 75 m was significant for prevalent asthma (1 degree of freedom, Wald chi-square 4.39; $p = 0.04$) and for current wheeze ($p = 0.01$), but not for lifetime asthma.

Among long-term residents who had no allergic symptoms, greater than 2-fold increased risks of all three outcomes were associated with living in a residence within 75 m of a major road (Table 4). However, there were no significant interactions of allergy with this exposure for any of the three outcomes.

Among boys, there was little evidence of increased risk associated with residential distance to a major road (Table 5). Among girls, strong associations with living within 75 m of a major road were observed for all three outcomes, and the difference between boys and girls was significant for lifetime asthma (1 degree of freedom interaction, $p = 0.02$).

Table 2. Association of asthma and wheeze with distance to a major road [OR (95% CI)].[a]

Major road distance (m)	No.[b]	Lifetime asthma	Prevalent asthma	Current wheeze
All participants				
> 300	2,058	1.00	1.00	1.00
150–300	1,193	0.92 (0.73–1.15)	1.04 (0.82–1.33)	1.02 (0.82–1.27)
75–150	778	1.06 (0.82–1.36)	1.33 (1.02–1.72)*	1.30 (1.02–1.66)*
< 75	713	1.29 (1.01–1.66)*	1.50 (1.16–1.95)**	1.40 (1.09–1.78)**
Long-term residents				
> 300	813	1.00	1.00	1.00
150–300	483	0.86 (0.59–1.24)	0.83 (0.56–1.21)	0.97 (0.69–1.38)
75–150	294	1.03 (0.68–1.56)	1.09 (0.71–1.66)	1.09 (0.73–1.62)
< 75	266	1.46 (0.98–2.17)	1.64 (1.10–2.44)*	1.67 (1.14–2.43)**

[a]Adjusted for age, sex, language of questionnaire, community, and race. [b]Total exposed in each category of distance to a major road. *$p < 0.05$; **$p < 0.01$.

Table 3. Association of asthma and wheeze with distance to a major road among long-term residents, by parental history of asthma [OR (95% CI)].[a]

Major road distance	No parental asthma ($n = 1,330$)	Parental asthma ($n = 380$)
Lifetime asthma[b]		
> 300 m	1.00	1.00
150–300 m	1.06 (0.65–1.71)	0.62 (0.30–1.25)
75–150 m	1.13 (0.64–1.97)	0.75 (0.34–1.63)
< 75 m	1.85 (1.11–3.09)*	1.03 (0.47–2.24)
Prevalent asthma		
> 300 m	1.00	1.00
150–300 m	0.94 (0.57–1.58)	0.67 (0.33–1.37)
75–150 m	1.21 (0.69–2.14)	0.80 (0.37–1.74)
< 75 m	2.46 (1.48–4.09)**	0.79 (0.34–1.82)
Current wheeze		
> 300 m	1.00	1.00
150–300 m	1.02 (0.64–1.64)	0.96 (0.51–1.80)
75–150 m	1.37 (0.81–2.31)	0.88 (0.42–1.83)
< 75 m	2.74 (1.71–4.39)**	0.87 (0.40–1.90)

[a]Adjusted for age, sex, language of questionnaire, community, and race. [b]Participants from Lake Arrowhead were excluded from the model for stratum with no parental asthma, because otherwise the model failed to converge. *$p < 0.05$; **$p < 0.01$.

Among children with no family history of asthma, we examined further the relationship of asthma and distance to a major road within 500 m of the home, using smoothed models. Among long-term residents, an increasing rate of prevalent asthma was observed with residential proximity to the nearest major road, and the risk decreased to background levels at 150–200 m (Figure 2). This trend was observed only among children living at the same address since 2 years of age. Children moving to the current residence after 2 years of age showed no effect of proximity to a major road. A similar pattern of effects was observed for lifetime asthma and wheeze (data not shown).

The effects of pollutants in fresh traffic exhaust modeled from traffic volume, distance, and meteorology were generally consistent with those observed for proximity to a major road (see online supplemental material). There were significant associations of nonfreeway (but not of freeway or total) traffic-modeled exposure with prevalent asthma and current wheeze, and these effects were stronger in long-term residents (Table S-2 in the online supplemental material). The stratum-specific pattern of traffic-modeled effects was also stronger in those with no parental history and with no allergic symptoms and among girls (Table S-3 in the online supplemental material).

Discussion

Asthma and wheeze were strongly associated with residential proximity to a major road. These associations were strongest among children with no parental history of asthma who had lived at the same address since early in life. In this group, the highest risk occurred adjacent to the major road, and risk decreased to background rates at 150–200 m from the road. Larger risks of asthma associated with long-term residence within 75 m of a major road were observed among girls than among boys.

If traffic-related pollutants were responsible for the observed associations with asthma, the increased risk among the longer-term residents might be expected because they had a larger cumulative exposure to the pollutant indicators used in this analysis. However, the absence of any effect of a major road among children moving to their residence after 2 years of age (Figure 2) may indicate vulnerability during the prenatal period or infancy. Although the study design did not allow us to distinguish between these two possibilities, there is evidence that other early-life exposures may increase the risk of asthma (Martinez 1999). Recent case–control and cohort studies have found an increased risk of asthma with early-life exposure to local residential traffic-related pollutants (Brauer et al. 2002; Zmirou et al. 2004). In addition, several recent studies suggest that early-life (especially *in utero*) exposure to tobacco smoke, which like fresh vehicular exhaust is a complex mixture of air pollutants, is more strongly associated with increased risk of subsequent asthma than is exposure later in childhood (Gilliland et al. 2001, 2002). The larger effect of proximity to a major roadway among girls in our study also is consistent with previous reports (Oosterlee et al. 1996; Pershagen et al. 1995; Shima et al. 2003; van Vliet et al. 1997; Venn et al. 2001).

We previously found that children with an increased risk of incident asthma associated with exercise in high-ozone environments were less likely to have a parental history of

Table 4. Association of asthma and wheeze with distance to a major road among long term residents, by child's history of allergy [OR (95% CI)].[a]

Major road distance (m)	No allergic symptoms (n = 942)	Allergic symptoms (n = 723)
Lifetime asthma[b]		
> 300	1.00	1.00
150–300	0.92 (0.43–1.97)	0.87 (0.53–1.41)
75–150	1.04 (0.41–2.62)	0.96 (0.57–1.61)
< 75	2.27 (1.04–4.94)*	1.31 (0.76–2.25)
Prevalent asthma		
> 300	1.00	1.00
150–300	0.98 (0.42–2.26)	0.77 (0.46–1.27)
75–150	0.81 (0.25–2.55)	1.01 (0.60–1.69)
< 75	2.52 (1.07–5.93)*	1.29 (0.76–2.21)
Current wheeze		
> 300	1.00	1.00
150–300	1.50 (0.72–3.12)	0.80 (0.50–1.28)
75–150	0.72 (0.23–2.25)	1.03 (0.63–1.68)
< 75	2.58 (1.14–5.86)*	1.25 (0.75–2.07)

[a]Adjusted for age, sex, language of questionnaire, community, and race. [b]Participants from Lake Arrowhead were excluded from models for stratum without allergy for prevalent and lifetime asthma, because otherwise the models failed to converge. *$p < 0.05$.

Table 5. Association of asthma and wheeze with distance to a major road among long term residents, by child's sex [OR (95% CI)].[a]

Major road distance (m)	Boys (n = 945)	Girls (n = 901)
Lifetime asthma[b]		
> 300	1.00	1.00
150–300	0.87 (0.54–1.40)	0.88 (0.48–1.61)
75–150	1.15 (0.69–1.92)	0.68 (0.31–1.48)
< 75	0.94 (0.54–1.64)	2.51 (1.39–4.54)**
Prevalent asthma		
> 300	1.00	1.00
150–300	0.77 (0.46–1.30)	0.90 (0.50–1.61)
75–150	1.37 (0.82–2.31)	0.53 (0.23–1.24)
< 75	1.31 (0.75–2.29)	2.13 (1.18–3.85)*
Current wheeze		
> 300	1.00	1.00
150–300	0.96 (0.60–1.53)	0.99 (0.58–1.69)
75–150	1.27 (0.77–2.10)	0.72 (0.35–1.46)
< 75	1.41 (0.84–2.37)	1.95 (1.11–3.41)*

[a]Adjusted for age, language of questionnaire, community, and race. [b]Participants from Lake Arrowhead were excluded from model for stratum with girls for lifetime asthma, because otherwise the model failed to converge. *$p < 0.05$; **$p < 0.01$.

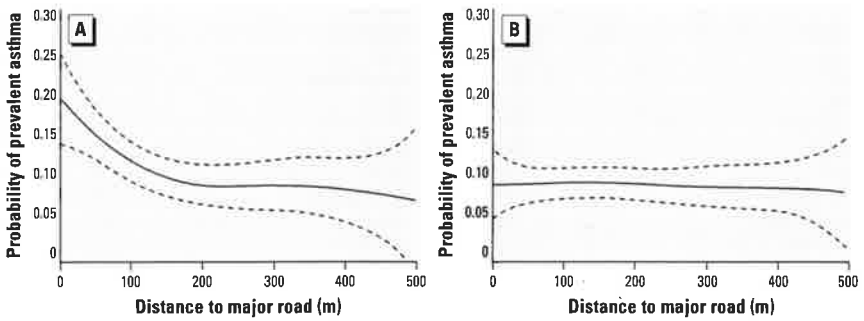

Figure 2. Prevalence of asthma by distance of residence to a major road within 500 m, among long-term (*A*) and short-term (*B*) residents with no family history of asthma. Dotted lines indicate 95% confidence interval.

asthma (McConnell et al. 2002), and another recent study found that the risk of traffic-associated prevalent asthma was larger in children without parental history (Gordian et al. 2005). However, both family history of asthma and child allergy are strong risk factors for asthma independent of exposure to air pollution (London et al. 2001; Peden 2000). In our study, among long-term residents living > 300 m from a major road, parental history was associated with a 3.6-fold increased risk of prevalent asthma [95% confidence interval (CI), 2.3–5.8] and child symptoms of allergy with a 6.4-fold increased risk (95% CI, 4.5–9.1). Therefore, one possible explanation for the larger effects of traffic exposure in children without these strong risk factors is that other risks, for example, dietary factors, indoor allergens, or other environmental exposures, produced asthma in the high-risk group, regardless of traffic-related exposures. It is possible that, among those with parental asthma or allergic symptoms, there was no additional risk of childhood asthma associated with traffic or that any small additional effect of traffic was undetectable in the high background rate of asthma in these children.

Parental history of asthma is an indication of genetic susceptibility, so the absence of risk among those with parental history may also indicate that asthma caused by pollutants in fresh traffic exhaust is less likely to be inherited, or at least is not mediated through the same genetic pathways that account for asthma in the parents of these children. Nonallergic asthma is one possible alternative pathway, which may be consistent with the stronger observed effect of traffic among children without hay fever or other allergic symptoms. Like parental history of asthma, allergic symptoms are associated with atopic asthma (Peden 2000). Atopy is characterized by a positive skin test or immunoglobulin E–specific response to environmental allergens. Recent studies indicate that nonallergic asthma (without airway eosinophilia or atopy) may account for as much as half of all asthma (Beasley et al. 2001; Douwes et al. 2002; Pearce et al. 1999), and it has been suggested that risk factors for this asthma phenotype, including particulate air pollution, may differ from those for allergic asthma (Douwes et al. 2002). Some studies of the risk of asthma and wheeze due to second-hand smoke, another mix of oxidant pollutants, have shown stronger effects among children without atopy or atopic symptoms (Kershaw 1987; Palmieri et al. 1990; Strachan and Cook 1998; Strachan et al. 1996a, 1996b). In addition, drug-induced and occupational asthma commonly occur in the absence of atopy, and many of the exposures responsible for "irritant-induced asthma" in the workplace are also present in the general population (Gautrin et al. 2003; Kitani et al. 1993). However, in other studies, stronger associations of asthma and wheeze with traffic-related pollutants were found among atopic children (Janssen et al. 2003; Zmirou et al. 2004) and with second-hand tobacco smoke exposure among children with an atopic parent (Jaakkola et al. 2001). In addition, laboratory evidence suggests that exposure to oxidant air pollution promotes the effect of allergens on asthma severity and on the pathogenesis of asthma (Jenkins et al. 1999; Kehrl et al. 1999; Li et al. 2003; Schelegle et al. 2003). Based on these studies, an effect of traffic-related pollutants might have been expected to be stronger among children with allergy. Further investigation is warranted to identify the reason for the apparent susceptibility of children without allergy and parental history of asthma in our study. Better phenotypic characterization of atopy both in the study children and in their parents and of allergen exposure in children would be useful to interpret the relationship of these characteristics to traffic and asthma.

In a previous cohort in the Children's Health Study, we observed strong associations of lifetime asthma with residential ambient NO_2, an indicator of variability within communities of traffic-related pollutants, which was measured at a sample of homes (Gauderman et al. 2005). Measured NO_2 was moderately correlated with total traffic-modeled pollution ($R = 0.59$). Strong associations also were observed with residential distance to a freeway and with traffic-modeled exposure from freeways (but not from non-freeway traffic–modeled pollution). We have now extended these observations to a larger population and to residential distance to other major roadways. The association of asthma in our new cohort with non-freeway traffic–modeled exposure, but not with freeway-modeled exposure, may reflect differences in the distribution of freeways and major roads around homes in the different cohorts. The association of asthma with non-freeway traffic–modeled exposure is consistent with the observed association with distance to a major road, because there were few children within 75 m of a freeway in our study. Residential distance to a major roadway also is computationally easier to estimate from data that are more readily available than the meteorologic and traffic volume data required to model exposure. An increased risk associated with proximity to a major roadway also is more easily explained to policy makers and to the general public than is the risk associated with traffic-modeled exposure.

Our results are also consistent with several European studies that found increased risks of childhood asthma with increased traffic counts in close proximity to the home (Morris et al. 2000; Nicolai et al. 2003; van Vliet et al. 1997; Venn et al. 2001; Zmirou et al. 2004). One large British study that focused on traffic within 150 m of children's homes found a gradient in risk that increased markedly with decreasing residential distance to a main road (Venn et al. 2001). There have been few other studies of traffic and childhood asthma in the United States. A recent study in northern California found an association between measured traffic-related pollutants at schools and childhood asthma (Kim et al. 2004). However, another large study in southern California based on records of children covered by Medicaid (public insurance for low-income persons) found no association between asthma prevalence and traffic counts within 168 m of the home, although an association with asthma medication was observed (English et al. 1999). Some of the inconsistencies in the literature could perhaps be explained by the failure of many studies to account for the pattern of effect modification by parental history of asthma and by age and duration of residential exposure to traffic-related pollutants that vary markedly at different locations. The larger effects of traffic in girls has been observed in previous studies of traffic and asthma and related symptoms, but the reason for the apparent susceptibility of girls is not known (Oosterlee et al. 1996; Pershagen et al. 1995; Shima et al. 2003; van Vliet et al. 1997; Venn et al. 2001).

A causal relationship between asthma and traffic-related exposures is biologically plausible, because ambient particulate matter and other oxidant pollutants have been shown to elicit responses relevant to the pathogenesis of asthma (Li et al. 2003). In addition, studies in southern California and elsewhere have shown that the largest gradients in traffic-related pollutants occur within the 150–200 m from roadways over which we saw decreasing risk of asthma (Gilbert et al. 2003; Zhu et al. 2002). In studies in which NO_2 and other markers of traffic-related exposure have been measured in close proximity to major roadways, variability has usually been best explained by traffic volume within 300 m (Briggs et al. 2000; Gilbert et al. 2003; Ross et al. 2005), although weaker correlations have also been observed over longer distances from the highest volume traffic corridors (Gauderman et al. 2005; Gilbert et al. 2003, 2005; Ross et al. 2005).

We considered bias as an explanation for our results. Parents with asthma who were susceptible to environmental triggers might have selected homes away from major roads, perhaps even before the children were born. If the children of these parents had high rates of asthma, this might have explained the observed lack of effect of a major road in families with parental asthma. There is some

evidence that parents may intervene to reduce household exposure to indoor allergens, another perceived risk for asthma and asthma severity (Almqvist et al. 2003; van Strien et al. 2002). However, this bias is unlikely to explain our results, because we examined and found no significant differences in rates of parental history of asthma by exposure to a major road (data not shown). Selection bias related to factors influencing participation could not be evaluated, because characteristics of nonparticipants are not known. However, there were some modest differences between participants whose addresses could be geocoded, who were of higher socioeconomic status than were participants whose addresses could not be geocoded. For example, of those with family income < $7,500, 85% could be geocoded, compared with 93% of those with ≥ $100,000. Of those without insurance, 85% could be geocoded, compared with 90% of those with insurance. The differences between those whose homes could and could not be geocoded were heavily influenced by 299 subjects (of 579 total that could not be geocoded) who completed a questionnaire but did not provide an address. However, none of the three asthma outcomes was associated with absence of a home geocode, and the associations of asthma with traffic were robust to our adjustment for socioeconomic status. It has also been suggested that traffic-related noise might cause asthma through a pathway mediated by stress (Ising and Ising 2002). However, to date there is little evidence to support this hypothesis. Other potential confounders, including sociodemographic factors, second-hand or *in utero* tobacco smoke exposure, or housing characteristics that are commonly associated with asthma also did not explain our results. A final possible limitation to the interpretation of these results is the assessment of asthma by questionnaire. However, self-report of physician-diagnosed asthma has been reported to accurately reflect what physicians have told the patient, at least in adults, and validity of questionnaires as reported by repeatability of response is good (Ehrlich et al. 1995). For these reasons, self-report of physician-diagnosed asthma has been widely used in epidemiologic studies and has been recommended as the preferred outcome assessment for use in large population-based studies, because a more precise diagnosis is not available (Burr 1992). In addition, the consistency of associations with lifetime asthma, prevalent asthma based on a combination of symptoms reporting and doctor diagnosis, and recent wheezing suggests that diagnostic bias is unlikely to have explained the observed results.

We conclude that living in a residence with more nearby traffic increases the risk of childhood asthma. Children with no parental history of asthma who had long-term residential exposure (or early-life exposure) constituted a susceptible population, and the risk was larger for girls than for boys. Because a substantial number of southern California children live near a major road, this exposure is potentially an important public health problem that could be remediable by transportation and residential development policy and by more effective control of vehicular emissions. Among those long-term residents with no parental history of asthma who lived within 75 m of a major road, 59% of asthma was attributable to residential proximity to the road. Further investigation is warranted to understand why the absence of parental asthma history increased susceptibility to traffic-related exposure.

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Environmental Public Health Tracking of Childhood Asthma Using California Health Interview Survey, Traffic, and Outdoor Air Pollution Data

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Background: Despite extensive evidence that air pollution affects childhood asthma, state-level and national-level tracking of asthma outcomes in relation to air pollution is limited.

Objectives: Our goals were to evaluate the feasibility of linking the 2001 California Health Interview Survey (CHIS), air monitoring, and traffic data; estimate associations between traffic density (TD) or outdoor air pollutant concentrations and childhood asthma morbidity; and evaluate the usefulness of such databases, linkages, and analyses to Environmental Public Health Tracking (EPHT).

Methods: We estimated TD within 500 feet of residential cross-streets of respondents and annual average pollutant concentrations based on monitoring station measurements. We used logistic regression to examine associations with reported asthma symptoms and emergency department (ED) visits/hospitalizations.

Results: Assignment of TD and air pollution exposures for cross-streets was successful for 82% of children with asthma in Los Angeles and San Diego, California, Counties. Children with asthma living in high ozone areas and areas with high concentrations of particulate matter < 10 μm in aerodynamic diameter experienced symptoms more frequently, and those living close to heavy traffic reported more ED visits/hospitalizations. The advantages of the CHIS for asthma EPHT include a large and representative sample, biennial data collection, and ascertainment of important sociodemographic and residential address information. Disadvantages are its cross-sectional design, reliance on parental reports of diagnoses and symptoms, and lack of information on some potential confounders.

Conclusions: Despite limitations, the CHIS provides a useful framework for examining air pollution and childhood asthma morbidity in support of EPHT, especially because later surveys address some noted gaps. We plan to employ CHIS 2003 and 2005 data and novel exposure assessment methods to re-examine the questions raised here.

Key words: air pollution, asthma, children, environmental public health tracking, epidemiology, geographic information system, traffic. *Environ Health Perspect* 116:1254–1260 (2008). doi:10.1289/ehp.10945 available via *http://dx.doi.org/* [Online 9 April 2008]

Asthma is one of the most prevalent chronic conditions affecting children in the United States today. According to National Health Interview Survey data for 2005, > 9 million children < 18 years of age (13%) had ever been diagnosed with asthma (Bloom et al. 2006). The California Health Interview Survey (CHIS) for 2005 indicated a lifetime prevalence of 16% for this age group. Asthma is a multifactorial disease in which genetic susceptibilities influence responses to environmental exposures (Gilmour et al. 2006). Exposure to outdoor air pollution has been widely studied as a potential risk factor for asthma, and it is generally well established that short-term increases can exacerbate respiratory symptoms in children with asthma (Gilmour et al. 2006; Thurston and Bates 2003; Trasande and Thurston 2005). Ozone, particulate matter < 10 and < 2.5 μm in aerodynamic diameter (PM_{10} and $PM_{2.5}$), and nitrogen dioxide are the pollutants linked most consistently with exacerbation of asthma symptoms. Although long-term exposures to O_3, PM_{10}, and NO_2 have been associated with chronic respiratory impairments such as reduced lung function and growth, bronchitis, and chronic cough, evidence for the impact of air pollution on asthma incidence is less conclusive (Gilmour et al. 2006; McConnell et al. 2002; Trasande and Thurston 2005). Recently, focus has turned to respiratory effects caused by exposure to specific motor vehicle exhaust components such as polycyclic aromatic hydrocarbons adsorbed to particles from diesel engines and ultrafine particles (< 0.1 μm in aerodynamic diameter), which are able to penetrate cellular targets in the lung and enter systemic circulation (Künzli et al. 2003; Li et al. 2002, 2003; Pandya et al. 2002). Various measures of traffic exhaust exposure have been associated with adverse respiratory outcomes, including reduced lung function and growth; asthma hospitalizations; and prevalence of asthma, wheeze, bronchitis, and allergic rhinitis (English et al. 1999; Gauderman et al. 2005, 2007; Kim et al. 2004; McConnell et al. 2006).

Despite the impact of asthma on children's health, there is no comprehensive system of surveillance at the state or national level for this disease. The national Behavioral Risk Factor Surveillance System (BRFSS) [Centers for Disease Control and Prevention (CDC) 2008a] provides only limited data on asthma prevalence and has poor geographic resolution (i.e., the estimates of asthma prevalence are considered valid only at the state level and thus are not useful for assessing trends in relation to environmental exposures). The National Health Interview Survey (NHIS) (CDC 2008c) and the National Health and Nutrition Examination Survey (CDC 2008b) use a sampling design to represent the population of the entire United States and provide data that cannot be used even at the state level for asthma prevalence estimation; neither of these national surveys yield detailed data on asthma symptoms. Asthma hospitalization data are available from every state, but such data represent only a small fraction of the burden of exacerbations of the disease. Because of the growing body of evidence linking outdoor air pollution exposure to both exacerbation and possibly causation of asthma, tracking its occurrence and severity in relation to pollutant exposures is an important public health goal.

A review by the Pew Environmental Health Commission found that existing efforts to gather information on chronic diseases, such

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as asthma, and their potential environmental links are highly fragmented and inadequate for truly understanding where, why, and how often these diseases occur; thus, they issued a call to close this environmental health gap (Environmental Health Tracking Project Team 2000; McGeehin et al. 2004). In response, a new initiative to establish a national environmental public health tracking (EPHT) network led by the CDC was launched in 2002. EPHT is the ongoing collection, integration, analysis, and dissemination of data from environmental hazard monitoring, human exposure tracking, and health effect surveillance (McGeehin et al. 2004). As part of this initiative, the goal of the project described here was to develop a model tracking system that links asthma data from the CHIS with existing information on outdoor air pollution exposures, specifically ambient air monitoring station and traffic data. If successful, such linkage and analysis could provide a way to assess impacts of future air pollution control strategies on reducing asthma symptoms in children in California and provide an ongoing mechanism for EPHT of asthma. Here we present results and lessons learned from this first tracking effort based on CHIS 2001 data and ambient air monitoring and traffic data for Southern California.

Methods

Data sources. **Health data.** Eligible subjects were individuals 0–17 years of age for whom health data were collected during 2000–2001 as part of the CHIS, who resided in Los Angeles or San Diego County during the same period, and who reported a physician diagnosis of asthma at some point in their lives. The CHIS is a two-stage, geographically stratified, random-digit-dialed telephone survey of California households. One adult was interviewed from each selected household. In households with adolescents and/or children (12–17 and 0–11 years of age, respectively), one adolescent and/or child was randomly selected for an interview. Information on the child respondent was collected from an adult who was most knowledgeable about the child. Except for insurance information provided by the interviewed adult, adolescents were directly interviewed after a parent or guardian gave permission. Information on demographic characteristics, health conditions, health-related behaviors, access to health care, and insurance coverage was collected. Questions pertaining to asthma were modified from existing national health surveys (NHIS and BRFSS), with additional assessment of symptom frequency in children with asthma. Respondents were also asked to report the name of their residential street and the nearest cross-street. Detailed descriptions of CHIS 2001 sampling and survey methods are reported elsewhere (Center for Health Policy Research 2002). This research was approved by the University of California Los Angeles Office for the Protection of Research Subjects and informed consent was obtained from all CHIS 2001 participants.

Within households in Los Angeles and San Diego Counties, interviews were completed for 1,391 adolescents and 3,405 children. We selected 612 respondents (12.8% of those interviewed) who reported ever having been diagnosed with asthma by a physician. Respondents who reported a lifetime diagnosis of asthma were asked to report the frequency of asthma symptoms such as coughing, wheezing, shortness of breath, chest tightness, or phlegm during the 12 months preceding the interview date. In addition, respondents were asked whether they had ever visited a hospital emergency department (ED) or had been hospitalized because of asthma during this period.

Exposure data. Exposure to outdoor air pollution was assessed using two sources of existing information: *a*) routine measurement data collected by the California Air Resources Board and South Coast Air Quality Management District at an existing network of air monitors during 1999–2001, and *b*) annual average daily traffic (AADT) data from the California Department of Transportation (Caltrans) for the year 2000. Detailed information on the Caltrans AADT data was previously reported (English et al. 1999; Gunier et al. 2003; Reynolds et al. 2004; Wilhelm and Ritz 2003). Briefly, the AADT represents the annual average number of vehicles per day traveling in both directions along a given road segment based on rotating traffic counts conducted every 3 years. During noncount years, the AADT is estimated using traffic trends for that location. Counts are collected for all highways and most major roads in the state, but counts on smaller, residential roads with low traffic volume are not typically taken.

Linkage. We estimated traffic density (TD) for each subject based on their reported street of residence and nearest cross-street. Specifically, reported residential cross-streets were geocoded using geographic information system (GIS) software and data (ArcView StreetMap 2000 version 1.1; ESRI, Redlands, CA). We then identified each subject's probable home street segment, which had the reported nearest intersection at the center bound on both ends by the adjacent cross-streets (Meng et al. 2007). We drew a 500-foot buffer around the probable home street segment of each subject and identified all roadways within this buffer that had an AADT value based on the year 2000 Caltrans data. The 500-foot criterion is based on environmental measurement data showing that the impact of direct traffic emissions on ambient concentrations becomes insignificant at approximately this distance (Zhou and Levy 2007). However, the buffers used here were > 0.028 mi^2 in area (as would be the case with a 500-foot radius circle around a specific geocoded home location), because the buffers were defined not by a point but by a probable street segment and its length. On average, the buffers were 0.068 mi^2 in area (range, 0.031–0.199 mi^2). There was not much difference in buffer size between urban areas (average, 0.067 mi^2; range, 0.033–0.168 mi^2) and suburban areas (average, 0.069 mi^2; range, 0.031–0.199 mi^2). Most subjects resided in urban areas (71%); 29% were suburban, and none lived in rural areas.

Similar to methods of Gunier et al. (2003) and Reynolds et al. (2004), we estimated the TD value for each subject by first calculating the vehicle miles traveled (VMT) for each attributed road segment within the buffered area; VMT was estimated by multiplying the AADT by the road segment length. We then calculated TD as the sum of the VMT for all road segments in the buffer divided by the area of the buffer, that is,

$$TD = \Sigma(AADT \times L) / A_B, \qquad [1]$$

where TD is traffic density (vehicles × miles/day/mi^2), $AADT$ is the annual average daily traffic count (vehicles/day), L is the length of roadway segment (miles), and A_B is the area of the 500-foot buffer around the probable street segment (square miles). Subjects with no Caltrans-counted streets within their buffers (n = 47) were included in the low-traffic referent category, because we assumed that these individuals had only residential streets with low AADT near their homes.

In addition to TD values, we assigned to each subject the annual average concentrations of O_3, NO_2, $PM_{2.5}$, PM_{10}, and carbon monoxide measured at the nearest monitoring station within 5 miles of the reported residential cross-street intersection (82% of subjects). We used ArcView GIS (ESRI) software to estimate the distance between each respondent's residential cross-street intersection or ZIP code population-weighted centroid (for the 18% of subjects whose reported cross-street could not be geocoded) and the nearest station that measured each pollutant. Subjects residing outside a 5-mile range were excluded from the pollutant analyses. Annual average concentrations were calculated for the 1-year period before the interview date. These averages were based on hourly measurements for the gaseous pollutants (CO, NO_2, and O_3) and 24-hr average measurements for PM_{10} and $PM_{2.5}$ (with most stations recording measurements every 6 and 3 days for these pollutants, respectively). The 5-mile range was chosen to ensure a large enough radius for sufficient sample size balanced against a possible increase in exposure misclassification due to greater residential distances from monitoring stations.

Statistical analyses of exposure–asthma outcome relationships. We evaluated associations between air pollution and TD and asthma morbidity using logistic regression. Specifically, we examined differences in our exposure metrics for *a*) children with asthma reporting daily or weekly symptoms in the previous year versus those reporting less than weekly symptoms, and *b*) children with asthma reporting at least one asthma-related ED visit or hospitalization in the previous year versus children with asthma not reporting such visits. The analyses incorporated sampling weights that adjusted for unequal probabilities of selection into the CHIS sample. We initially grouped TD values into quintile categories according to their distributions in the total population. Because the effect estimates in the three middle quintiles were similar, we collapsed them and thus created three categories roughly based on these quintile distributions: *a*) low TD, defined as ≤ 20,000 daily VMT/mi^2 (approximately ≤ 20th percentile); *b*) medium TD, defined as 20,001–200,000 daily VMT/mi^2 (approximately 21–80th percentile); *c*) high TD defined as > 200,000 daily VMT/mi^2 (approximately the 80th percentile).

Measured air pollutants (O_3, PM_{10}, $PM_{2.5}$, CO, NO_2) were evaluated continuously, using both single and multipollutant models. We evaluated changes in point and 95% confidence interval (CI) estimates when including covariates such as age, sex, race/ethnicity, poverty level, insurance status, delays in receiving care for asthma, asthma medication use, and county in our models (Table 1). Based on a 10% change-in-estimate criterion (Rothman and Greenland 1998), race/ethnicity and poverty level were included in our final models. Some subjects were excluded because of missing data for these covariates. Final sample sizes for each model are reported in the tables.

Table 1. Weighted prevalence of frequent symptoms or ED visits/hospitalizations by demographic characteristics among CHIS children with reported asthma diagnoses, Los Angeles and San Diego Counties.

	Daily or weekly symptoms (Total children with asthma = 597)		ED visit or hospitalization (Total children with asthma = 607)	
Characteristic	Prevalence (%)	No. total population	Prevalence (%)	No. total population
Age (years)				
0–5	4.2	117	19.8	119
6–11	7.1	257	11.8	263
12–17	18.5	223	7.9	225
Sex				
Male	12.6	341	11.2	346
Female	8.6	256	12.9	261
Race/ethnicity				
Latino	9.6	176	16.1	182
Asian/other	13.4	81	9.1	83
African American	16.6	97	15.6	99
White	8.7	243	7.8	243
Household federal poverty level (%)[a]				
< 100	16.6	107	19.8	109
100–299	9.6	221	11.5	225
≥ 300	8.4	269	7.5	273
County				
Los Angeles	11.1	481	13.7	489
San Diego	9.8	116	6.8	118
Insurance status				
Currently uninsured	23.7	45	18.1	45
Uninsured any time in past 12 months	15.8	25	19.2	25
Insured entire past 12 months	9.1	527	10.9	537
Delays in care[b]				
Yes	27.4	24	37.5	24
No	10.2	571	11.1	580
Taking asthma medication				
Yes	17.6	296	20.4	300
No	4.7	300	4.4	306

[a]Percentages were defined using 2001 federal poverty guidelines ($9,044 for one person, $11,559 for a family of two; incomes at ≥ 300% federal poverty level were three times these amounts). [b]Individuals who reported delaying or foregoing any medical care they felt they needed (such as seeing a doctor, a specialist, or other health professional) for asthma were assigned a value of 1 for delays in care.

Table 2. Pearson correlation coefficients for annual average air pollutant concentrations for residents within 5 miles of a monitoring station.

Pollutant	Mean (range)	O_3	PM_{10}	$PM_{2.5}$	NO_2	CO	TD
O_3 (pphm)	2.1 (1.1–4.2)	1					
PM_{10} (μg/m^3)	37.7 (26.2–46.9)	–0.70	1				
$PM_{2.5}$ (μg/m^3)	19.3 (10.6–24.6)	–0.79	0.85	1			
NO_2 (pphm)	3.0 (1.4–4.4)	–0.82	0.83	0.89	1		
CO (ppm)	1.0 (0.34–1.8)	–0.67	0.41	0.60	0.57	1	
TD (daily VMT/mi^2)	152,311 (0–1,557,027)[a]	–0.16	0.12	0.16	0.17	0.02	1

[a]Average includes 47 subjects assigned TD = 0 because of no Caltrans-counted streets in buffer.

Results

Data sources. **Health data.** Of the 612 children in Los Angeles and San Diego Counties participating in CHIS 2001 and reporting a physician asthma diagnosis, 56 (9.3%) reported suffering from daily or weekly symptoms, and 68 (11.2%) reported an ED visit or hospitalization for asthma in the previous 12 months. The prevalence of daily or weekly symptoms tended to increase with age, with the highest prevalence observed in children 12–17 years of age with asthma, whereas ED visits and hospitalizations were more prevalent in children ≤ 5 years of age (Table 1). Daily/weekly symptoms were more common in boys than girls. African Americans and Asians/others were more likely to be reported as suffering from daily/weekly symptoms, whereas African Americans and Latinos had higher rates of ED visits/hospitalizations than other racial/ethnic groups (Table 1). Children from a family with an income below the poverty level, experiencing delays in receiving care for asthma, taking asthma medication, and residing in Los Angeles County reported poorly controlled asthma more often than their counterparts without these characteristics (Table 1).

Exposure data. In Los Angeles County, 14 stations measured CO, 15 stations NO_2 and O_3, 8 stations PM_{10}, and 10 stations $PM_{2.5}$ during 1999–2001. In San Diego County, four stations measured CO, eight stations NO_2, nine stations O_3, six stations PM_{10}, and five stations $PM_{2.5}$. On average 8,314, 8,148, and 8,248 hourly air pollution values were available to estimate annual averages for CO, NO_2, and O_3, respectively. For PM_{10} and $PM_{2.5}$, 75 and 167 24-hr values were available to estimate annual averages, respectively. Annual average concentrations of NO_2, $PM_{2.5}$, and PM_{10} were highly positively correlated with each other and moderately correlated with CO (Table 2). Levels of O_3 were strongly negatively correlated with these pollutants, whereas residential TD measures were not correlated with any of the station-based pollutant measures.

Linkage. We were able to successfully geocode reported residential cross-street intersections and calculate TD values for 500 (81.7% of 612) respondents. Only three subjects who were geocoded could not be assigned a TD value because of missing AADT data in the Caltrans file. The reasons reported locations were not successfully mapped included missing information for one

or both streets (63%) and inability to find a reported cross-street in the reference street map (i.e., one or both streets were not found based on spelling provided or reported streets did not intersect, 37%). The mean TD value was 152,311 (median of 86,513), with a range of 0–1,557,027 daily VMT/mi^2 (Table 2). The percentage of subjects excluded from analyses based on annual average air pollution averages varied depending on pollutant (because not every station measured every pollutant, and we used a 5-mile exclusion criterion, as explained above). Approximately 26%, 47%, 36% of subjects were excluded from the O_3, PM_{10}, and $PM_{2.5}$ analyses, respectively. Mean annual average pollutant concentrations did not differ substantially for subjects included versus excluded from analyses based on this criterion; however, the percentages of children with asthma reporting daily or weekly symptoms or ED visits/hospitalizations were lower in the excluded population (by approximately 2–5% depending on pollutant and outcome).

Statistical analyses of exposure–asthma outcome relationships. Based on logistic regression models, we did not observe associations between our asthma symptom outcome measures and NO_2 or CO; thus, we limit the following discussion to residential TD, O_3, and particles (PM_{10} and $PM_{2.5}$) (Tables 3 and 4). We observed an approximately 2-fold increase in the odds of daily/weekly symptoms in children with asthma for each 1 part per hundred million (pphm) increase in annual average O_3 [odds ratio (OR) = 1.96; 95% CI, 1.23–3.13], and this estimate did not change appreciably when we controlled for race/ethnicity and poverty level or when we added particle measures to the model, although the 95% CIs widened because of the inclusion of these covariates and the reduction in sample size (Table 3). There were suggestive associations between particles (PM_{10} and $PM_{2.5}$) and daily/weekly symptoms after adjustment for O_3, but estimates were imprecise. We also observed an approximately 2-fold increase in the odds of ED visits/hospitalizations for asthma per 1-pphm increase in O_3 after adjusting for particles (particle and O_3 exposure estimates were strongly negatively correlated). Furthermore, we also estimated 2- to 3-fold increases in the odds of ED visits/hospitalizations per 10-μg/m^3 increases in PM_{10} and $PM_{2.5}$ after adjusting for O_3 (Table 3).

We observed associations between residential proximity to traffic and asthma-related ED/hospitalizations but not for daily/weekly symptoms (Table 4). We estimated an approximately 3-fold increase in the odds of ED visits/hospitalizations for children with asthma in the highest TD exposure category (TD > 200,000 daily VMT/mi^2) compared with those with TD values ≤ 20,000 daily VMT/mi^2 after adjusting for race/ethnicity and poverty level (OR = 3.27; 95% CI, 1.08–9.89). A model that included TD, O_3, and particle measures was consistent with these findings (results not shown). Approximately 48% of the subjects with TD > 200,000 daily VMT/mi^2 had a freeway within their buffer. (Approximately 41% of children in Los Angeles County versus ~ 84% in San Diego County had a freeway in their buffer, yet of all children with TD values > 200,000, ~82% resided in Los Angeles.)

Discussion

This project demonstrated methods for linking and analyzing existing health and environmental databases as part of the national EPHT initiative currently under development in the United States (McGeehin et al. 2004). Based on this linkage/analysis effort, we documented that children with asthma living in more highly polluted areas—as assessed by ambient air monitoring data and measures of traffic near homes—experienced worse asthma morbidity than those living in less polluted areas. Our findings are in general agreement with existing evidence that short- and long-term exposure to O_3 and particulate matter increases childhood asthma morbidity (Gilmour et al. 2006; Trasande and Thurston 2005) and previous reports that children with asthma residing near heavy traffic are more likely to seek medical attention or be hospitalized for asthma than those residing near low-traffic roadways (Edwards et al. 1994; English et al. 1999; Lin et al. 2002). More important, however, our goal was to evaluate the overall success of this project in terms of an EPHT framework. In the following sections, we discuss the advantages and disadvantages of the data sources, linkage, and analyses presented here for EPHT of air pollution and childhood asthma morbidity.

Data sources. **Health data.** The CHIS is the largest statewide health survey in the country. CHIS 2001 included 18,393 children (ages 0–17 years) compared with 13,376 children included in the 2000 NHIS. Because of the sampling design, CHIS data can provide disease prevalence estimates at the local level (e.g., county level) and for specific racial/ethnic groups (the survey is conducted in six languages). These factors, along with the biennial data collection, are all strengths within the context of EPHT. Another major strength is collection of residential information at the cross-street (CHIS 2001 for Los Angeles and San Diego Counties) and residential address level (CHIS 2003 and 2005 for all counties), which allows improved spatial resolution for air pollution exposure assessment.

Limitations of the CHIS asthma data include their cross-sectional nature and reliance on self-report of physician diagnoses.

Table 4. Association (OR, 95% CI) between residential TD (in daily VMT/mi^2) and asthma outcomes in CHIS children 0–17 years of age.

	Daily/weekly symptoms		ED visit or hospitalization	
	TD only (40, 453)[a]	TD, race/ethnicity, poverty level (40, 453)[a]	TD only (52, 448)	TD, race/ethnicity, poverty level (52, 448)[a]
TD ≤ 20,000	1.00	1.00	1.00	1.00
20,001 < TD ≤ 200,000	1.12 (0.53–2.34)	0.95 (0.44–2.06)	3.05 (1.11–8.38)	2.45 (0.87–6.88)
TD > 200,000	0.77 (0.30–2.03)	0.61 (0.22–1.69)	5.01 (1.72–14.6)	3.27 (1.08–9.89)

[a]Values represent number of children with asthma with daily/weekly symptoms or ED visits/hospitalizations in previous year, total number of children with asthma.

Table 3. Association [OR (95% CI)] between annual average air pollution concentrations and asthma outcomes in CHIS children 0–17 years of age.

	Daily/weekly symptoms				ED visit or hospitalization			
	Single pollutant only (crude)	Single pollutant, race/ethnicity, poverty level	Two pollutants (O_3 + PM_{10}), race/ethnicity, poverty level	Two pollutants (O_3 + $PM_{2.5}$), race/ethnicity, poverty level	Single pollutant only (crude)	Single pollutant, race/ethnicity, poverty level	Two pollutants (O_3 + PM_{10}), race/ethnicity, poverty level	Two pollutants (O_3 + $PM_{2.5}$), race/ethnicity, poverty level
O_3[a]	44, 391	44, 391	36, 269	36, 327	53, 390	53, 390	38, 272	47, 322
(per 1 pphm)	1.96 (1.23–3.13)	2.09 (1.28–3.41)	2.29 (1.01–5.23)	3.51 (1.45–8.46)	1.16 (0.74–1.81)	1.35 (0.85–2.14)	2.89 (1.32–6.34)	2.48 (1.14–5.38)
PM_{10}[a]	36, 269	36, 269	36, 269	—	38, 272	38, 272	38, 272	—
(per 10 μg/m^3)	1.09 (0.63–1.87)	1.09 (0.62–1.93)	1.82 (0.86–3.87)		1.46 (0.85–2.52)	1.46 (0.84–2.55)	2.76 (1.33–5.71)	
$PM_{2.5}$[a]	36, 327	36, 327	—	36, 327	47, 322	47, 322	—	47, 322
(per 10 μg/m^3)	0.70 (0.29–1.67)	0.69 (0.27–1.72)		3.89 (0.86–17.5)	1.26 (0.57–2.80)	1.09 (0.47–2.50)		3.68 (0.98–13.8)

[a]Values represent number of children with asthma with daily/weekly symptoms or ED visits/hospitalizations in previous year, total number of children with asthma.

As with all cross-sectional data, there is potential temporal ambiguity between exposure and disease. In this study, we did not have lifetime residential histories, and we assigned monitoring stations and estimated TD based on current home location. Thus, our estimates could be biased depending on residential mobility patterns of children with asthma. If families with children with poorly controlled asthma tended to move away from highly polluted areas after learning that air pollution might worsen asthma, our pollution associations may be underestimated. Subsequent CHIS surveys collect information on timing of asthma diagnoses and length of residence in the same house and neighborhood. Thus, in future analyses, we will be able to examine the extent to which this bias may have affected our results. In general, collection of information on residential and school histories would help improve exposure assessment further and allow a better quantification of associations between air pollution and asthma morbidity in children as part of EPHT efforts (as discussed below). CHIS 2001 relied on parental or self-reports of physician-diagnosed asthma and related symptoms and thus may have missed a certain segment of the population with undiagnosed asthma. CHIS 2003 asked about symptoms among individuals who were not physician diagnosed. Finally, the CHIS is a telephone-based survey and generally achieves a response rate of 40%. Estimating the magnitude and direction of bias due to nonresponse on our study results would require additional data collection (i.e., a follow-up study of a sample of nonresponders to determine health and air pollution exposure status). A discussion of other potential health data sources for tracking childhood asthma and its relation to air pollution is provided in Supplemental Material (online at http://www.ehponline.org/members/2008/10945/suppl.pdf).

Exposure data. The advantages of using existing government air monitoring data to assess air pollution exposures for EPHT are that the data are readily available and, in general, measurements have been consistently recorded over many years and offer a fine temporal resolution (i.e., hourly, daily, or every third or sixth day measurements are available). The tradeoff for this fine temporal resolution is lower spatial coverage, that is, the number of stations available to characterize concentration gradients throughout an urban area is limited. Such data may be inadequate for accurate exposure assessment, especially for pollutants like CO, nitrogen oxides (NO_x), and ultrafine particles that exhibit small-scale spatial variability in concentrations depending on proximity to sources (Zhou and Levy 2007). Another major issue with regard to exposure assessment is accounting for interindividual exposure variability due to personal mobility and time spent outdoors versus indoors.

Epidemiologic studies are increasingly using residential and/or school proximity to traffic as surrogate measures of motor vehicle exhaust exposure (Salam et al. 2008), as we did in this study. Caltrans traffic data are a readily available source of information for generating such measures (see the Traffic Density Mapping Tool of the California Environmental Health Tracking Program; www.ehib.com/tools), but there are a number of limitations involved (Wilhelm and Ritz 2003). Briefly, these shortcomings include *a)* lack of information on vehicle engine types (gasoline vs. diesel) and vehicle ages in a given 24-hr traffic count, which are important determinants of emission levels, *b)* lack of information concerning the influence of meteorology (e.g., wind direction) on dispersion of these emissions, and *c)* differences between outdoor and indoor exhaust concentrations based on ventilation characteristics of the home. However, as noted previously, a growing number of studies are linking relatively simple measures of traffic exposure such as the one we used in this study to adverse respiratory outcomes in children (English et al. 1999; Gauderman et al. 2005, 2007; McConnell et al. 2006). For example, Gauderman et al. (2005) reported that a relatively simple measure of residential distance to freeway was as strongly and precisely associated with childhood asthma as a more complex exposure estimate based on air dispersion modeling of freeway emissions and even measured NO_2 concentrations. However, whether this would be the case in other settings is currently unknown; very few studies have used more complex models that account for time–activity patterns of children and spatial heterogeneity of air pollution concentrations within urban areas. Alternatives for air pollution exposure assessment include geostatistical methods such as kriging (e.g., Jerrett et al. 2005; Kunzli et al. 2005) and land use–based regression modeling (e.g., Jerrett et al. 2007; Ross et al. 2006), as discussed below.

Linkage. An advantage of the CHIS for linkage of health and environmental data is knowledge of residential locations at the cross-street (CHIS 2001) and address (CHIS 2003 and 2005) level. Knowledge of residential locations is particularly important for assessing source-specific exposures, such as exposures to certain key traffic exhaust pollutants (e.g., ultrafine particles) whose concentrations change rapidly within approximately 300 m of the source (roadway) (Zhou and Levy 2007). Mapping home locations to the census tract or even census block group level likely results in exposure misclassification for such pollutants. Here, only residential cross streets were available, which likely introduced error into the traffic exposure metric; future CHIS surveys collect information on residential addresses. Knowledge of residential locations at the cross-street and address level also allows determination of distance to existing monitoring stations; thus, subjects living far from stations can be excluded from analyses (based on the assumption that accuracy of exposure assessment decreases as one moves farther away from a station). The disadvantages of using such exclusion criteria are that they reduce sample size and may limit generalizability.

Geostatistical methods such as kriging and land use–based regression modeling are alternatives for assessing air pollution exposure [see Jerrett et al. (2005) for an in-depth discussion of these methods and others such as inverse distance weighting and air dispersion modeling]. Kriging spatially interpolates pollution levels measured at existing monitoring stations across urban areas. A major advantage is that it allows the estimation of both predicted values and their standard errors (kriging variance) at unmeasured locations (Jerrett et al. 2005). However, the predictive capability of this method is dependent on the density of monitoring stations in a given area. Reliance on existing monitors (which are generally limited in number) normally results in surfaces that oversmooth the true pattern of pollution and may introduce large errors in estimates over portions of a study area for which few observations are available (Jerrett et al. 2005). This problem may be more severe for pollutants known to vary significantly over small scales, such as NO_x and other direct traffic pollutants. For these pollutants, land use–based regression (LUR) detects small-area variations in air pollution more effectively than kriging (Briggs 1997; Lebret et al. 2000). In the LUR modeling approach, concentrations of vehicle exhaust markers such as NO_x are measured simultaneously at many locations throughout an urban area, using relatively inexpensive passive monitors (e.g., Ogawa monitors). Various GIS parameters (such as traffic and roadway density, population density, and land use) are used to predict the measured concentrations (Jerrett et al. 2007). The developed model can then be used to estimate concentrations at home and school locations of study subjects based on GIS parameter values at these locations. Although these methods allow generation of air pollution exposure estimates at a wide number of locations, a number of statistical assumptions are needed (Jerrett et al. 2005). They also require additional expertise to implement, and in the case of LUR, additional data collection, which can be labor intensive. Furthermore, LUR models developed in one area may not be readily transferable to other urban areas.

Another major issue in air pollution exposure assessment is accounting for time–activity patterns of the subjects (time spent at home, at school, in vehicles) and extent of infiltration of outdoor air pollution into indoor spaces (homes and schools). Exposure misclassification resulting from the lack of such information may be one explanation for the differences we observed in particle and TD results for the two outcomes (daily/weekly symptoms vs. ED visits/hospitalizations) in this study. Specifically, the particle associations for daily/weekly symptoms were weaker than for ED visits/hospitalizations. This may reflect more nondifferential exposure misclassification of our residence-based particle measures for older children more likely to spend time away from home during the day; although daily/weekly symptoms were more frequently reported for this age group, ED visits/hospitalizations were more common in younger children (Table 1). The same explanation could also hold for the null association we observed between residential TD and daily/weekly symptoms. Although levels of O_3 tend to be more spatially homogeneous within communities (Avol et al. 1998; Geyh et al. 2000; Monn 2001), several O_3 exposure assessment studies in Southern California children have noted the important contribution of time–activity patterns (e.g., time spent outdoors) and housing characteristics (e.g., use of air conditioning, age and size of house, use of open windows and fans for ventilation) to personal O_3 levels (Avol et al. 1998; Geyh et al. 2000; Liu et al. 1997; Xue et al. 2005). In this study, we observed associations between ambient O_3 measures and both outcomes, but point estimates were generally lower for ED visits/hospitalizations (younger children). This may suggest that station-based measures of O_3 are more accurate estimates of exposure for older children, who may spend more time outdoors and who may engage in outdoor activities that increase their exposure, compared with younger children (infants and young toddlers), who may spend more time indoors and thus generally have lower O_3 exposures. Subsequent CHIS surveys collected information on school locations so that potential differences in exposure based on location of the children can be taken into account and the impact on estimates of association can be assessed. Increased sample sizes when adding CHIS 2003 and 2005 data will also allow us to determine the robustness of our findings, which were relatively imprecise when we relied on 1 year of data for Los Angeles and San Diego Counties only. Future EPHT tracking efforts focusing on O_3-related health effects may also be improved by collecting housing information, such as air conditioning use and house size, and age and time–activity information, such as time spent at school and outdoors. Collection of such data, in concert with more spatially resolved outdoor pollution gradients (e.g., through kriging or LUR), would allow the development of more complex exposure models for air pollution (see, for example, Marshall et al. 2006 and Wu et al. 2005). This type of modeling may also be possible without the collection of time–activity and housing information directly from subjects as part of surveys, that is, by using data from the U.S. Census and existing travel demand surveys coupled with stochastic modeling. [See Marshall et al. (2006) for an example using existing Southern California Association of Governments travel demand and mobility data.] However, the usefulness of such stochastic modeling for EPHT needs further investigation.

Statistical analyses of exposure–asthma outcome relationships. The CHIS includes a large, statewide sample, thereby facilitating statistical analyses of associations between air pollution and asthma while taking into consideration potential confounding factors. For this particular demonstration project, residential cross-street information was available only for respondents in Los Angeles and San Diego Counties; thus, our resulting air pollution effect estimates were relatively imprecise. CHIS 2003 and 2005 each included approximately 3,000 children with a reported asthma diagnosis or asthma-like symptoms and residential address information, providing much larger sample sizes for future analyses. In addition to a relatively large sample size, CHIS data include information on a number of potential confounding factors including race/ethnicity, income, health insurance status, usual source of care, type of usual source of care, experience of delays in receiving asthma care, and asthma medication use. For this study, we lacked information on some potentially important confounders of the association between air pollution and asthma morbidity, including exposure to secondhand tobacco smoke and indoor allergens such as pets, cockroaches, and molds, parental history of asthma, day care attendance, and breast-feeding history. Such factors may also be important to investigate as effect measure modifiers of the relationship between air pollution exposure and asthma exacerbation. Subsequent CHIS surveys (CHIS 2003 and 2005) will allow us to examine potential residual confounding of our outdoor air pollution effect estimates by some of these factors. Systematic collection of all of these potential confounding variables may not be feasible within the framework of EPHT. Nevertheless, within an EPHT framework it may be necessary to first establish a minimally sufficient set of variables necessary to allow estimation with little residual confounding.

The goals of EPHT are not only to quantify and examine links between exposure to environmental agents—such as air pollution—and disease, but also to determine whether increases or decreases in exposure result in subsequent changes in health indicators. From a public health perspective, such information supplies data to support policy decisions—for example, mandating cleaner-burning motor vehicles and fuels. Previous studies have capitalized on natural experiments (Friedman et al. 2001; Pope 1996) or prospectively followed schoolchildren (Avol et al. 2001; McConnell et al. 2003) to demonstrate a lowering of asthma morbidity resulting from changes (reductions) in air pollution. However, serial cross-sectional surveys such as the CHIS may be more amenable to EPHT for assessing the impact of air pollution regulations, as they provide asthma prevalence data on a more routine basis and cover a diverse and representative sample of children (in this case, for California). The specific mechanics for relating changes in regulatory reductions in air pollution to consequent changes in asthma health end points are beyond the scope of this paper. We currently have exposure metrics only for the 2001 CHIS participants. However, major factors that would need to be taken into account for such analyses include *a)* disentangling the effects of meteorology versus emissions reductions on air pollution levels, *b)* evaluating and accounting for potential confounding factors that may also change over time (e.g., changes in access to health care, use of asthma management plans) in statistical models, and *c)* attributing emissions reductions and subsequent health improvements to specific regulations. It may also be of interest to examine "what if" scenarios whereby the impacts of projected air pollution levels with and without the implementation of a specific emission reduction program on subsequent health end points are evaluated. This may be particularly important in areas where major changes in population or VMT are occurring.

Conclusions

We demonstrated a method of linking and analyzing existing health and environmental data as part of the national EPHT initiatives currently under development in the United States. Our data linkage and analyses indicate that children with asthma living in high O_3 and PM_{10} areas in Los Angeles and San Diego Counties experience symptoms more frequently than those living in less-polluted neighborhoods. Children with asthma living close to heavy traffic also report more ED visits and hospitalizations than those with less traffic near their home. Limited asthma surveillance information other than data from hospital discharge records is collected at the state or national level. The advantages of the CHIS data for EPHT are its relatively large sample

size and representation of the diverse California population, biennial data collection including information on potentially important covariates, and residential locations at the address level, allowing for more spatially refined exposure assessment. Disadvantages include the cross-sectional nature of data collection, reliance on parental reports of asthma diagnoses and symptoms, lack of information on some potential confounders, and lack of residential and school histories. Some of these shortcomings were addressed in CHIS 2003 and 2005. Overall, the results from these first linkage efforts indicate the potential importance of collecting routine data on children's home and school locations (especially for studies interested in traffic exhaust impacts). Additionally, timing of asthma diagnoses, residential histories, and assessment of respiratory symptoms in undiagnosed children may be required to rule out potential biases in EPHT linkage efforts. Collection of information on children's time–activity patterns (time spent outdoors) and housing characteristics (e.g., use of air conditioning and level of ventilation) and advanced exposure modeling (LUR and kriging) may further improve exposure assessment.

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Childhood Incident Asthma and Traffic-Related Air Pollution at Home and School

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Background: Traffic-related air pollution has been associated with adverse cardiorespiratory effects, including increased asthma prevalence. However, there has been little study of effects of traffic exposure at school on new-onset asthma.

Objectives: We evaluated the relationship of new-onset asthma with traffic-related pollution near homes and schools.

Methods: Parent-reported physician diagnosis of new-onset asthma (n = 120) was identified during 3 years of follow-up of a cohort of 2,497 kindergarten and first-grade children who were asthma- and wheezing-free at study entry into the Southern California Children's Health Study. We assessed traffic-related pollution exposure based on a line source dispersion model of traffic volume, distance from home and school, and local meteorology. Regional ambient ozone, nitrogen dioxide (NO_2), and particulate matter were measured continuously at one central site monitor in each of 13 study communities. Hazard ratios (HRs) for new-onset asthma were scaled to the range of ambient central site pollutants and to the residential interquartile range for each traffic exposure metric.

Results: Asthma risk increased with modeled traffic-related pollution exposure from roadways near homes [HR 1.51; 95% confidence interval (CI), 1.25–1.82] and near schools (HR 1.45; 95% CI, 1.06–1.98). Ambient NO_2 measured at a central site in each community was also associated with increased risk (HR 2.18; 95% CI, 1.18–4.01). In models with both NO_2 and modeled traffic exposures, there were independent associations of asthma with traffic-related pollution at school and home, whereas the estimate for NO_2 was attenuated (HR 1.37; 95% CI, 0.69–2.71).

Conclusions: Traffic-related pollution exposure at school and homes may both contribute to the development of asthma.

Key words: air pollution, asthma, child, epidemiology, vehicular traffic. *Environ Health Perspect* 118:1021–1026 (2010). doi:10.1289/ehp.0901232 [Online 6 April 2010]

The role of air pollution in the development of new-onset asthma remains controversial, and the contribution of this environmental risk factor to the pandemic remains unclear (Eder et al. 2006; Sarnat and Holguin 2007). Although increasing evidence indicates that living near heavy traffic is associated with increased rates of asthma, some well-designed studies have found only weak or no associations (Heinrich and Wichmann 2004; Oftedal et al. 2009).

These inconsistencies may reflect incomplete assessment of exposures to traffic in the microenvironments in which children spend most of their time. Exposure at locations other than home, especially at school where children spend a large proportion of waking hours and may engage in physical activity that would increase the ventilation rate and dose of inhaled pollutants, may have strong influences on occurrence of asthma. However, few studies have examined the effect of traffic-related pollution at schools on asthma rates among children. Two cross-sectional Dutch studies that examined this question reported higher rates of respiratory symptoms among children in schools near roadways with heavy traffic, especially truck traffic (Janssen et al. 2003; van Vliet et al. 1997). A northern California study found that schools downwind from busy freeways had higher concentrations of oxides of nitrogen (NO_x) and higher asthma prevalence rates (Kim et al. 2004). However, in a subsequent analysis of these same data, the effect of school exposure was attenuated and no longer significant after adjusting for modeled residential traffic-related exposure (Kim et al. 2008).

Nitrogen dioxide (NO_2) is routinely measured at regulatory monitoring stations. However, these measurements reflect both background regional concentrations and local sources near the stations. Some studies have assessed local exposure to traffic-related pollution using measured NO_2 (or NO_x) as surrogate for the complex mixture of traffic-related pollutants that occurs in close proximity to roadways (Brauer et al. 2007; Gauderman et al. 2005; Jerrett et al. 2008; Kim et al. 2004; Kramer et al. 2000; Nordling et al. 2008). NO_2 can feasibly be measured at a large number of locations, and it has been widely used as a proxy for the mixture of traffic-related pollutants that vary markedly depending on distance from roadways, season, wind speed, and wind direction. However, the mixture of pollutants in close proximity to roadways includes transition metals and organic aerosols, which are more plausible causes of asthma than NO_2 (Li et al. 2003).

The Southern California Children's Health Study (CHS) was designed to investigate the chronic effects of air pollution on respiratory health. Previous analyses have found associations of asthma with residential distance to major roads and modeled and measured pollutant markers for intracommunity variation in exposure to traffic (Gauderman et al. 2005; Jerrett et al. 2008; McConnell et al. 2006; Salam et al. 2007b) and with regional pollutants in susceptible children (Islam et al. 2008; McConnell et al. 2002). In the current analysis, a prospective, longitudinal evaluation of new cases of asthma has allowed assessment of exposure before the development of asthma in an ongoing cohort of school children recruited in kindergarten and first grade, an age at which physician-diagnosed asthma becomes reliable and valid (Martinez et al. 1995). Exposure to traffic-related pollutants has been estimated at participating schools and at residences. In addition, NO_2, ozone (O_3), and particulate matter < 10 μm (PM_{10}) and < 2.5 μm in aerodynamic diameter ($PM_{2.5}$) have been measured continuously during the lifetime of most study participants at a location in each community representative of exposure in the neighborhoods where children live (Kunzli et al. 2003; McConnell et al. 2006). This design allowed examination of the joint effects of traffic-related pollution exposure at school and home and of regional measured pollution at community

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monitors. We hypothesized that exposure at school and home both contribute to the risk of new-onset asthma.

Methods

Study population. Characteristics of this cohort have been described previously (McConnell et al. 2006). Briefly, 5,349 children attending kindergarten and first grade were enrolled into a new CHS cohort during the 2002–2003 school year from 45 schools in 13 communities. Communities were selected to represent the range and mixture of regional particulate pollutants, NO_2, and O_3 in southern California.

Table 1. Characteristics of participants at study entry and incidence of asthma on follow-up.

Characteristic	n[a] (%)	Cases	IR[b] (95% CI)
Age at entry (years)			
< 6	496 (20)	22	17.0 (11.2–25.9)
6	1,178 (47)	58	19.0 (14.7–24.6)
> 6	823 (33)	40	18.9 (13.9–25.8)
Race/ethnicity			
Hispanic white	1,380 (55)	59	16.8 (13.0–21.7)
Non-Hispanic white	905 (36)	45	18.9 (14.0–25.2)
African American	77 (3.1)	6	33.9 (15.2–75.4)
Asian	97 (3.9)	8	30.8 (15.4–61.5)
Other/unknown	38 (1.5)	2	22.7 (5.7–90.8)
Sex			
Female	1,307 (52)	62	18.2 (14.2–23.3)
Male	1,190 (48)	58	19.2 (14.8–24.8)
History of allergy			
Yes	764 (34)	56	28.7 (22.1–37.4)
No	1,508 (66)	50	12.8 (6.7–24.4)
Play team sport			
Yes	918 (39)	47	19.7 (14.8–26.2)
No	1,463 (61)	67	17.9 (9.2–34.5)
Parental history of asthma			
Yes	389 (17)	31	31.3 (22.0–44.6)
No	1,899 (83)	76	15.4 (7.2–33.3)
Maternal smoking during pregnancy			
Yes	156 (6.3)	11	27.9 (15.5–50.5)
No	2,308 (93.7)	107	18.0 (5.4–60.3)
Secondhand smoke			
Yes	164 (6.7)	7	17.0 (8.1–35.6)
No	2,281 (93.3)	110	18.7 (4.1–84.0)
Mildew			
Yes	532 (22)	29	21.1 (14.7–30.4)
No	1,911 (78)	85	17.3 (7.9–37.9)
Pests in home			
Yes	1,603 (67)	75	18.1 (14.4–22.7)
No	777 (33)	36	18.2 (9.7–33.9)
Dogs in home			
Yes	730 (30)	30	15.7 (11.0–22.4)
No	1,706 (70)	86	19.7 (9.1–42.8)
Cats in home			
Yes	462 (19)	17	14.0 (8.7–22.5)
No	1,972 (81)	98	19.4 (7.2–52.2)
Indoor NO_2 source			
Yes	1,799 (73)	88	18.8 (15.3–23.2)
No	675 (27)	31	18.3 (9.9–33.9)
Wildfire exposure[c]			
Yes	251 (12)	16	24.6 (15.1–40.1)
No	1,764 (88)	79	17.3 (6.2–48.2)
Health insurance			
Yes	2,135 (88)	106	19.2 (15.9–23.3)
No	300 (12)	10	13.1 (5.7–30.3)
Household income (US$)			
≤ 14,999	329 (14)	16	19.5 (11.9–31.8)
15,000–49,999	714 (31)	38	21.1 (15.4–29.0)
> 50,000	1,292 (55)	57	16.6 (12.8–21.6)
Parental education			
Less than high school	508 (21)	23	17.9 (11.9–26.9)
At least high school	452 (19)	20	17.5 (11.3–27.1)
Some college	850 (36)	47	20.0 (14.9–26.9)
College and above	560 (24)	24	13.9 (9.1–21.3)
Total	2,497	120	18.7 (15.6–22.3)

[a]Numbers may not add up to 2,497 in some subgroups because of missing data. [b]Crude incidence (IR) rate per 1,000 person-years. [c]Occurred in 2003 during first year of follow-up.

Parents provided informed consent and completed a baseline and yearly questionnaire with information about demographic characteristics, respiratory illness, and risk factors for asthma at study entry. Children with a history of physician-diagnosed asthma at study entry (n = 715, 13.4%) were excluded from this follow-up, because the focus was on risks for incident asthma. To ensure that the study population was free of any previously undiagnosed asthma, we then also excluded children with a history of wheeze (n = 868, 16.2%) and additional children with missing information about wheeze, or missing or a "don't know" answer about asthma (n = 394, 7.4%). These exclusions resulted in a total of 3,372 children classified as disease free at baseline. We excluded another 340 children with no residential traffic information because home address could not be geocoded [disproportionately from a single community, as described in the Supplemental Material (doi:10.1289/ehp.0901232)] and another 535 children who did not participate in any of the annual follow-up questionnaires. The final sample included 2,497 children.

New-onset asthma and covariates. Children with physician-diagnosed asthma reported on a yearly questionnaire during 3 years of follow-up were defined to have new-onset asthma. Children missing questionnaires in any year continued to contribute person-time to the models until they answered yes (at which time they were censored) or were lost to follow-up. The date of new onset of asthma could not be precisely defined. Therefore, the date of onset was assigned to the midpoint of the interval between the date of the questionnaire when asthma diagnosis was first reported and the date of the previous questionnaire reporting asthma status, and this date was used for estimating follow-up time in all statistical analyses. Sociodemographic characteristics, exposure to cigarette and wildfire smoke, health insurance, housing characteristics, history of allergy, and parental asthma were assessed by questionnaire [see Supplemental Material (doi:10.1289/ehp.0901232) for details].

Community ambient air pollution measurements. Ambient levels of O_3, NO_2, PM_{10}, and $PM_{2.5}$ were measured continuously during follow-up at a central site monitor in each community, as described in the Supplemental Material (doi:10.1289/ehp.0901232) and in previous reports (Peters et al. 1999). Because there is marked diurnal variation in O_3 and exposure occurs largely between 1000 and 1800 hours, this average was used in all analyses. Average temperature and relative humidity were also obtained during follow-up from measurements at the monitoring stations.

Local traffic-related pollutant exposure. Exposure to traffic-related pollution was estimated using methods described previously (McConnell et al. 2006), and several metrics

of local traffic related exposure were compiled. Participant residence and school addresses were geocoded, and we estimated distances to the nearest freeway or other highways or arterial roads and traffic density within 150 m of each child's residence and school. Concentrations of pollutants from local vehicle emissions at homes and schools were estimated separately from freeway and nonfreeway sources, using a line source dispersion model of the incremental contribution by these nearby sources to oxides of nitrogen above the regional background levels (Benson 1989). The modeled annual concentration estimates (parts per billion) were based on distance to roadways, vehicle counts, vehicle NO_x emission rates, wind speed and direction, and height of the mixing layer in each community. However, these modeled exposures reflect the mixture of multiple pollutants from nearby traffic, and the high correlation of pollutants in the mixture precludes identifying the effect of any specific pollutant in the mixture, as described in the Supplemental Material (doi:10.1289/ehp.0901232). For brevity, these modeled exposures will be referred to as traffic-related pollutants (TRP). An estimate of combined school and home exposure to traffic was made by weighting the estimates by approximate time at school (35 hr weekly, or 21%) and assigning the remaining 79% of time to the home. Health effect estimates based on models in which the contribution of school to the combined exposure was reduced to 16% to account for a lack of school exposure during summer recess were very similar (data not shown).

Statistical methods. We fitted a multilevel Cox proportional hazards model that allows for assessment of residual variation in time to asthma onset and also for clustering of children around schools and communities (Ma et al. 2003). Letting u_i and u_{ij} denote community- and school (within community)-level random effects, with u_{ij} assumed to be positive and independent conditional on u_i, we fit the following model:

$$h_{ijl}(t) = h_{0s}(t)\, u_{ij}\, exp(\beta X_{ijl} + \delta^T Z_{ijl}), \quad [1]$$

where, for the lth subject in the ith community, and jth school, $h_{ijl}(t)$ is the hazard function at age t; $h_{0s}(t)$ is the baseline hazard function in stratum s (defined by age at study entry and sex), X_{ijl} is a traffic pollution exposure metric, and Z_{ijl} are other individual covariates, such as secondhand smoke exposure, pets in the home, and other possible confounders. All models included race/ethnicity.

The model allowed for joint evaluation of the effects of exposure to traffic-related pollutants at homes and at schools and to ambient pollutants measured at community central sites, with effects scaled to the interquartile range (IQR) for each metric of residential exposure (e.g., for TRP from the line source dispersion model) and to the total range across the 13 communities, respectively. Traffic exposure at homes and school were correlated. Therefore, in models including both exposures, home traffic estimates were deviated from the corresponding school metric to minimize the chance of collinearity and allow for valid independent effect estimates (for example, r-square for school with deviated home TRP were < 0.38 for freeway, nonfreeway, and total TRP). Although the school-specific estimates of exposure were of primary interest, models including the average residential exposure corresponding to each school (and residential exposures deviated from this average) were also fitted [see Supplemental Material (doi:10.1289/ehp.0901232) for details]. Potential confounders were evaluated one at a time, based on whether the estimates for the pollutant associations changed by > 10%. We assessed heterogeneity of traffic pollution effects by level of community central site regional pollutant measurements by comparing nested models using a partial likelihood ratio test with and without interaction terms. We examined any potential nonlinearity in the exposure–response relationship using cubic spline terms, piecewise polynomials joined smoothly at a number of break points (Hastie and Tibshirani 1990), for the exposure terms and comparing the nested models using a partial likelihood ratio test. All analyses were conducted using software designed to run within R software (R Development Core Team, Vienna, Austria) for implementing random effects Cox proportional hazards models (Krewski et al. 2009; Ma et al. 2003). All hypotheses were tested assuming a 0.05 significance level and a two-sided alternative hypothesis.

Results

Most of the 2,497 disease-free children included in this analysis were ≤ 6 years of age (range, 4.8–9.0 years) at study entry (Table 1). There were 120 new cases of asthma, resulting in an incidence rate of approximately 18.7 cases per 1,000 person-years (based on 6,434 person-years of follow-up). Rates did not differ by age at study entry or sex. About half the children were Hispanic whites, and they had the lowest rates of asthma (16.8/1,000 person years), whereas African Americans had the highest rate (33.9/1,000 person-years), but this was based on only six cases. Substantially higher rates were also observed for children with history of allergy, parental history of asthma, and maternal smoking during pregnancy.

Both residential and school TRP metrics had a wide range with skewed distributions (Table 2). For example, the mean and median for nonfreeway residential TRP were 7.3 and 6.1 ppb, respectively, and the IQR was 8.0 ppb, with a total range from 0.08 to a maximum of 55.1 ppb. A few homes and schools had very low freeway TRP (largely in a single community that has no freeways). There was ≥ 3-fold variation in the multiyear average NO_2, PM_{10}, and $PM_{2.5}$ from the lowest to highest pollution community for measurements at the central site monitors. O_3 levels varied by 2-fold.

The associations of new-onset asthma were strongest with nonfreeway TRP. The hazard ratio (HR) at homes was 1.51; 95% confidence interval (CI), 1.25–1.81; $p < 0.001$

Table 2. Distribution of annual average residential traffic-modeled pollution and of community central site measurements.

	Mean	Median	IQR	Minimum	Maximum	Range
Residential traffic (ppb)						
Nonfreeway TRP	7.3	6.1	8	0.08	55.1	55
Freeway TRP	11.1	7.3	13	< 0.0001	134.5	134.5
Total TRP	18.4	14.6	20.9	0.23	144.1	143.9
School traffic (ppb)						
Nonfreeway TRP	6.1	5.9	5.9	0.3	19.7	19.4
Freeway TRP	10.9	8.2	15.2	< 0.0001	39.9	39.9
Total TRP	17	11.9	18.5	0.7	51.4	50.7
Central-site measurements						
NO_2 (ppb)	20.4	21.8	12.8	8.7	32.3	23.6
PM_{10} (μg/m^3)	35.5	34	11.7	17.6	61.5	43.9
$PM_{2.5}$ (μg/m^3)	13.9	15.1	9.7	6.3	23.7	17.4
1000–1800 hours O_3 (ppb)	44.6	43.6	11.1	29.5	59.8	30.3

Table 3. Association between new-onset asthma and modeled exposure at home and school.

Traffic-related exposure[a]	Home HR[b] (95% CI)	School HR[b] (95% CI)	Combined[c] HR[b] (95% CI)
Nonfreeway TRP	1.51 (1.25–1.81)*	1.45 (1.06–1.98)	1.61 (1.29–2.00)*
Freeway TRP	1.12 (0.95–1.31)	1.08 (0.86–1.34)	1.12 (0.94–1.35)
Total TRP	1.32 (1.08–1.61)**	1.20 (0.91–1.58)	1.34 (1.07–1.68)

[a]Scaled to the IQR at homes for each metric (from Table 2). [b]HR (95% CI, adjusted for race/ethnicity and for baseline hazards strata of age at study entry and sex) with random effects for community and school. [c]Combined weighted for time at home and school. *$p < 0.001$. **$p < 0.01$.

(Table 3). The HR for exposure at schools was almost as large as for residential exposure (HR = 1.45; 95% CI, 1.06–1.98), and the combined average exposure weighted for time at school and home had a slightly stronger association (HR = 1.61; 95% CI, 1.29–2.00). These associations were not confounded by any of the covariates in Table 1 or by community relative humidity, which we have previously found to confound TRP exposure (Jerrett et al. 2008). There also were no significant interactions of either residential or school nonfreeway TRP with sex, allergy, or parental history of asthma, which we have previously shown modified effects of traffic exposure (McConnell et al. 2006). Similar effect sizes were observed in sensitivity analyses restricted to lifetime residents at the same address, analyses excluding children diagnosed in the first year of follow-up and excluding children who changed schools during the follow-up period. There was little evidence of nonlinearity in the exposure–response relationship based on sensitivity analyses comparing the fit of a smoothed cubic spline model of asthma with a linear model (p-value > 0.80) for the partial likelihood ratio test for models with 3 and 5 knots compared with the linear model. Weaker associations were observed with total TRP at homes (HR = 1.32; 95% CI, 1.08–1.61) and school (HR = 1.20; 95% CI, 0.91–1.58). There were weaker and nonsignificant associations with TRP modeled from freeways. Nonfreeway TRP was moderately to strongly correlated with freeway TRP at homes (R = 0.64) and at schools [R = 0.70; Supplemental Material, Table E-1, (doi:10.1289/ehp.0901232)]. However, in models co-adjusted for freeway and nonfreeway TRP, the nonfreeway effect estimates for school and home were similar to the unadjusted estimates.

Of the regional community central-site pollutants, NO_2 was associated with more than double the risk of new-onset asthma (HR = 2.17; 95% CI, 1.18–4.00; p = 0.01) over the range of exposure (23.6 ppb) across the 13 study communities (Table 4). In a model with NO_2, school and residential nonfreeway TRP exposure, the estimate for NO_2 was attenuated (HR = 1.37; 95% CI, 0.69–2.71; Table 5). However, the adjusted associations of asthma with nonfreeway TRP at homes (HR = 1.46) and schools (HR = 1.45) in Table 5 were very similar to the unadjusted effect estimates (HR = 1.51 and 1.45, respectively, shown in Table 3). In sensitivity analyses, a similar pattern of attenuation of regional NO_2 effect estimates was observed in models weighted for time at school and home (results not shown), and in models adjusted for total (freeway plus nonfreeway) TRP exposure at school and home [Supplemental Material, Table E-2 (doi:10.1289/ehp.0901232)]. There were neither statistically significant interactions of central site pollutants with residential TRP nor any consistent patterns suggestive of different effects in high and low regional pollutant communities (results not shown). We have previously shown that O_3 modified the association of team sports participation with new-onset asthma in an older cohort (McConnell et al. 2002), but there was neither a main effect of sports nor confounding or effect modification of effects of residential TRP or ambient central monitor pollutant exposure.

Distance to freeways and other major roads and traffic volume on those roads have been used in other studies as independent predictors of traffic exposure, so we examined the distribution [Supplemental Material, Table E-3 (doi:10.1289/ehp.0901232)] and correlations of these metrics (Supplemental Material, Table E-1) and the association at school and home with asthma (Supplemental Material, Tables E-4 and E-5). In general, these exposure metrics were weakly to moderately correlated with the TRP metrics but were not consistently associated with asthma in our data set.

Discussion

The study is unique in its prospective assessment of the relationship of new-onset childhood asthma to community regional air pollution and near-source traffic-related exposure at home and in a large number of schools. The results indicate that associations of asthma with traffic-related pollution from nearby sources at schools were independent of estimated effects of exposures at homes, and these effects were of similar size over a range of exposure common in Southern California. Evidence of a school effect comparable with that associated with the much larger amount of time spent at home could potentially be explained by physical education and other exercise at school that may increase ventilation rate and dose of pollutants to the lungs and thereby increase the risk associated with exposure. Traffic-related pollutant levels may also be considerably higher during the morning hours, when children are arriving at school, especially during temperature inversions that occur largely in the winter months when children are attending school (Kim et al. 2002; Ning et al. 2007). It is possible that school exposure may have reflected time spent in other locations in the child's neighborhood rather than school-specific exposure, and this possibility merits further investigation. However, time at school during the school year accounts for more than one-third of all waking hours, and in this age group it is likely that most of the child's remaining nonschool time was spent at home (Xue et al. 2004). The increased risk associated with TRP could not be explained by confounders commonly associated with asthma or by ambient NO_2 or other currently regulated regional pollutants measured at the central-site monitoring stations. These results strengthen an emerging body of evidence from both toxicologic and epidemiologic studies that air pollutants from nearby traffic contribute to the development of asthma (Salam et al. 2008).

NO_2 measured at community monitoring stations has been associated with wheeze prevalence in an older CHS cohort (Peters et al. 1999) and with increased risk of asthma incidence in a Japanese cohort (Shima et al. 2002). We have recently reported associations of both incident and prevalent asthma with ambient residential NO_2 measured outside homes of a relatively small sample of children from another CHS cohort in many of the same communities, but NO_2 in that study was a marker for intracommunity variation in the mixture of TRPs (Gauderman et al. 2005; Jerrett et al. 2008). Other studies also indicate that associations of respiratory disease with intracommunity variation in NO_2 reflect the effects of other TRP rather than NO_2 (Kramer et al. 2000). A study that measured NO_2 and NO_x in central neighborhood

Table 4. Association of new-onset asthma with community central site pollutant measurements.

Pollutant	HR[a] (95% CI)
NO_2	2.17 (1.18–4.00)
PM_{10}	1.35 (0.64–2.85)
$PM_{2.5}$	1.66 (0.91–3.05)
O_3	0.76 (0.38–1.54)

[a]HR (95% CI) across the range of exposure in the 13 communities (23.6 ppb for NO_2, 43.9 μg/m^3 PM_{10}, 17.4 μg/m^3 $PM_{2.5}$, and 30.3 ppb for 1000–1800 hours O_3), adjusted for race/ethnicity and for baseline hazards strata of age at study entry and sex with random effects for community and school.

Table 5. Mutually adjusted associations of new-onset asthma with community central site pollutant measurements and nonfreeway TRP at home and school.[a]

Central site pollutant	HR[b] (95% CI) for ambient pollutant, adjusted for TRP at home and school	HR[b] (95% CI) for home TRP, adjusted for school TRP and ambient pollutant	HR[b] (95% CI) for school TRP, adjusted for home TRP and ambient pollutant
NO_2	1.37 (0.69–2.71)	1.46 (1.16–1.84)*	1.45 (1.03–2.06)
PM_{10}	1.40 (0.62–3.17)	1.46 (1.16–1.85)*	1.53 (1.10–2.12)*
$PM_{2.5}$	1.30 (0.66–2.56)	1.48 (1.19–1.85)*	1.49 (1.07–2.08)
O_3	1.01 (0.49–2.11)	1.50 (1.20–1.86)*	1.54 (1.10–2.14)*

[a]Mutually adjusted across each row (i.e., effect of each community pollutant was examined separately in a model including both home and school TRP). [b]HR (95% CI) for central-site pollutants scaled across the range of exposure in the 13 communities (23.6 ppb for NO_2, 43.9 μg/m^3 PM_{10}, 17.4 μg/m^3 $PM_{2.5}$, and 30.3 ppb for 1000–1800 hours O_3); household nonfreeway TRP was deviated from school, scaled to the IQR for home exposure (8 ppb from Table 2). *p < 0.01.

locations found that associations with asthma were attenuated by adjustment for modeled residential traffic exposure (Kim et al. 2004, 2008). In the current study, the attenuated association between asthma and NO_2 continuously measured at the community monitor in models with adjustment for TRP also suggests that NO_2 was not causally related to asthma. However, interpretation of this result is not clear because exposure measured at the community monitor may have misclassified exposures of children in parts of the community with significant local sources of NO_2. We did not find evidence for main effects of regional O_3 and PM with asthma, consistent with results from previous analyses in older CHS cohorts, although we have previously shown that O_3 modified the effect of outdoor exercise in a genetically susceptible subgroup (Islam et al. 2009; McConnell et al. 2002).

Associations with asthma were significant for TRP exposure estimates modeled from local nonfreeway roadway proximity, traffic volume, and meteorology. There was little evidence for an effect of major roadway proximity alone, for traffic density, or for pollution from freeways. The absence of a freeway TRP effect suggests that causal pollutants may be highly reactive, resulting in steep spatial gradients from freeway sources and little exposure at the longer distances of homes and schools to freeways compared with distance to other major roads [Supplemental Material, Table E-3 (doi:10.1289/ehp.0901232)]. Several previous studies have found that the largest gradients in traffic-related pollutants occur within 150 m from roadways (Gilbert et al. 2003; Zhu et al. 2002), and variability has often been best explained by traffic volume within 300 m (Briggs et al. 2000; Gilbert et al. 2003; Ross et al. 2006). High-volume traffic on secondary roads may also produce pollutants resulting from frequent stops and accelerations, which have been found to be associated with asthma symptoms (Ryan et al. 2005). However, in an older cohort from some of these same communities, we observed associations of freeway-modeled exposures and residential freeway proximity with respiratory outcomes (Gauderman et al. 2005, 2007). Different traffic metric associations with respiratory health may have been due to variation in the distribution of freeway and nonfreeway traffic around homes in the different cohorts and the relatively small number of children living near freeways (< 7% within 150 m in the current cohort). It is also possible that the accuracy of exposure models for the freeway and nonfreeway sources varied in the two cohorts or that the greater mobility of older children resulted in health effects associated with spending time in areas such as parks in close proximity to freeways near homes.

An important distinction between the TRP and simpler traffic metrics is the inclusion of meteorology (average annual wind speed and direction and height of the mixing layer) in addition to proximity and volume. Traffic density alone [Supplemental Material, Table E-4 (doi:10.1289/ehp.0901232)] and proximity to a major roadway (Supplemental Material, Tables E-4 and E-5) had weaker positive associations with incident asthma that were not statistically significant. Therefore, inconsistent findings in some of the (largely cross-sectional) studies that have evaluated associations with asthma (Heinrich and Wichmann 2004) may reflect the failure to account for the impact of meteorology on exposure. We have recently measured NO_x at > 900 locations in these communities. Approximately two thirds of the within-community variability in NO_x was explained by TRP, a substantially higher amount of variation than was explained by any other traffic metric (unpublished data). We have also previously observed that both TRP and residential traffic proximity were associated with increased asthma prevalence in this cohort (McConnell et al. 2006).

The prospective design is a strength of this study. In other prospective studies of birth cohorts in Sweden and the Netherlands, modeled traffic-related pollutants, including NO_x and/or $PM_{2.5}$, were associated with incidence of wheeze and atopy (Nordling et al. 2008) and doctor-diagnosed asthma (Brauer et al. 2007) in children < 5 years of age, when the diagnosis of asthma may be difficult to distinguish from transient wheeze not predictive of asthma. As these cohorts mature, the relationship to asthma is likely to become more clear. For example, associations between $PM_{2.5}$ light absorption, roadway proximity, and asthmatic bronchitis and atopy were found in a study of two German birth cohorts that followed some children to 6 years of age (Morgenstern et al. 2008). Associations with allergic symptoms in these cohorts were less robust at an earlier age (Gehring et al. 2002). Other recent results from prospective studies of birth cohorts (Gehring et al. 2009) and of children (Shima et al. 2002) followed to older ages have generally shown positive associations between markers of residential TRP and asthma, although a recent Norwegian study found no association with modeled exposure at the birth address of a large cohort of children (Oftedal et al. 2009). Genetic studies examining pathways likely to mediate effects of air pollution also strengthen the causal inference that traffic-related pollutants may cause asthma. We have shown elsewhere that the risk of asthma associated with traffic exposure and with traffic-related ambient PM was modified in a predictable way by functional gene variants in pathways associated with asthma, including inflammation and airway remodeling (Salam et al. 2007a) and oxidative stress (Islam et al. 2008; Salam et al. 2007b). In another analysis of the data from this cohort, we have shown that the estimated effect of TRP was modified by socioeconomic status and psychosocial stress (Shankardass et al. 2009).

There are some limitations to these data. Our conservative estimates of TRP for the center of school buildings are likely to underestimate the exposure of students in buildings closer to the roadways, especially during dropoff and pickup of students at the beginning and end of the school day. The impact of this underestimate of exposure on the assessment of health effects is not clear, although in general it might be expected that this exposure misclassification would be nondifferential with respect to asthma and would result in underestimation of the effect of traffic exposure. Other limitations include the relatively short (3-year) period of follow-up and the retrospective questionnaire assessment of early-life risk factors, as this was not a pregnancy cohort. Our cohort excluded children who left the community before school entry and who had asthma or wheeze at study entry. However, our cohort allowed us to examine risk factors in school children, an age during which there has been little study of new-onset asthma.

Asthma is a clinical syndrome with no sensitive and specific diagnostic test available to confirm clinical assessments. Because of the clinical nature of the assessment, the reported physician diagnosis of asthma we used has been recommended and widely used as a method to classify asthma status in epidemiologic studies, although this approach has limitations (Asher et al. 1995; Burr 1992). If misclassification of true asthma based on this approach were random with respect to exposure to air pollution, then the observed associations would be likely to have underestimated the effect of exposure. Bias could explain our results if asthma misclassification were related also to exposure. This could occur if exposure were associated with access to care and differences in practice among physicians that have the potential to influence asthma diagnosis (Samet 1987). However, adjustment for factors that mediate access to care including family income, parental education, and having medical insurance did not alter our results, indicating that confounding due to differential access to care was unlikely to explain these findings. The validity of questionnaire-reported physician diagnosis has also been established in a subset of CHS participants (Salam et al. 2007a). In addition, any child with a history of wheezing at study entry was excluded from the analysis, so it is unlikely that the exacerbation of undiagnosed prevalent asthma by traffic explained these associations. Loss to follow-up could also potentially result in bias because of selection. Sociodemographic characteristics varied between the analysis cohort and children

not contributing to the analysis because of loss to follow-up or missing or poorly matched geocodes for addresses precluding estimation of residential exposure [Supplemental Material, Table E-6 (doi:10.1289/ehp.0901232)]. Hispanic children were less likely than non-Hispanic white children to be included in the follow-up analysis, as were children without insurance and with lower parental education and income. However, adjusting for these factors had little impact on the pattern of observed effects. In addition, there was little difference between exposure to nonfreeway TRP in the study sample (7.3 ppb) and in the group not included because of loss to follow-up (7.6 ppb). Therefore, it is unlikely that selection bias due to loss to follow-up explained our results.

Conclusions

An estimated 6.2 million children have asthma in the United States, making it the most common chronic disease in childhood (Moorman et al. 2007), and rates have increased markedly in developed countries over the past several decades (Braman 2006). Morbidity results in impaired quality of life for the affected child and other family members, in increased use of health services, and in school absences that have large social and economic costs (Wang et al. 2005). Our results indicate that children exposed to higher levels of traffic-related air pollution at school and home are at increased risk of developing asthma. Almost 10% of public schools in California are located within 150 m of roadways with > 25,000 vehicles daily (Green et al. 2004). Students in urban areas in eastern U.S. cities are even more likely than children in Los Angeles to attend schools near major highways (Appatova et al. 2008). Therefore, exposure to TRP is potentially an important public health problem affecting large populations of children. Planning transportation and other urban development to limit population exposure to traffic exhaust, as well as more effective control of vehicular emissions, may result in substantial long-term public health benefits.

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Examining Associations between Childhood Asthma and Traffic Flow Using a Geographic Information System

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Using geographic information systems (GIS) and routinely collected data, we explored whether childhood residence near busy roads was associated with asthma in a low-income population in San Diego County, California. We examined the locations of residences of 5,996 children ≤ 14 years of age who were diagnosed with asthma in 1993 and compared them to a random control series of nonrespiratory diagnoses (n = 2,284). Locations of the children's residences were linked to traffic count data at streets within 550 ft. We also examined the number of medical care visits in 1993 for children with asthma to determine if the number of visits was related to traffic flow. Analysis of the distribution of cases and controls by quintiles and by the 90th, 95th, and 99th percentiles of traffic flow at the highest traffic street, nearest street, and total of all streets within a 550-ft buffer region did not show any significantly elevated odds ratios. However, among cases, those residing near high traffic flows (measured at the nearest street) were more likely than those residing near lower traffic flows to have two or more medical care visits for asthma than to have only one visit for asthma during the year. The results of this exploratory study suggest that higher traffic flows may be related to an increase in repeated medical visits for asthmatic children. Repeated exposure to particulate matter and other air pollutants from traffic exhaust may aggravate asthmatic symptoms in individuals already diagnosed with asthma. *Key words:* case–control, childhood asthma, geographic information system, GIS, Medi-Cal, traffic. *Environ Health Perspect* 107:761–767 (1999). [Online 10 August 1999]
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Asthma is the most common chronic disease among children and the most common diagnosis leading to childhood hospitalization in the United States (*1*). Air pollutants that are found near busy roads as a product of traffic exhaust, such as NO_2 and particulate matter, have been shown to be associated with respiratory illness in children (*2,3*).

Several studies conducted in the last 5 years have used proximity to traffic flow as a proxy for exposure to traffic exhaust. These studies found associations between traffic flow and increased risks of childhood hospital admissions (*4*), respiratory symptoms (*5,6*), and decreased lung function (*7*). In these studies, data on traffic flow were obtained by self-report (*6*) using the highest traffic volume in the school district (*5*), measuring distances from residences to streets on maps and assigning traffic density (*7*), and linking traffic flow data to residential postal zip codes (*8*).

We were interested in examining whether a childhood residence near busy roads was associated with asthma in a low-income population in San Diego County, California. We also wanted to determine whether a child's risk for multiple asthma-related medical care visits increased as a function of traffic flow in the study area. San Diego County was chosen due to the availability of comprehensive traffic information and because health data were available from an ongoing geographic information systems (GIS) study of environmental, demographic, and health characteristics in the California/Baja California border region (*9*).

Recent availability of geocodable health data (ability to locate residences in space) and more widespread use of GIS give health researchers greater ability to link exposure information to individual addresses. We investigated whether GIS could be useful in linking traffic volume information to asthma cases and a random control series that were obtained from routinely collected billing information, which would provide a more accurate exposure assessment than linking asthma cases to average exposure values in an area, as is often done in an ecologic study design approach.

Methods

Health data. We obtained data on childhood asthma cases and controls from the Medi-Cal (California's Medicaid Program) paid claims database, maintained by the Medical Care Statistics Program of the California Department of Health Services (Sacramento, CA). This database contains information on all Medi-Cal beneficiaries in California. The Medi-Cal program pays for the health care of qualifying individuals on public assistance, those who are "medically needy" (blind, disabled, elderly), or those who are medically indigent. Data are collected by the month the Medi-Cal claim is paid, which may occur several months after the claim is submitted. This large database acquires approximately 8 million records per month. Collected data items include the patient's address, date of birth, sex, race/ethnicity, medical diagnosis [based on the *International Classification of Diseases*, Revision 9 (ICD-9) code (*10*)], the date the patient was seen by their provider, and the type of medical visit (physician visit, inpatient, outpatient, or pharmacy).

For study cases (n = 14,636) we selected all claims paid between January 1993 and June 1994 with a diagnosis of asthma (ICD-9 code 493) in a child ≤ 14 years of age residing in any of three California counties: San Diego, Imperial, or Monterey. For study controls we selected an equivalent random sample (n = 14,636) of all claims paid during the same time period with any diagnoses in children ≤ 14 years of age. Because digital traffic flow data for 1993 were available only for San Diego County, we limited cases and controls to San Diego County residents (n = 11,067 for cases and 11,174 for controls). A total of 4,655 potential control records were for pharmacy visit claims. Because these records lacked ICD-9 coding to identify the claimant's type of illness, we excluded them from the control population to prevent including asthma cases among the controls. The remaining claim records were for physician, inpatient, or outpatient visits. After restricting the data to children who were seen by a provider in 1993, we were left with a total of 7,222 cases and 3,928 controls. Because a child who had more than one

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Medi-Cal claim during the year could be included more than once in the database, we used only data from each child's first visit of the year (n = 7,053 for cases and 3,902 for controls).

We geocoded cases and controls using ARC/INFO software (version 7.1; Environmental Systems Research Institute, Redlands, CA). A total of 5,996 (85.0%) cases and 3,323 (85.2%) controls were successfully address-matched to a 1:24,000 scale street network layer obtained from the San Diego Association of Governments (SANDAG; San Diego, CA) (*11*). Address matching was not possible for claim records that had post office box, incomplete, or non-San Diego County address information.

Controls were further excluded if they were already counted as cases (n = 64), had missing or miscoded diagnosis information (n = 203), or had medical diagnoses related to respiratory disease (ICD-9 460–519; n = 754) or neoplasms (ICD-9 140–239; n = 18). We removed those claims related to respiratory disease from the control series in order to exclude diagnoses such as asthma and bronchitis. Those with neoplasms were excluded because previous studies have found associations between traffic exhaust and childhood cancers (*12*). This left a total of 2,284 controls available for analysis. The final group of selected controls was similar in sex, race, and age to the pool of potential controls.

Traffic data. We obtained average daily traffic (ADT) flow data for 1993 for all roads in San Diego County from SANDAG (*13*). SANDAG maintains a complete ADT flow database that is linked to the 1:24,000 scale street network layer. The ADT values are calculated by local and county governments using traffic counters and are collected for nearly all the highways and major arterials in the county. A much smaller proportion of local roads is also measured. The ADT values for state highways and collector roads, obtained from the California Department of Transportation (CALTRANS), are incorporated into the SANDAG database. The ADT values are defined as the average number of cars per weekday and are calculated from a minimum of 48 hr of weekday traffic flow.

Census data. To identify a block group for each case and control residence, we linked each residence to SANDAG block group boundary layers. We used data from the 1990 U.S. Census (STF3 file) (*14*) to determine percentages of labor-force unemployment (for persons ≥ 16 years of age), childhood poverty, educational status, urban/rural status, median household income, and home heating fuel use for each block group. We defined childhood poverty as the percent of children < 18 years of age with a 1989 income below the poverty level. We defined educational status as the percent of persons ≥ 18 years of age with less than a high school diploma. A block group was considered urban if 75% or more of the population resided inside an urbanized area. The percent of occupied households using electricity, utility gas, coal, and wood for home heating fuel was determined for each block group.

Data analysis. For each case and control residence, we constructed a 550-ft (168.8-m) radius circular buffer region around the geocoded residence location using ARC/INFO (Figure 1). Because an examination of several air emission dispersion models indicated 80–90% decay of pollutants between 492 and 656 ft (*15,16*), we chose a 550-ft buffer as an approximation of the exposure influence of traffic. All street segments with attributed ADT data were captured for each case and control within the buffered region. In this way we were able to link each study subject with the number of street segments having ADT data within the buffer region. For each study subject we were also able to determine the distance from the subject's residence to each street segment and the number of cars per day traveled on each street segment within the subject's buffered region.

We examined the differences in proportions by case–control status for 1990 census block group level variables and for individual level variables (sex, race/ethnicity, age, and visit type—physician visit, outpatient, or inpatient). We used Wilcoxon rank sum tests to assess the statistical significance of differences in average traffic volume for the street with the highest traffic volume within the buffer, the closest street to the residence, and for the sum of all streets within the 550-ft buffer region by case–control status. If two streets were equidistant from the residence, the street with the highest traffic volume was selected as the closest street [n = 10 (0.2%) for cases and n = 2 (0.2%) for controls]. We also calculated odds ratios (ORs) and 95% confidence intervals (CIs) (based on the traffic flow distribution for the controls) for each quintile of traffic flow and for the 90th, 95th, and 99th percentiles of flow for the three street classifications. We used multivariate logistic regression to compute odds ratios and 95% confidence intervals adjusted for potentially confounding variables (*17*). Models were run separately by sex. We used the Hosmer-Lemeshow test (*18*) to compute model fit.

Because pollutants disperse from the highway as a factor of distance and turbulence

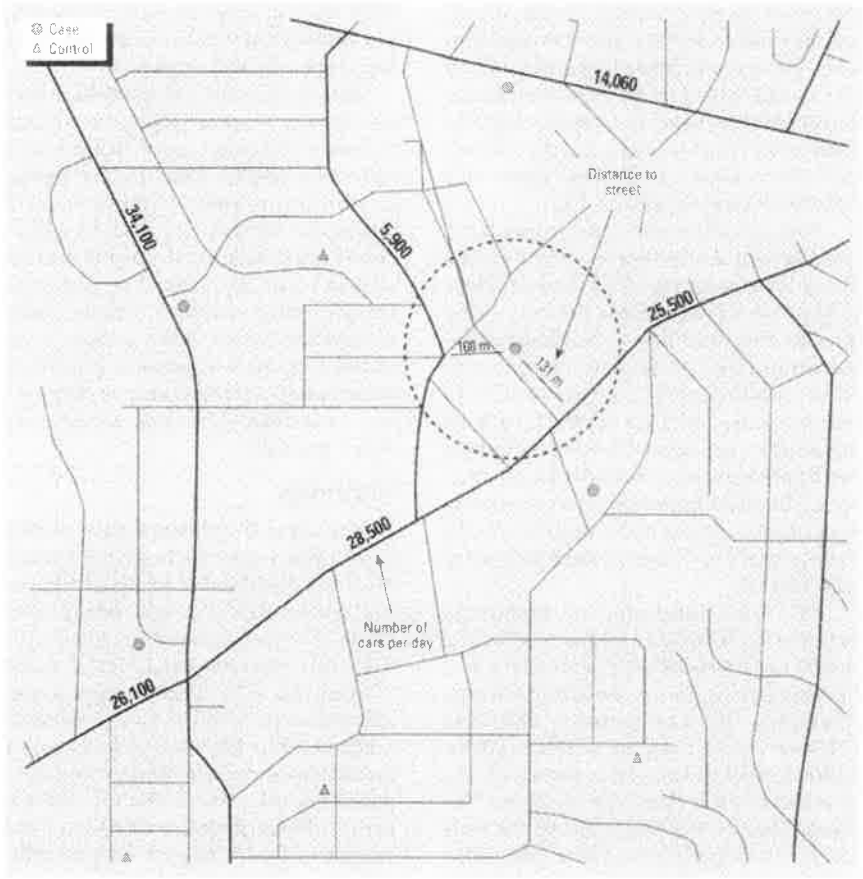

Figure 1. Buffer (550 ft; 168.8 m) encircling cases and controls to capture traffic flow.

factors such as wind speed and atmospheric stability (*4*), we applied weights from two simple dispersion models to the traffic flow data. Most dispersion models assume that plumes of motor vehicle exhaust emissions, as they move downwind, spread in a Gaussian manner in vertical and horizontal directions. We used a model adapted by Pearson et al. (*19*), which assumes no wind and that pollutants are essentially inert and disperse from the source (Pearson model). This model uses a Gaussian probability distribution which assumes that 96% of the pollutants disperse at 500 ft. The model was expressed by

$$Y = \left(\frac{1}{0.4\sqrt{2\pi}}\right) e^{-\frac{0.5(D/500)^2}{(0.4)^2}},$$

where D is the distance from the street to the residence and Y is the weighting value.

For comparison, we also used a Gaussian curve dispersion model with assumptions of neutral wind stability, a wind speed of 1 m/sec, and a pollution concentration height of 1 m (Gaussian model) (*20*). Traffic at distances < 20 m from each case and control residence was weighted by 1.0 for this second model. Using weightings from these two models (Pearson and Gaussian models), we recalculated odds ratios and 95% confidence intervals for the three street classifications: *a*) the street with the highest traffic volume, *b*) the closest street to the residence, and *c*) at all streets within the 550-ft buffer region.

We also analyzed the relationship between case–control status and the actual distance from the residence to the closest street and the street with the highest traffic flow (as opposed to weighting the traffic flow counts by distance). We computed odds ratios and 95% confidence intervals for case–control status at 100-ft intervals (0–100, 101–200, 201–300, 301–400 and > 400 feet) of the distance between the residence and the street.

In addition to analyzing the relationship between case–control locations and traffic data, we tested for spatial clustering of cases among the case–control locations (irrespective of proximity to traffic). We assessed the spatial pattern of masked data (perturbed points) using the Cuzick and Edwards test (*21*) (with a separate analysis by L. Waller and L. Zhu). This test compares the observed number of a case's nine nearest neighbors who are also cases among the set of case–control locations. A high number of cases among the nearest neighbors of other cases would suggest spatial case "clustering." To assess significance of the observed test statistic value, we randomly assigned the total number of cases to the case–control locations 200 times and calculated the statistic for each random assignment. This provided a simulation-based estimate of the test statistic distribution under the null hypothesis of no spatial pattern.

To examine whether traffic flow may be related to the frequency of medical care visits for asthma, we computed the number of physician, inpatient, and outpatient visits during 1993 for each case. The amount of average daily traffic (at the street with the highest traffic volume, at the closest street to the residence, and at all streets within the 550-ft buffer region) was compared for cases with two or more visits for asthma and for cases with only one visit during the year. We calculated the odds ratios and 95% confidence intervals for each quintile of traffic flow (based on the traffic flow distribution for those with one visit), and for the 90th, 95th, and 99th percentiles for the three street classifications (highest, nearest, and total). We also analyzed the relationship between the number of each case's medical care visits for asthma and the actual distance from that child's residence to the nearest street, as well as the distance from the residence to the street with the highest traffic flow. We used SAS, Version 6.12 (*22*), and EPI-INFO, Version 5.01a (*23*), for all statistical analyses.

Results

Case–control analysis. Table 1 shows the distribution of ICD-9 codes among the controls. Approximately one-fourth of the controls had diagnoses for nervous system and sense organ diseases, primarily disorders of refraction (nearsightedness/farsightedness), conjunctiva disorders, ear infections, and hearing loss. Other large diagnostic categories included injuries (14%), ill-defined conditions (symptoms that point with "equal suspicion to two or more diseases or two or more systems of the body, without the necessary study to make a final diagnosis"; 10.9%), and contact with the health care system (V codes; 15.6%).

Tables 2 and 3 show the distribution of cases and controls by individual and census characteristics. Cases were more likely to be male and black, and less likely to be Hispanic. Cases were also older, more likely than controls to be seen in a physician's office, and less likely than controls to have hospital outpatient visits. Cases and controls had similar block group census characteristics such as unemployment, poverty level, and

Table 1. Distribution of controls by *International Classification of Diseases*, Revision 9 (ICD-9) code.[a]

ICD-9 code	No. (%)
Total	2,284 (100.0)
Contact with health care system (V codes)[b]	357 (15.6)
Infectious/parasitic disease (001–139)	183 (8.0)
Endocrine/nutritional/metabolic disorders (240–289)	62 (2.7)
Mental disorders (290–319)	53 (2.3)
Nervous system and sense organ diseases (320–389)	551 (24.1)
Circulatory system (390–459)	10 (0.4)
Digestive system (520–579)	154 (6.7)
Genitourinary system (580–629)	52 (2.3)
Complications of pregnancy, childbirth (630–676)	7 (0.3)
Skin disease (680–709)	159 (7.0)
Musculoskeletal disease (710–739)	60 (2.6)
Congenital anomalies (740–759)	34 (1.5)
Perinatal period (760–779)	32 (1.4)
Ill-defined conditions (780–799)	249 (10.9)
Injury (800–999)	321 (14.1)

[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County. [b]Includes communicable disease, family history, reproductive problems, specific procedures, etc.

Table 2. Individual characteristics of asthma cases and controls.[a]

	Cases		Controls			
	No.	Percent	No.	Percent	OR	CI
Total	5,996	100.0	2,284	100.0		
Sex						
Male	3,595	60.0	1,160	50.8	1.45	1.32–1.60
Female	2,401	40.0	1,124	49.2	1.0	ref
Race/ethnicity						
White	1,789	29.8	632	27.7	1.0	ref
Hispanic	2,251	37.5	1,182	51.8	0.67	0.60–0.76
Black	1,254	20.9	303	13.3	1.46	1.25–1.71
Other	674	11.2	144	6.3	1.65	1.35–2.03
Unknown	28	0.5	23	1.0	0.43	0.24–0.78
Age						
< 1 year	288	4.8	322	14.1	1.0	ref
1–4 years	2,466	41.1	882	38.6	3.13	2.61–3.74
5–14 years	3,242	54.1	1,080	47.3	3.36	2.81–4.01
Type of visit						
Outpatient	1,326	22.1	992	43.4	1.0	ref
Inpatient	107	1.8	12	0.5	6.67	3.55–12.81
Physician office	4,563	76.1	1,280	56.0	2.67	2.40–2.96

Abbreviations: CI, 95% confidence interval; OR, odds ratio; ref, reference.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County.

household income (Table 3), confirming similar low socioeconomic status at the neighborhood level. We observed no significant differences between cases and controls in the census block group prevalence of home heating methods.

Out of all cases and controls, approximately 75% lived within 550 ft of a street with traffic flow data (Table 4). Controls were more likely than cases to live closer to the street with the highest traffic flow within the 550-ft buffer region ($p < 0.01$) and were more likely to have higher average traffic volume at the nearest street ($p = 0.03$) (Table 4). We saw no statistically significant difference between cases and controls in average traffic volume of the street with the highest traffic flow or in average traffic volume at all streets combined in the 550-ft buffer region. Bivariate analysis of the traffic volume variables and child's age, race, sex, type of visit, and urban status (which varied by case–control status) showed that race, type of visit, and urban status varied by traffic volume and were therefore treated as potentially confounding variables in multivariate analysis.

The analysis of case and control distribution by quintiles and at the 90th, 95th, and 99th percentiles of traffic flow at the highest, nearest, and total of all streets within the 50-ft buffer region did not show any significantly elevated odds ratios (Table 5). This did not change after controlling for race, type of visit, and urban/rural status (Table 5). No significant differences by sex were observed in sex-specific multivariate analysis. Results of the Hosmer-Lemeshow test showed good model fit for the quintile analysis [highest street: goodness of fit (GOF) statistic = 7.59, $p = 0.47$; nearest street: GOF statistic = 7.0, $p = 0.54$; total streets: GOF statistic = 3.57, $p = 0.89$). Weighting the nearest or highest traffic volumes by the Pearson or Gaussian decay curve models did not result in any meaningful changes in the odds ratios (not shown). Analysis of the distribution of cases and controls with distance to the nearest street or to the street with the highest traffic flow did not show any elevated odds ratios or dose–response patterns.

Because controls with injury diagnoses ($n = 321$) may have had traffic-associated injuries related to road proximity, all analyses were repeated without these controls. We noted no significant changes in the odds ratios.

Evaluation of spatial autocorrelation revealed evidence of spatial patterns within the set of nine nearest neighbors of the cases (Cuzick and Edwards test statistic = 18,072, $p = 0.005$). While the results indicate some clustering of cases, the logistic regression analysis indicates that this pattern is not associated with traffic volume (i.e., clustering of cases is not limited to areas of high traffic volume).

Table 3. Census block group characteristics of asthma cases and controls.[a]

	Cases ($n = 5,996$) Mean ± SD	Controls ($n = 2,284$) Mean ± SD
Percent unemployed in labor force (≥ 16 years of age)	9.0 ± 5.1	8.7 ± 5.3
Percent children < 18 years with 1989 income below poverty level	27.2 ± 19.1	27.3 ± 19.3
Percent persons ≥ 18 years with a high school diploma	31.4 ± 16.0	31.9 ± 16.6
Percent of households using home heating fuel		
Electricity	29.5 ± 18.3	30.2 ± 18.5
Utility gas	65.3 ± 19.9	64.0 ± 20.4
Coal	0.01 ± 0.1	0.01 ± 0.1
Wood	1.0 ± 2.9	1.2 ± 3.4
Median household income ($)	27,319 ± 10,848	27,410 ± 10,741
Urban[b]		
Yes	5,880 (98.1)	2,219 (97.2)
No	116 (1.9)	65 (2.9)
Total	5,996 (100.0)	2,284 (100.0)

Abbreviations: SD, standard deviation.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County. [b]≥ 75% of the population lives inside urbanized area; values are shown as number (percent).

Table 4. Traffic flow characteristics.[a]

	Cases ($n = 5,996$)		Controls ($n = 2,284$)		p-Value (χ^2 or Wilcoxon rank sum)
Street with traffic flow data within 550 feet of residence	N	(%)	N	(%)	
Yes	4,484	(74.8)	1,748	(76.5)	
No	1,512	(25.2)	536	(23.5)	NS
Average number of attributed streets within 550 ft	2.21		2.24		NS
Average distance from residence to street with highest traffic flow (ft)	281.7		269.2		<0.01
Average distance from residence to nearest street with traffic flow data (ft)	200.8		203.9		NS
Traffic volume at street with highest traffic flow (cars/day)					
Average	26,477		25,912		NS
Median	15,471		15,600		
Range	500–239,800		400–213,000		
Traffic volume at nearest street with traffic flow data (cars/day)					
Average	16,645		17,464		0.03
Median	10,600		11,400		
Range	500–239,800		400–205,300		
Traffic volume at all streets within 550 ft (cars/day)					
Average	42,880		41,497		NS
Median	21,950		19,800		
Range	500–729,200		400–775,000		

NS, not statistically significant.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County.

Visit analysis. Of all cases that had traffic count data within 550 ft of their residences, 97.8% ($n = 4,385$) had only one medical care visit for asthma during the year, whereas 2.2% ($n = 99$) had two or three visits during the year. Those who had two or three visits were more likely than those with only one visit to be Hispanic (49.5 vs. 39.9%), to be male (67.7 vs. 59.1%), to have an outpatient hospital visit for asthma (36.4 vs. 22.1%), and to have fewer physician visits (61.6 vs. 76.1%). None of the cases with more than one medical visit had a change of address during the year.

Analysis of traffic flow quintiles on streets within 550 ft of the residence showed that children with two or more medical care visits were more likely to have higher traffic flows at the nearest street than were children with only one visit (Table 6). Although there were elevated odds ratios at each traffic flow quintile, only the second quintile was statistically significant (5,501–9,000 cars/day vs. < 5,500 cars/day; OR = 2.14; 95% CI, 1.06–4.39). Adjustment for race (which was associated with the number of medical care visits and traffic volume among the cases) resulted in slight increases in the point estimates of the odds ratios (Table 6). Additional control for the type of medical care visit did not significantly change the magnitude of the odds ratios. The odds of a case having two or more medical visits (compared to one visit) was almost three times higher for individuals with

Table 5. Crude and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for asthma for children 14 years of age and younger by quintiles of traffic flow.[a]

Traffic flow (cars/day)	Case (*n*)	Control (*n*)	Crude OR (CI)	Adjusted OR[b] (CI)
At nearest street				
1st quintile (ref) (≤ 5,800)	969	352	1.0	1.0
2nd quintile (5,801–9,400)	1,013	367	1.00 (0.84–1.19)	0.93 (0.78–1.11)
3rd quintile (9,401–14,400)	864	339	0.93 (0.77–1.11)	0.82 (0.68–0.98)
4th quintile (14,401–22,400)	779	346	0.82 (0.68–0.98)	0.76 (0.63–0.91)
5th quintile (> 22,400)	859	349	0.89 (0.75–1.07)	0.79 (0.66–0.95)
90th percentile (> 27,900)	434	174	0.91 (0.73–1.13)	0.80 (0.64–1.00)
95th percentile (> 40,200)	231	87	0.96 (0.73–1.28)	0.92 (0.69–1.22)
99th percentile (> 167,700)	41	13	1.15 (0.59–2.28)	0.90 (0.47–1.74)
At highest street				
1st quintile (ref) (≤ 8,200)	988	353	1.0	1.0
2nd quintile (8,201–12,500)	886	352	0.90 (0.75–1.07)	0.84 (0.70–1.01)
3rd quintile (12,501–18,000)	785	351	0.80 (0.67–0.96)	0.76 (0.64–0.92)
4th quintile (18,801–26,400)	876	348	0.90 (0.75–1.07)	0.85 (0.71–1.02)
5th quintile (> 26,400)	949	349	0.97 (0.81–1.16)	0.89 (0.75–1.07)
90th percentile (> 43,200)	490	172	1.02 (0.82–1.27)	0.99 (0.80–1.24)
95th percentile (> 127,400)	209	86	0.87 (0.65–1.16)	0.82 (0.62–1.10)
99th percentile (> 195,400)	46	17	0.97 (0.53–1.78)	0.68 (0.38–1.22)
Sum of all streets				
1st quintile (ref) (≤ 9,100)	954	361	1.0	1.0
2nd quintile (9,101–16,700)	813	343	0.90 (0.75–1.07)	0.83 (0.69–1.00)
3rd quintile (16,701–25,000)	722	347	0.79 (0.66–0.94)	0.76 (0.63–0.91)
4th quintile (25,001–50,100)	998	351	1.08 (0.91–1.28)	1.00 (0.83–1.19)
5th quintile (> 50,100)	997	346	1.09 (0.91–1.30)	1.05 (0.88–1.26)
90th percentile (> 90,500)	466	175	1.01 (0.81–1.25)	0.98 (0.79–1.22)
95th percentile (>161,400)	251	87	1.09 (0.82–1.45)	1.06 (0.80–1.41)
99th percentile (> 366,000)	48	17	1.07 (0.59–1.96)	1.02 (0.57–1.82)

ref, reference.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County. [b]Controlling for race (Hispanic, black, other, unknown vs. white), medical visit type, (inpatient/outpatient vs. physician visit) and urban/rural status (urban vs. rural).

Table 6. Crude and adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for two or more medical care visits versus one visit for cases for children 14 years of age and younger.[a]

Traffic flow (cars/day)	*N*		Crude OR (CI)	Adjusted OR[b] (CI)
	2 or 3 visits	1 visit		
On nearest street				
1st quintile (ref) (≤ 5,500)	13	896	1.0	1.0
2nd quintile (5,500–9,000)	28	902	2.14 (1.06–4.39)	2.14 (1.10–4.16)
3rd quintile (9,001–13,000)	19	840	1.56 (0.73–3.36)	1.64 (0.81–3.35)
4th quintile (13,001–21,200)	17	875	1.34 (0.61–2.94)	1.37 (0.66–2.84)
5th quintile (> 21,200)	22	872	1.74 (0.83–3.67)	1.85 (0.92–3.71)
90th percentile (> 27,500)	11	434	1.75 (0.72–4.19)	1.86 (0.82–4.18)
95th percentile (> 41,000)	9	215	2.89 (1.07–7.40)	2.91 (1.23–6.91)
99th percentile (> 161,000)	2	43	3.21 (0.34–14.83)[c]	3.58 (0.78–16.44)

Goodness-of-fit statistic for adjusted model = 2.25, $p = 0.94$.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County. [b]Controlling for race (Hispanic, black, other, unknown vs. white). [c]Exact confidence limits.

traffic flow greater than 41,000 cars/day (95th percentile) at the nearest street (OR = 2.89; 95% CI, 1.07–7.40).

We noted increasing risks of multiple medical care visits at the 90th, 95th, and 99th percentiles of traffic volume, although confidence intervals were wide due to the small number of cases with multiple medical care visits. Analysis of traffic flow quintiles for the highest and sum of all streets did not result in any statistically significant differences in cases by number of visits (not shown). We found no dose–response relationship for the analysis of medical visit status with distance to the nearest street or to the street with the highest traffic flow.

Analysis of medical visit status of cases by traffic flow for boys and girls separately showed that girls had significantly higher risks than boys for multiple medical care visits associated with traffic flow (Table 7). We observed that the higher risks associated with the 90th, 95th, and 99th percentiles of traffic volume were almost entirely driven by the female population, with odds ratios of 3.82, 7.89, and 18.82, respectively.

Discussion

In this study we found no evidence of increased asthma risk with higher traffic counts near children's residences, even after distance weighting traffic counts using pollution dispersion models and controlling for potential confounders in a multivariate logistic regression model. However, among children diagnosed with asthma, we found that the number of medical care visits increased with higher traffic counts of nearest streets, particularly at the 90th, 95th, and 99th percentiles. These findings are consistent with asthma literature, which classifies air pollution as a contributing factor rather than a causal factor to asthma development (*24*). Respiratory irritants such as SO_2, NO_2, and particulate matter, which are products of traffic exhaust, can cause respiratory irritation and have been shown to exacerbate asthmatic symptoms, but have not been thought to directly influence asthma prevalence (*24*). Shima and Adachi (*25*) found higher serum hyaluronate levels (thought to be a biomarker of lung damage) in children in Japan who live near busy roads, as compared to those who lived farther away, but only among children who already had elevated IgE levels. This suggests that exposure to automobile exhaust may be particularly damaging to children already suffering from atopic illness.

We found an elevated risk of increased medical care visits among asthmatic children at the second quintile (5,500 cars/day) of traffic flow, but no further dose–response effect until the 95th percentile (41,000 cars/day). One explanation could be that exhaust fumes from traffic have a step-type threshold effect that was reached at the second quintile of exposure. This phenomenon has also been seen in children of smoking mothers for whom no increased incidence of asthma was noted until the mother smoked 10 or more cigarettes per day (*26*). Because children in our study were from a low-income urban area, baseline exposures for most subjects were relatively high.

When examining the risk of multiple medical care visits for asthma with traffic flow, we found that girls had significantly higher risks than boys at the highest percentiles of traffic flow (90th, 95th, and 99th). This finding is similar to that of Oosterlee et al. (*27*), who found that girls had higher risks of chronic respiratory symptoms than boys among those living near streets with high traffic flows in the Netherlands. Higher risks of decreased lung function associated with residence near major roads were also found in girls by Brunekreef et al. (*7*). Comparison of our results in California with studies done in the Netherlands should be made with caution because of differences in types of vehicle emissions and emission controls. In a recent study by Peters et al. (*28*), ozone exposure was found to be related to decreased lung function in children with asthma, primarily in girls. Although boys have been found to have

Table 7. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for more than two versus one medical care visit for cases by traffic volume on nearest street, by sex, for children ≤ 14 years of age.[a]

Traffic flow (cars/day)	Adjusted OR[b] (CI) boys	Adjusted OR[b] (CI) girls
At nearest street		
1st quintile (ref) (≤ 5,500)	1.0	1.0
2nd quintile (5,501–9,000)	1.51 (0.67–3.40)	4.21 (1.19–14.90)
3rd quintile (9,001–13,000)	1.73 (0.77–3.90)	1.36 (0.30–6.13)
4th quintile (13,001–21,200)	1.28 (0.55–3.01)	1.64 (0.39–6.94)
5th quintile (> 21,200)	1.62 (0.72–3.65)	2.51 (0.64–9.84)
90th percentile (> 27,500)	1.27 (0.46–3.54)	3.82 (0.90–16.24)
95th percentile (>41,000)	1.56 (0.48–5.06)	7.89 (1.84–33.75)
99th percentile (>161,000)	NA[c]	18.82 (2.85–124.27)

Abbreviations: NA, not available; ref, reference.
[a]From 1993 California Department of Health Services Medi-Cal paid claims data for San Diego County. [b]Controlling for race (Hispanic, black, other, unknown vs. white). [c]Insufficient sample size.

higher prevalences of asthma and may be at higher risk of respiratory illness (*29*), Peters et al. (*28*) suggest that girls with asthma may be more affected by pollution because of sex-related differences in rates of growth in height and maximum lung size (with girls achieving this earlier than boys), which may affect their response to air pollutants.

Although we found evidence of increased number of asthma-related medical care visits with higher traffic, it is not clear if repeat visits are a marker of increased severity (or number) of asthmatic symptoms or of more comprehensive patient care. However, because children in this low-income population are less likely to have routine medical care, they may be at higher risk of asthma exacerbation due to air pollution than non-poor populations (*30*).

In this case–control study, we used GIS to facilitate our analysis of childhood residence and traffic flows. The GIS allowed quick linkage of traffic count information to geocoded addresses. Further, the GIS facilitated efficient computation of traffic counts at the nearest street to the residence of the case control and at the street with the highest traffic, and summed the total traffic count at all streets within a 550-ft buffer area. By providing individual-level estimates, this method results in less exposure misclassification than applying average exposure values in an area using an ecologic study design. Additionally, because we did not obtain exposure information via questionnaire or self-report, this study was not subject to information bias that could explain our results. Because we lacked information on time–activity patterns, we were unable to determine if the children were exposed to traffic exhaust at other locations, such as schools and day-care center playgrounds. This potential misclassification is likely to be random with respect to case–control status and therefore would result in biasing our risk estimates toward the null.

We used traffic counts as a proxy for vehicle exhaust, and weighted the counts as a function of the distance from the child's residence to the roadway. We did not model particulate matter or other constituents of traffic exhaust from traffic counts, as performed by Buckeridge et al. (*31*) in a study in southeast Toronto, Ontario, Canada. We lacked the necessary data for vehicle exhaust pollutant modeling such as vehicle type and the number of traffic stops. Analysis of aerial photos, as performed by Buckeridge et al. (*31*), would be useful in obtaining information on the number of lanes in a street and traffic stops.

We were limited in this study by the number of covariates, which could potentially confound the relationship between traffic and asthma risk. In particular, we lacked information on secondhand smoke exposure and residence history. If cases who lived near busier roads were more likely to have household members who smoked than cases who lived near less busy roads, then failure to control for smoking could have resulted in a spurious positive finding for increased risk of medical care visits with higher traffic counts. However, no difference in passive smoke exposure was found in populations living near busy and quiet roads in the Netherlands (*27*), nor was any relationship found between smoking status and distance from the residence to the road in a case–control study of 7,299 patients in East London, United Kingdom (*8*).

We lacked information on residence history, thus, we were unable to ascertain the duration of exposure for the case–control analysis. Oosterlee et al. (*27*) reported that among those who lived near busy streets, families of asthmatic children had lived at their present addresses for shorter times than families with nonasthmatic children. If this selective migration also operated in our study population, the increased risk we found for multiple medical visits and traffic flow may have been underestimated.

Elevated levels of ambient air pollutants, which were not assessed in this study, may increase the risk of asthma exacerbation (*32*). Delfino et al. (*33*) found a 25% increase in daily asthma symptoms for 12 subjects with a 90th percentile (25 ppb) increase in personal ozone measurements in San Diego during 1993. From 1983 to 1994, maximum levels of ambient ozone levels decreased in San Diego County (from 280 to 150 ppb), whereas maximum levels of particulate matter (< 10 μm in diameter) more than tripled (from 38 to 129 mg/m^3) during 1984–1994 (*34*). Concurrently, age-adjusted childhood asthma hospitalization rates decreased 9% annually in the county from 1983 to 1994 (*34*). Use of data from Medi-Cal, which includes both inpatient and outpatient visits to define individual cases and controls, is an improvement over studies of area hospitalization rates. Hospitalizations for asthma occur in acute cases; therefore, moderate or mild asthma events may be missed.

We found evidence of spatial clustering for asthma cases in this population, although not near busy roads. Asthma case clustering could be due to the increased asthma risk occurring in families or to some unknown geographic clustered environmental factor such as cockroach allergens (*35*). Because of the limited availability of study covariates, we were unable to fully explore the reasons for the spatial patterns of asthma found in this low-income area.

In summary, in this study we found evidence that asthmatic children living near busy roads may have an increased risk of repeated medical care visits, as compared to asthmatic children living near lower traffic flows. This suggests that higher traffic flows may be related to an increase in severity or number of asthmatic symptoms requiring repeated medical visits in asthmatic children. Repeated exposure to particulate matter and other air pollutants from traffic exhaust may aggravate asthmatic symptoms in individuals already diagnosed with asthma. Although we found no relationship between traffic flow and the initiation of asthma, this finding should be explored further in studies with more refined measures of exposure.

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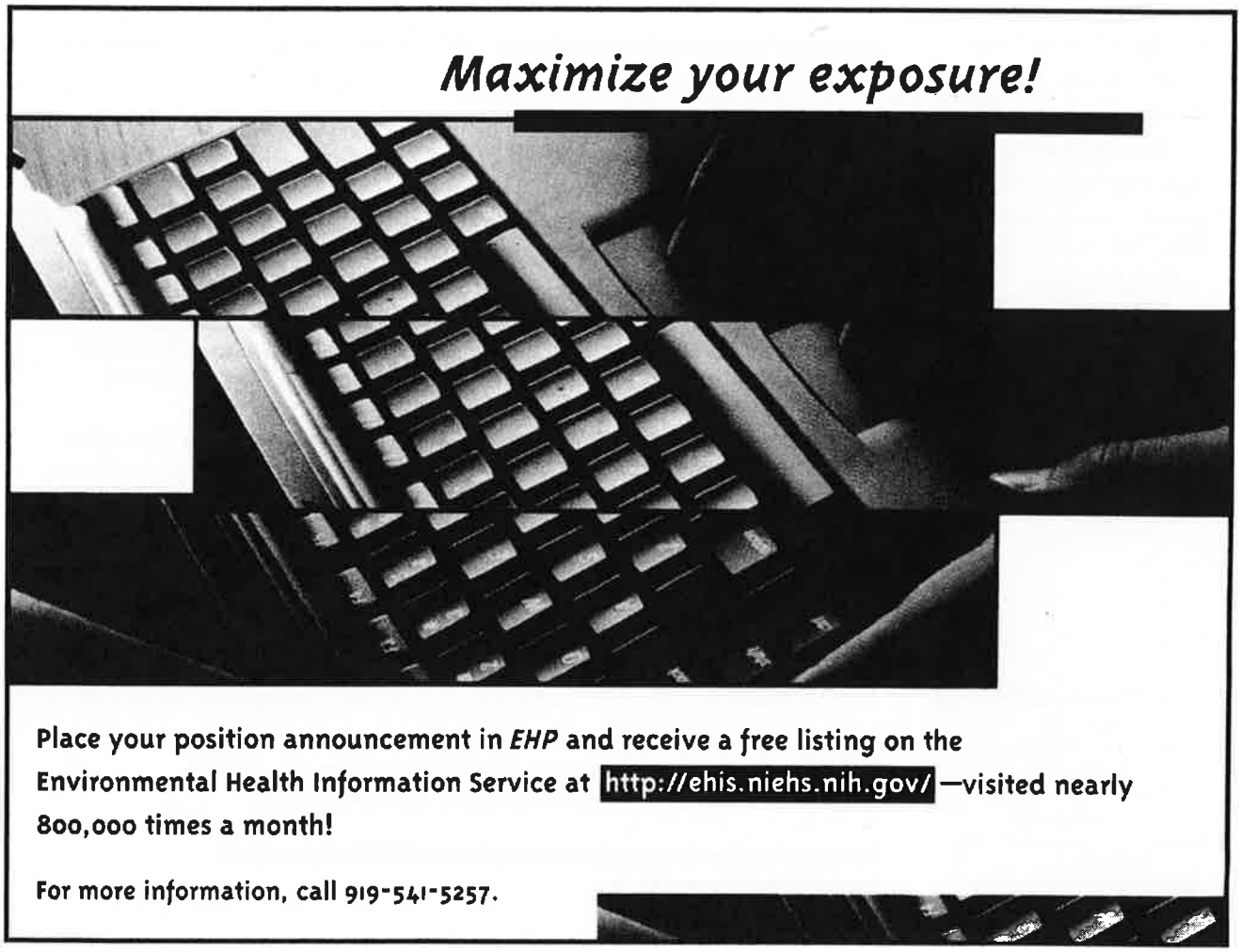

Hazard proximities of childhood cancers in Great Britain from 1953–80

E G Knox, E A Gilman

Abstract

Study objectives **– Firstly, to examine relationships between the birth and death addresses of children dying from leukaemia and cancer in Great Britain, and the sites of potential environmental hazards; and secondly to measure relative case densities close to, and at increasing distances from, different hazard types.**

Design **– Home address postcodes (PCs) and their map coordinates were identified at birth and at death in children who died from leukaemia or cancer. Potentially hazardous industrial addresses and PCs were listed from business and other directories, and map coordinates obtained from the *Central Postcode Directory* or else located directly on Ordnance Survey (OS) maps. Railway lines and motorways were digitised from OS maps. Numbers of deaths (and births) at successive radial distances from these hazards were counted and compared with expected numbers. The latter were based on a count of all PCs at similar distances. Relative case density ratios at successive distances from the hazards were obtained from observed and expected numbers, aggregated over similar sites. This was repeated for different hazard types and results were tested for evidence of systematic centrifugal case density gradients.**

Participants and setting **– All 22 458 children dying from leukaemia or cancer aged 0–15 years, in England, Wales, and Scotland, between 1953 and 1980.**

Main results **– Relative excesses of leukaemias and of solid cancers were found near the following: (1) oil refineries, major oil storage installations, railside oil distribution terminals and factories making bitumen products; (2) motor car factories, coach builders, and car body repairers; (3) major users of petroleum products including manufacturers of solvents, paint sprayers, fibreglass fabricators, paint and varnish makers, plastics and detergent manufacturers, and galvanisers; (4) users of kilns and furnaces including steelworks, power stations, galvanizers, cement makers, brickworks, crematoria and aluminium, zinc, and iron/steel foundries; (5) airfields, railways, motorways and harbours. The findings for leukaemias and for solid cancers were indistinguishable. The hazard proximities of birth addresses were stronger than for death addresses. For children who had moved house between birth and death, the proximity effect was limited to the birth addresses.**

Conclusions **– Childhood cancers are geographically associated with two main types of industrial atmospheric effluent namely: (1) petroleum derived volatiles and (2) kiln and furnace smoke and gases, and effluents from internal combustion engines.**

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Our previous studies showed that childhood leukaemias and cancers occurred in small geographical clusters.[1,2] Among 9411 childhood registrations of leukaemia and lymphoma in Great Britain between 1966 and 1983, for whom we knew the residential postcode (PC) coordinates, there were 264 case pairs/triplets who were separated by less than 150 m; and in England and Wales there were 520 registration pairs who shared a common census enumeration district (ED). Among 22 458 childhood leukaemia and cancer deaths in Great Britain between 1953 and 1980, 503 pairs shared a common PC. All these numbers were about twice the expected values. The excesses were statistically significant, and they were not explained by national variations of ED and PC sizes (numbers of households and of postal delivery points). Studies of house numbering revealed a further excess of pairs which bridged the boundaries of adjacent PCs.

The close address clusters were about 300 m in diameter, but there was also a surrounding zone of moderately enhanced risk, tapering towards normal over a radius of 2–3 km. PC clustering was stronger among birth addresses than among death addresses. It was not limited to the leukaemias and it involved all childhood cancers. Some of the close pairs were concordantly affected siblings living at the same address, but their exclusion did not alter the overall conclusion. The scale of the geographical clusters was commensurate with that of previously demonstrated space-time clusters among the 1966–83 leukaemia registrations, and also among the births and onsets of the fatal 1953–80 leukaemias.[3,4,5]

If the geographic clustering is genuine and not a demographic artefact, as is now clear, then it must reflect the existence of localised environmental hazards. Many of them must have persisted over periods of years, although the space–time interactions show that some could have been transient or intermittent. They act most powerfully within a few hundred metres, but some of them reach out to a few kilometres.

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A previous study showed that the map locations of the 9411 registrations were closer to a variety of potentially hazardous sites than were a randomly chosen set of PCs.[6] The 264 sub-150 m pairs among them showed an even stronger relationship. The apparent hazards included oil refineries, oil storage and distribution depots, railway lines, and other industrial sites. Effective ranges extended as far as 5 km from the sources. This suggested a hazard related to large scale usages of fossil fuels, especially petroleum, operating through leakage or evaporation or combustion – perhaps all three. The matching secular trends of childhood leukaemia and of petroleum usage over many decades accords with this hypothesis.

However, there were several problems of interpretation. First, the proximity investigations were undertaken initially to test the reality of the then uncertain clustering, and any causal speculations involved a second use of the same data. This introduced an unsatisfactory circularity. Second, no child population denominators were available for geographical analyses conducted on this small scale. The use of randomly selected PCs as indicators of the geographical distribution of those at risk entailed several demographic uncertainties. The most important was the possibility that numbers of children in PCs close to industrial sites might show a systematic positive bias. This caveat was also elaborated by commentators.[7] Third, there was a question of environmental confounding; hazards of one kind might be related geographically to hazards of another kind, with a consequent risk of false attribution. This applied especially to railways. Finally, the close (300 m) spacing of the core clusters did not match the wider (c 5 km) radii of the main hazard proximity effects.

These problems demanded additional study. Like the previous one, this reinvestigation lacks a true measure of child exposure on the requisite fine scale. No such data are generally available. The fundamental problems of interpretation are therefore the same. However, it is based on a fresh data set – larger and more comprehensive, and including addresses and dates of birth as well as of onset and death. It records solid cancers as well as leukaemias. But most crucially it starts from the now well supported premise that persistent local hazards must indeed exist. This no longer needs to be demonstrated. The present task is simply to discover what they are.

Methods

The cancer data set used here is the second of the two noted above, and it has been described elsewhere.[6,8,9] It records all 22 458 deaths from leukaemias and other cancers in children aged 0–15 years in England, Wales, and Scotland between 1953 and 1980. After excluding deaths in the Northern Isles of Scotland, and a few recording errors, we were left with a working file of 22 448 death addresses. The birth addresses of many were also available. Although these data are limited to fatal cases, the great majority of the children affected in these years died within the time limits of the investigation; over 75% within five years of diagnosis.[10]

The existence and locations of putative environmental hazards were determined in several ways. Point hazards such as oil refineries, gasworks, airfields, and crematoria were identified through direct searches of Ordnance Survey (OS) maps and street atlases. Many factories were found in classified business directories.[11,12] Others such as motor car works, nuclear installations, benzene refineries, and TV transmitters were taken from specialised lists. The lists and directories supplied addresses and PCs, which were then translated to map references via the *Central Postcode Directory*. Where "business" PCs could not be traced in our "residential" directory we used the alphabetically nearest residential PC (last character) as the index position. Business addresses listed as "Company House" were avoided in favour of actual factories. Some locations were reconfirmed or amended through map inspections or site visits. Detected errors were typically between 100 and 300 m. Series of particular hazard types usually consisted of 30–50 locations taken from the index in alphabetic order. For a few uncertain results, extensions of these lists enabled us to reject a suspect finding.

Linear cartographic features – railways and motorways – were digitised from OS maps using a digitising tablet. This registered strings of point coordinates and allowed the labelling of their beginnings, ends, breaks, and branch points. Tablet coordinates were translated to map positions against the key punched positions of the map sheet corners. The railways were digitised in 11 separate regions, with London excluded because of its density and complexity. Motorways outside the recently completed M25 London Orbital (which was itself omitted) were digitised as a single set.

For the point hazards, numbers of cases and numbers of PCs were counted in successively greater circles around each source. Crude densities of cases per 1000 PCs within each annulus were calculated from the aggregated numbers, and many close proximity concentrations were found. However, we feared that higher case densities in some regions of the country may have coincided with geographical concentrations of certain industries. The calculations were therefore repeated using a form of indirect regional standardisation. This was based upon direct measurements of case densities in 56 urban and non-urban areas, as described later. Region specific expected numbers of cases, within successive annuli, were then calculated. The expected number was the product of the regional case density per PC and the local number of PCs within the annulus. The observed and expected results were summed to generate a standardised density ratio (SDR) for each successive circle around each hazard type. The method is set out in an appendix.

For linear map features (rail, road) we found and measured the shortest case to hazard distance. Each address was examined against every node in the net and perpendiculars were

Table 1 Residential postcode (PC) densities (per km²) at different distances from index points

Index	Distance (km) (No)	0–0.3	–0.6	–1.0	–2.0	–3.0	–4.0	–5.0	–7.0	–10.0
Random PCs	(500)	94.05	70.42	60.96	47.83	39.81	35.99	33.21	30.28	26.67
Random cases	(700)	98.12	75.68	66.31	52.66	46.04	42.48	38.94	34.79	30.37
Refineries*	(21)	0.51	2.25	5.04	9.56	15.06	14.11	16.35	15.82	13.84
Airfields	(192)	2.43	3.74	6.62	8.20	9.72	10.13	10.19	10.32	9.37
Oil stores†	(149)	25.80	33.75	36.80	32.38	26.14	22.85	19.86	16.52	15.44
Chemical works‡	(83)	37.67	33.76	34.91	35.47	32.01	28.68	27.10	24.84	17.92
Crematoria	(64)	27.63	50.53	59.65	53.75	49.60	42.81	37.33	32.06	27.76
Car making	(193)	62.21	58.98	63.03	56.09	47.92	42.18	36.38	31.17	27.67
Safe sites¶	(195)	105.21	88.48	77.99	67.06	57.10	51.35	47.72	41.65	42.90

* Including 6 benzene refineries.
† Combined major oil storage locations, and railside (mainly) oil distribution terminals.
‡ Combined "petrochemical" and other "industrial chemicals" works plus other factories noted to have storage tank facilities.
¶ See text.
Total land area of England, Scotland and Wales is 229 870 km². The overall density of the 1 372 359 residential postcodes is 5.97/km².

dropped to inter-node segments. Cancer addresses were allocated to parallel bands on either side of the line rather than to annuli. A similar allocation of all PCs was impractical here, so numbers in different distance bands were estimated from the locations of 10 000 randomly selected PCs. The counted numbers were adjusted according to the sampling fraction, and this was used in turn to calculate a regionally standardised expected number, as above.

In the absence of population based denominators for individual PCs, alternative small group analyses were considered. A reclassification of addresses by census ED could have supplied an alternative set of coordinates, and could have provided local estimates of child years at risk. Unfortunately, appropriate census ED data were not available for the greater part of the study period. This choice would also have discarded the superior geographical resolution of the PCs. There are approximately 11 PCs per ED in England and Wales, and other studies using census based child denominators found in practice that EDs had to be aggregated into even larger blocks. These studies also failed to show clustering at these relatively coarse levels.[13 14] This approach was unlikely to meet the needs of a hazard proximity study pursuing very close range effects: and which was founded upon an initial demonstration of clustering within much smaller areas.

The matched case-control format of our main data source suggested another method for displaying comparable proximity patterns among non-cancer children. Unfortunately, control addresses had not been postcoded, matched controls were available only for about two thirds of cases, and they had been geographically matched according to place of residence at the time that the "case" had died. They were not thus really suitable for this task. Over matching in this respect was likely to obscure any true proximity effects. In pragmatic terms the extra work was beyond current resources and it seemed unlikely to attract them.

As with our previous proximity study, the crucial demographic question was whether apparent case concentrations might result entirely from systematic concentrations of PCs with large child populations around the types of hazard examined. Again, no appropriate data were available within the distance ranges considered. Regional standardisation eliminates only one component of the problem and less resolved population groupings (eg EDs) could offer no advantages. This focal demographic artefact was entirely hypothetical, and unsupported by external evidence, but we felt that it should be considered seriously. Fortunately, we found ways of examining the issue within our own data.

Results

SHORT RANGE DEMOGRAPHIC EFFECTS

Crude case densities per 1000 PCs were measured in different parts of the country: 56 rectangular areas covering major cities and inner cities, and residual surrounding areas. They were greater in population-dense than in sparse areas. The mean overall density was 16.36 deaths per 1000 residential PCs, but individual values descended from Coventry (29.98), Scunthorpe (27.78), Doncaster (24.30), Glasgow (24.01), Liverpool (23.27), Hull (23.15), Birmingham (23.07), central London (22.41) ... to ... Southampton (14.32), Gloucester (13.49), Brighton (12.06), Exeter (11.16), and Bournemouth (9.34); and the residual non-urban zones of northern England, Wales, and southwest England (10.28, 9.60, 7.79).

Although correlations with high population density have been demonstrated elsewhere,[14] these regional variations do not accurately follow known mortality patterns. They must stem in part from the different ways in which PC boundaries are delineated. PCs in population-dense industrial zones probably have more delivery points (DPs) per PC, and possibly more children per DP. Such excesses would create a false overall association between industrial hazards and cancer deaths per 1000 PCs. This necessitated the development of the stratification and standardisation procedures outlined above.

Unfortunately, broad area standardisations provide no protection against the falsifying effects of high DP PCs in the immediate vicinities of industrial premises. There is no available measure of industrially related demographic distributions on this scale. However, we tested the proposition indirectly by calculating densities of PCs per km² around each of the hazard types examined. The results are sum-

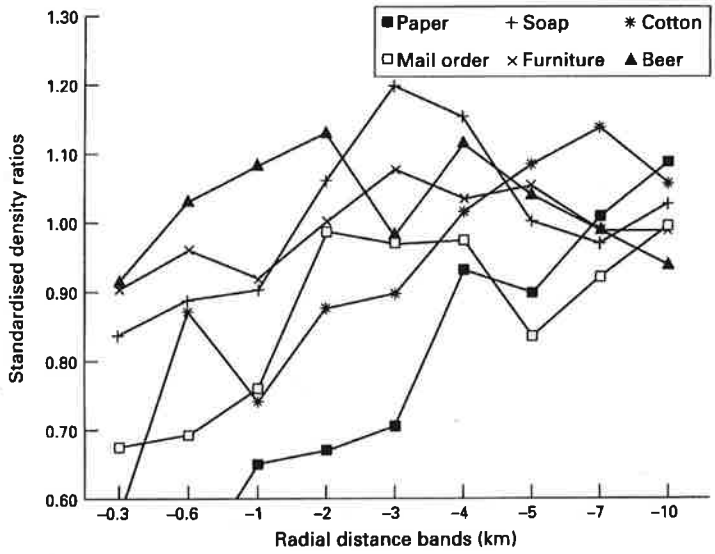

Figure 1 Industrial/residential competition.

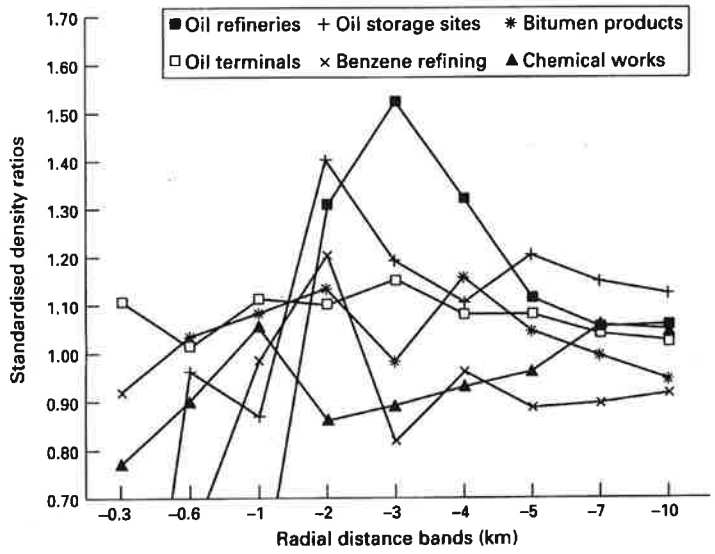

Figure 2 Oil refining and storage.

marised in table 1. PC densities are compared around:

- A random sample of PCs;
- A random sample of cancer deaths; and
- Potential hazards of several different types.

Local PC densities around random PCs and around cancer cases were much greater than the overall national value (5.97 PCs/km^2) based upon total land area. This is because the sample selection process is biased in favour of densely inhabited regions. At increasing distances, the high densities tapered towards the national value as this selection effect became diluted. The much lower values around some of the hazard sites, sometimes increasing at greater distances, probably indicate a dissociation between homes and factories. Many large installations are situated in rural areas, or at coastal or estuarine sites, or grouped together in industrial estates so that there are few residential PCs nearby. Oil refineries, nuclear power stations, and airfields, for example, virtually exclude human habitation within a km or more of their centres. Other hazards with evidence of local industrial-residential competition included chemical works, oil distribution terminals, crematoria, and harbours. However, not all hazard types exhibited these residential exclusions, notably the less segregated installations labelled as "safe sites".

Factories closely integrated with residential areas might show very local competition for space within individual PCs. This could result in artificially low densities of cancers per 1000 PCs. This was confirmed directly. Figure 1 shows SDRs surrounding the premises of 36 paper manufacturers, 21 soap manufacturers, 35 cotton spinners and weavers, 34 brewers, 31 mail order firms, and 38 furniture makers – the 195 locations aggregated as the "safe sites" of table 1. In these instances we know that the low short range case densities per 1000 PCs did not stem from low PC densities per km^2. Together with their later regression towards the mean regional values, these findings most probably indicate short range domestic-industrial competition for space within PCs adjacent to or containing a factory.

The general regional relationship between low PC densities per km^2 and low case densities per 1000 PCs, the latter probably mediated through an excess of low DP PCs, also accords with these short range findings; but there was no way in which this could be specifically confirmed within the available data. Formal exclusion of a demographic artefact capable of mimicking source related case concentrations was not possible. However, this artefact remains entirely hypothetical, without the support of prior evidence; and despite these searches of our own data, no positive evidence in its favour was found. Indeed, the balance of evidence indicates an artefact capable only of reducing factory-adjacent cancer density estimates.

PETROLEUM BASED INDUSTRIES

Figure 2 shows the pattern of SDRs in separate bands around 15 major oil refineries, 17 major oil storage areas, and 168 lesser oil distribution terminals and storage sites – mostly rail to road or rail to factory transfer sidings or factory depots. Short range SDRs were irregular through small numbers; but for the refineries and major storage sites there was a clear SDR excess between 1 and 5 km (see table 2 for the aggregated results). Figure 2 also illustrates results for 45 manufacturers of industrial chemicals, excluding the 10 classified as petrochemical factories. Petrochemical factories showed a non-significant excess and the others showed none at all; nor did the six major UK benzene refineries, plants which at this time produced about a million tonnes per year. Critics of our previous paper thought that our results supported a link between benzene and leukaemia,[6 7] but we found no direct evidence on this, then or now.

Table 2 Proximities of death addresses to point hazards

Hazard type	*No of sites*	*Distance (km)*	*No of cases*	*No expected*	*SDR**	χ^2	*P*
Oil refineries	15	1.0–5.0	329	259.9	1.27	18.34	<0.001
Oil farms	17	1.0–5.0	735	621.5	1.18	20.74	<0.001
Oil distribution terminals	168	1.0–5.0	3752	3411.0	1.10	34.08	<0.001
Bitumen products	22	0.0–5.0	1664	1512.8	1.10	15.11	<0.001
Detergent makers	33	0.0–3.0	1200	1039.3	1.23	24.85	<0.001
Plastics formers	28	0.0–2.0	283	249.2	1.14	4.59	<0.05
Car makers	31	0.0–5.0	2031	1789.2	1.14	32.70	<0.001
Coach builders	32	0.0–5.0	2025	1840.1	1.10	18.48	<0.001
Solvent makers	32	0.0–3.0	921	785.2	1.17	23.48	<0.001
Spray painting	33	0.0–5.0	2049	1846.7	1.11	22.16	<0.001
Galvanizers	69	0.0–5.0	6580	5922.1	1.11	73.09	<0.001
Paint makers	28	0.0–5.0	3679	3467.7	1.06	12.88	<0.01
Rubber manufacturers	97	0.0–5.0	5595	5273.2	1.06	19.65	<0.001
Varnish makers	13	0.0–2.0	337	286.3	1.18	8.97	<0.01
Fibreglass fabrication	37	0.0–3.0	1331	1201.2	1.11	14.03	<0.001
Adhesives makers	29	0.0–3.0	798	688.1	1.16	17.57	<0.001
Powder coating	26	0.0–5.0	784	682.4	1.15	15.11	<0.001
Steel works	44	0.0–5.0	1829	1739.5	1.05	4.61	<0.05
Aluminium casting	24	0.0–2.0	354	310.1	1.14	6.20	<0.01
Zinc casting	43	0.0–3.0	1523	1389.9	1.10	12.74	<0.001
Car batteries	14	0.0–3.0	459	363.0	1.26	25.37	<0.001
Power stations	69	0.0–3.0	1039	881.4	1.18	28.18	<0.001
Cement works	12	0.0–3.0	209	158.8	1.32	15.86	<0.001
Crematoria	64	0.0–1.0	302	214.6	1.41	35.60	<0.001
Crematoria	64	0.0–3.0	2167	1830.9	1.18	61.70	<0.001
Brickworks	27	0.0–2.0	142	117.6	1.21	5.08	<0.02
Airfields	192	0.0–3.0	876	743.6	1.18	23.57	<0.001
Rail yards	106	0.0–1.0	409	362.5	1.13	5.97	<0.02
Harbours	82	0.0–2.0	688	585.6	1.17	17.91	<0.001

* SDR (standardised density ratio) in this table accumulates individual bands shown in figures across the ranges given in column 3. Most sources gave significant results at several boundaries – exemplified here by the crematoria. For most hazards the table displays SDRs at 2.0, 3.0, or 5.0 km, usually selecting the maximum O/E (observed/expected) ratio. Significance levels were sometimes greater at other boundaries.

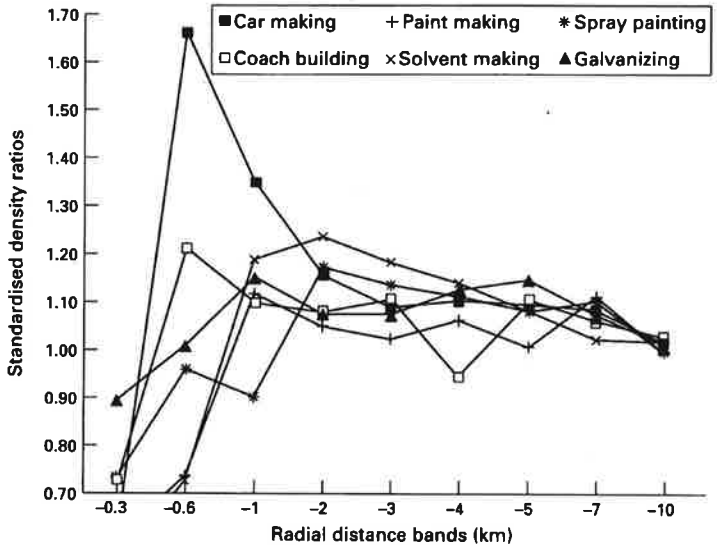

Figure 3 Automobile manufacture.

AUTOMOBILE INDUSTRY

It has been claimed [15] that a greater quantity of volatile organic compounds is discharged when painting a car than is emitted from its exhaust during the whole of its road career. The 31 car factories examined (fig 3) showed a clear excess of leukaemias and cancers, which was greatest at 0.3–0.6 km, but extended up to 5 km. Coach building and body repair firms (32 sites) had a less powerful but significant effect over the same range.

Non-car factories pursuing related trades, and using similar organic materials, were also examined. Among them were 69 galvanizing plants – which use solvents for metal cleaning – and 33 spray painting contractors. They showed pronounced excesses at ranges beyond the very shortest. Paint manufacture (28 sites) had a less powerful effect. These three, together with car manufacture and coach building, comprise the 193 sites whose adjacent PC densities are described in table 1. Brake manufacture (13 sites) and tyre making (23 sites) showed no effects. Other forms of rubber manufacture showed a small relative excess within 5 km (1.06) but none at shorter ranges (0–3 km). Among other solvent based processes examined, there were excesses in the vicinities of varnish makers, fibreglass fabricators, adhesives makers, and factories undertaking powder coating of metals. Despite the use of solvent based metal cleaning, 71 electroplating factories exhibited no apparent effect. Factories (22 sites) making halogenated hydrocarbons – chlorinated and fluorinated – likewise had no apparent effect, but 32 other solvent manufacturers showed highly significant cumulative excesses ($p<0.001$) up to 5 km. After some short range irregularities, significant effects were also found for 22 makers of bitumen based products, 33 detergent factories, and 28 plastics factories.

METAL CASTING AND REFINING

The associations with car manufacture and with galvanizing suggest alternative non-solvent exposures: namely to metal casting, metal forming and welding. Examination revealed (fig 4 and table 2) short range risks associated with aluminium, zinc, and iron and steel casting. Steelworks showed a moderate effect. Firms offering contract welding construction showed nothing, but the addresses are probably remote from their on-site work locations. Casting and refining of lead (22 sites) showed no excesses, although car battery factories exhibited a strong effect. Lead casting is a relatively low temperature process and the battery making effect may be related to the manufacture of battery casings, plastics forming or solvent usage, rather than lead processing.

"Hot process" associations were not limited to metal forming. After some short range irregularities, powerful medium range excesses were evident for power stations, crematoria and cement works. Cement rail terminals had no effect. Brickworks showed a local effect but potteries (48 sites) and gasworks (98 sites) did not. The contrast between brick and pottery manufacture may be an effect of the relative scales of production, and the absence of an apparent hazard from gasworks may be explained in terms of local population exclusions or perhaps in terms of their history. In 1965/6 20 million tonnes of coal and oil were used in the production of gas, but by 1970/1 this was down to 8 million; and by 1976 to 0.2 million, as gas manufacture finally vanished.[16] Even before 1970, and still in the era of gas manufacture, many local gasworks were closed or reduced to simple storage sites following the introduction of high pressure coal and oil gasification processes. These new processes, and the import of liquefied natural gas, led to the

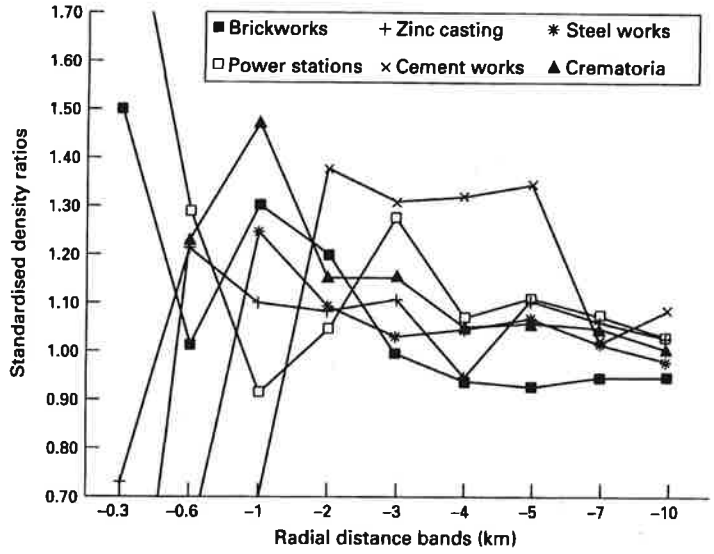

Figure 4 Kilns and furnaces.

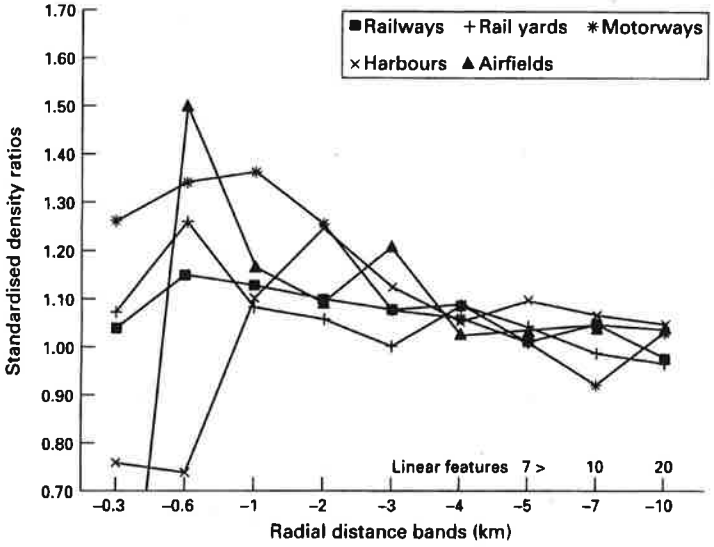

Figure 5 Transport facilities.

construction of high pressure long distance pipelines and to centralised production.

TRANSPORT ROUTES

Motorways

Distances were measured between all leukaemia/cancer bearing PCs and the nearest motorway (outside and excluding the M25) and a frequency distribution compiled. This was repeated for 10 000 randomly selected PCs and the result used to calculate regionally standardised expected values. SDRs were calculated for an extended set of parallel distance bands up to and beyond 20 km. There was a significant excess of cases within 4.0 km of a motorway, and a significant deficit beyond 4.0 km. The calculation was repeated without the use of local standardisation, with the same result.

Railways

The railway network was examined in the same way. The pooled observed and expected numbers across all 11 rail regions showed a strong and significant excess within 2 (or 3 or 4) km of a rail line and a significant deficit beyond 4 km. The distance distributions differed in different regions but there were significant SDR excesses in at least one sub-4 km band in 9 of the 11 regions. The main exception was in industrial Lancashire and north Cheshire, which showed no difference. Here, a very high proportion of control PCs were within 2 km of a railway and thus afforded no effective basis for comparing near and far distances. Railway goods yards showed a significant short range SDR of 1.13. Harbours, most of them serviced by railway lines, and heavily laden with other industrial sources, showed an SDR of 1.17 within 2.0 km.

Distance related SDRs within 4 km of railways and motorways were readjusted using pooled data beyond 4.0 km as the reference level, and these adjusted relative risks are shown (on an extended scale) in figure 5. As with many industrial sites, as well as with harbours and airfields (also in fig 5), the railway risk was lower at 0–0.3 km than at rather greater ranges, probably from residential-industrial competition for space in railway adjacent PCs. For the motorways this was less pronounced. The rail and motorway analyses were repeated for the 9411 leukaemia registrations, with the same results.

OTHER INDUSTRIES

A wide range of industrial locations gave negative results. In addition to those already mentioned they included agricultural fertilizer rail terminals, MOD rail sidings, TV transmitters, cake and biscuit bakers, dry battery makers, magnetic tape makers, nuclear plants, PVC compound manufacturers, and makers of wood preservatives.

AIRBORNE DRIFT

Easting and northing biases of cases, relative to expected cases, were measured in all analyses. Nothing was found. For some, the calculations were repeated with specific drifts added to the coordinates of the hazards, to represent the effects of the prevailing wind. The results were compared with those of zero and counter prevailing wind drifts. No evidence of any drift was found and centrifugal risk gradients generally diminished with increasing imposed biases, in any direction. In the case of motorways, for example, a "dummy" linear hazard displaced 8 km N and 8 km E from the actual network anulled the cancer proximity pattern.

BIRTH ADDRESS OR DEATH ADDRESS?

This question was tested first against a composite list of those "hot process" sites whose individual effects had already been demonstrated. It included steelworks (44 sites), power stations (69 sites), cement works (12

sites), crematoria (64 sites), aluminium casting works (24 sites), and zinc casting works (43 sites). Proximities were measured against the following:

- The full list of death addresses,
- The subset known not to have changed their PCs between birth and death,
- The birth addresses of those who moved, and
- The death addresses of those who moved.

The full death address list showed an observed/expected (O/E) ratio of 1.15 within 3 km. Non-movers showed a ratio of 1.19. The birth addresses of movers also showed 1.19 and the death addresses of movers a ratio of 1.15. All these excesses were highly significant ($p<0.001$), suggesting that while early exposures were more important than later ones, they could also be effective at later ages. This corresponds with our earlier findings in relation to PC sharing pairs.[2] However, detailed examination revealed that many of those who had changed their PCs had moved only a short distance. The mean distance was 35.6 km, but the distribution was highly skewed and 49.9 % of this group had moved less than 5.0 km and 65.1 % less than 10.0 km.

The examination was therefore repeated among 3213 children who had moved more than 10 km, and where both addresses were accurately coded. Within 3.0 km of the hazard closest to either address, the birth places showed an O/E a ratio of 1.09 (572/526.9). The ratio for the death addresses around the same sources was only 0.80 (420/526.9). Each was significant, as was the difference between the numerators (set on a common denominator). Their contrary directions reflect the imposed separation, through selection, of the two addresses; if one address is close to a particular hazard then the other is necessarily some distance away. Similar results were obtained for other hazard types and for different migration distances. Among children who had moved more than 5.0 km and where at least one address was within 10 km of a galvanising plant, we found 186 births but only 83 deaths within 3 km of the nearest source.

We conclude that early exposure is the more important. Cancer initiations in migrating children may indeed be limited to exposures occurring within a short period before or after birth. The hazard proximities of the death addresses, as well as of birth addresses, in the total data set could reflect only their strong geographical associations.

Discussion

The logistical and methodological difficulties of conducting intimate proximity analyses on so large a scale are reflected in the paucity of comparable reported studies. Apart from several proximity studies of childhood cancers and leukaemias around nuclear installations,[17–20] we found little which could be related to the findings described here. Several large scale geographical-ecological investigations have related socioeconomic factors and population densities to childhood leukaemia risks, but within much broader zones. Neither the relative distance methods developed for investigating particular nuclear sites[19–21] nor the assembly of enumerated civil populations into, for example, 10 km squares or 25 km radius circles,[17 22] met the technical needs of the present search.

The continuing demographic difficulties of a PC based method are clear, and a totally unambiguous demonstration of proximity clustering has still not been possible, but the present investigation stands on less error prone foundations than did the previous one.[6] First, our recent analyses have effectively dispelled caveats about the reality of short range case clustering[2] and the existence of geographically localised hazards is not now in doubt. Proximity studies are no longer concerned with this issue and can be directed solely at asking what those hazards might be. Second, some of the hazard associations match those previously postulated. The prior hypothesis of a petroleum associated leukaemia risk seems to be confirmed in a very large and independent set of disease data, and against a wider range of environmental sources. Third, the new analysis bases its estimates of geographical population distributions upon much larger numbers of PCs.

The main remaining source of ambiguity is a hypothetical systematic excess of children at risk in PCs close to suspect sources. A local excess of high DP PCs or of high child DPs could create a false apparent excess of cancers. Broad areal density standardisation (as used) provides no protection against hidden, small scale demographic structuring of this kind. Nor could the use of available population data, such as EDs or electoral wards, be expected to resolve this problem. However, our own data revealed low rather than high areal PC densities adjacent to many putative hazards. There were clear signs of competitive exclusion of residential PCs from the vicinities of very large plants: and of houses from those residential PCs which were very close to factories. Notably, low case densities were found within very short distances of non-volatile organic compound, non-combustion sites. Local demographic variations of these kinds could readily mask a genuine short range hazard related risk or create false appearances of a low risk; but they could not easily create a false excess. The major risk mimicking potential artefact therefore remains entirely hypothetical. There was never any prior evidence to show that it existed and our own indirect evidence now suggests that it does not.

Further reassurance against false positive demographic artefacts appeared in analyses of cases who had migrated between birth and death. The highly asymmetric ratios of 572/420 birth/death addresses within 3 km of a "hot process" source among children who had migrated 10 km or more and a 186/83 birth/death ratio within 3 km of galvanizing plants among children who had migrated 5.0 km or more are both independent of these demographic contingencies. These results also suggest a powerful general method for investigating age sensitive exposures where there are no demographic data available at all. We shall

describe its properties and develop its usage in a later study.

Effective apparent exposures were concentrated around two main kinds of installation namely: (a) producers, refiners, distributors, and industrial users of petroleum fuels and volatile petroleum products, and (b) manufacturing processes using high temperature furnaces, kilns, and combustion chambers. Some sources, notably internal combustion engines and oil fired furnaces, met both criteria. These specific associations and their absence around other sources were too coherent to suggest a demographic artefact. The few inconsistencies were probably due to different scales of production or restricted periods of operation. There was little to suggest systematic confounding from adjacent sitings of different processes. With few exceptions (eg pottery manufacture) the main factory based hazards were distributed independently across many regions. The rail and motorway associations were relatively ambiguous and could more readily be attributed to adjacent industrial hazards, but even here the most obvious direct common factor would be the use of petroleum fuel, especially diesel. Diesel is relatively non-volatile and it is difficult to envisage a spillage based or access based mechanism affecting both sources, so if fuel combustion is the common factor the most eligible materials must be blow-by fumes from crankcases and substances emitted from exhausts.

However, combustion fumes could not account for all the findings and there was no indication of any other class of effluent capable of explaining the full range of effects. None of the carcinogenic industrial species listed in the IARC monographs would easily serve as a common determinant.[23] The specific role of benzene, much discussed in the literature and in the media, was not supported by an examination of the major benzene manufacturing sites. Its reputed leukaemia toxicity may have led to special containment measures, but it would be surprising if none had escaped.

Rather than one specific effector, we must therefore consider the possibility that there might be several – from among the aromatics, other volatile organic substances, arsenic, metal fumes, soot and smoke compounds, radioactive elements, carbon monoxide, oxides of nitrogen and sulphur, and others. Alternatively, chemical species from different hazard types are known to combine in the atmosphere to form secondary pollutants.[24] The suspect primary substances are oxides of nitrogen and volatile organic substances, discharged singly or in combination. The secondary pollutants include ozone, aldehydes, ketones, nitrogenated organics, nitrates, hydrogen peroxide, ionised oxygen, and many others.

Alternative modes of access to such substances include the following:

- Diffusing gases and volatiles reaching the children or their pregnant mothers directly,
- Mediation through parental/occupational germ-cell injury,
- Occupational/domestic contamination via clothing or through other means.

The last is feasible only for a limited range of sources (eg car factories or paint spraying contractors) and it is unlikely that sufficient numbers of parents were employed in oil storage installations and crematoria to explain the excess cancers in their vicinities. This is also against an exclusively parental occupational germ-cell or fetal exposure mechanism. For a comprehensive single explanation we must favour direct exposure of pregnant women or young children to airborne substances diffusing into the surrounding environment.

The apparent absence of tumour type specificities for different sources possibly reflects an intimate mix of several potent substances with different tissue specificities or a single common effector capable of reaching many different tissues and invoking a polyvalent response, as in the case of prenatal medical irradiation.[9] Alternatively, it might reflect only the limited geographical resolution of our analyses. The O/E ratios are all modest and hazard adjacent type specific excesses could easily be buried among cases unrelated to the source in question.

Finally, we have to ask whether the moderately increased densities of cases around these hazards also explain the previously reported high local concentrations represented by same PC and same ED pairs.[1 2] The first reached out as far as 3 km and sometimes 5 km, while the close pairs were detected within diameters of about 300 m. They do not necessarily stem from a common mechanism, although two considerations suggest that they might. First, identified sources must vary in the toxicities or containment of their emissions, and case densities surrounding the more dangerous could be nearer to those within the small clusters. Second, populations within high exposure zones are themselves so patchy that any resulting cancers are necessarily concentrated within small sub-areas.

We can not at present resolve these issues, and patterns of exposure could be far more intricate than our present results have indicated. Combustion based and volatile organic substances must be emitted by many less obvious sources at lower elevations, diffusing over shorter ranges, and leading to more concentrated effects than those surrounding major installations. Candidates include domestic and commercial heating systems, oil storage bunkers, oil delivery spillages, garage blocks, small workshops, bus stations, school or hospital chimneys, municipal incinerators, petrol stations, and many others. There are also many close clustered exposures to non-effluent hazards, each capable of reaching cellular targets and damaging the genetic material, or immunological processes, or the mechanism of cell division. They include domestic radon and gamma radiation, medical radiation, ambient electromagnetic fields, infections of mother or child and prenatal drug exposures. All of them have been implicated although none offers a comprehensive and exclusive explanation.[9 22 25–30] To solve these questions we need

an extended and better differentiated record of exposures, and more discriminating modes of examination.

Appendix

CALCULATION OF STANDARDISED DENSITY RATIOS (SDR)

Consider a set of h hazards of similar type, H.

The area surrounding each hazard consists of concentric annuli, A_{hr}, containing O_{hr} observed cases and P_{hr} PCs.

Each hazard is located in one of 56 rectangular zones Z_z, in which k cases and n PCs give a zonal case density $D_z = k_z/n_z$ and the expected number of cases in each of the A_{hr} annuli is

$$E_{hr} = P_{hr} * D_z.$$

Summing observed and expected values for all hazards of a given type gives the SDR for each successive annulus around a hazard of type H as

$$SDR = \frac{\Sigma_h O_{hr}}{\Sigma_h E_{hr}}$$

The functions of regional standardisation are to calculate local expected numbers, weighted according to corresponding regional case densities; and to calculate ratios (SDR) between the aggregated observed and expected numbers. For restricted subsets of cases, such as specific tumours, or children who moved house, the regional case densities are first reduced in proportion with the size of the subset.

We thank Dr A M Stewart for access to the data of the Oxford Survey of Childhood Cancers. The postcoding of the OSCC records was part of a research programme supported by the Medical Research Council and by the Three Mile Island Public Health Fund (USA). EGK's expenses during the current work were defrayed through a Leverhulme Emeritus Research Fellowship.

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International Agency for Research on Cancer

World Health Organization

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IARC: DIESEL ENGINE EXHAUST CARCINOGENIC

Lyon, France, June 12, 2012 -- After a week-long meeting of international experts, the International Agency for Research on Cancer (IARC), which is part of the World Health Organization (WHO), today classified diesel engine exhaust as **carcinogenic to humans (Group 1)**, based on sufficient evidence that exposure is associated with an increased risk for lung cancer.

Background
In 1988, IARC classified diesel exhaust as *probably carcinogenic to humans (Group 2A)*. An Advisory Group which reviews and recommends future priorities for the IARC Monographs Program had recommended diesel exhaust as a high priority for re-evaluation since 1998.

There has been mounting concern about the cancer-causing potential of diesel exhaust, particularly based on findings in epidemiological studies of workers exposed in various settings. This was re-emphasized by the publication in March 2012 of the results of a large US National Cancer Institute/National Institute for Occupational Safety and Health study of occupational exposure to such emissions in underground miners, which showed an increased risk of death from lung cancer in exposed workers (1).

Evaluation
The scientific evidence was reviewed thoroughly by the Working Group and overall it was concluded that there was *sufficient evidence* in humans for the carcinogenicity of diesel exhaust. The Working Group found that diesel exhaust is a cause of lung cancer (*sufficient evidence*) and also noted a positive association (*limited evidence*) with an increased risk of bladder cancer (Group 1).

The Working Group concluded that gasoline exhaust was possibly carcinogenic to humans (Group 2B), a finding unchanged from the previous evaluation in 1989.

Public health
Large populations are exposed to diesel exhaust in everyday life, whether through their occupation or through the ambient air. People are exposed not only to motor vehicle exhausts but also to exhausts from other diesel engines, including from other modes of transport (e.g. diesel trains and ships) and from power generators.

Given the Working Group's rigorous, independent assessment of the science, governments and other decision-makers have a valuable evidence-base on which to consider environmental standards for diesel exhaust emissions and to continue to work with the engine and fuel manufacturers towards those goals.

Increasing environmental concerns over the past two decades have resulted in regulatory action in North America, Europe and elsewhere with successively tighter emission standards for both diesel and gasoline engines. There is a strong interplay between standards and technology – standards drive technology and new technology enables more stringent standards. For diesel engines, this required changes in the fuel such as marked decreases in sulfur content, changes in engine design to burn diesel fuel more efficiently and reductions in emissions through exhaust control technology.

However, while the amount of particulates and chemicals are reduced with these changes, it is not yet clear how the quantitative and qualitative changes may translate into altered health effects; research into

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this question is needed. In addition, existing fuels and vehicles without these modifications will take many years to be replaced, particularly in less developed countries, where regulatory measures are currently also less stringent. It is notable that many parts of the developing world lack regulatory standards, and data on the occurrence and impact of diesel exhaust are limited.

Conclusions
Dr Christopher Portier, Chairman of the IARC working Group, stated that "The scientific evidence was compelling and the Working Group's conclusion was unanimous: diesel engine exhaust causes lung cancer in humans." Dr Portier continued: "Given the additional health impacts from diesel particulates, exposure to this mixture of chemicals should be reduced worldwide."(2)

Dr Kurt Straif, Head of the IARC Monographs Program, indicated that "The main studies that led to this conclusion were in highly exposed workers. However, we have learned from other carcinogens, such as radon, that initial studies showing a risk in heavily exposed occupational groups were followed by positive findings for the general population. Therefore actions to reduce exposures should encompass workers and the general population."

Dr Christopher Wild, Director, IARC, said that "while IARC's remit is to establish the evidence-base for regulatory decisions at national and international level, today's conclusion sends a strong signal that public health action is warranted. This emphasis is needed globally, including among the more vulnerable populations in developing countries where new technology and protective measures may otherwise take many years to be adopted."

Summary evaluation
The summary of the evaluation will appear in The Lancet Oncology as an online publication ahead of print on June 15, 2012.

(1) JNCI J Natl Cancer Inst (2012) doi:10.1093/jnci/djs034
http://jnci.oxfordjournals.org/content/early/2012/03/05/jnci.djs034.abstract; and
JNCI J Natl Cancer Inst (2012) doi: 10.1093/jnci/djs035
http://jnci.oxfordjournals.org/content/early/2012/03/05/jnci.djs035.abstract

(2) Dr Portier is Director of the National Center for Environmental Health and the Agency for Toxic Substances and Disease Registry at the Centers for Disease Control and Prevention (USA).

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Link to the **audio file** posted shortly after the media briefing:
http://terrance.who.int/mediacentre/audio/press_briefings/

About IARC
The International Agency for Research on Cancer (IARC) is part of the World Health Organization. Its mission is to coordinate and conduct research on the causes of human cancer, the mechanisms of carcinogenesis, and to develop scientific strategies for cancer control. The Agency is involved in both epidemiological and laboratory research and disseminates scientific information through publications, meetings, courses, and fellowships.

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Annexes

Evaluation groups - Definitions

Group 1: *The agent is carcinogenic to humans*.
This category is used when there is *sufficient evidence of carcinogenicity* in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than *sufficient* but there is *sufficient evidence of carcinogenicity* in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2.
This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost *sufficient*, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (*probably carcinogenic to humans*) or Group 2B (*possibly carcinogenic to humans*) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms *probably carcinogenic* and *possibly carcinogenic* have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with *probably carcinogenic* signifying a higher level of evidence than *possibly carcinogenic*.

- **Group 2A:** The agent is *probably carcinogenic to humans*.
  This category is used when there is *limited evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals. In some cases, an agent may be classified in this category when there is *inadequate evidence of carcinogenicity* in humans and *sufficient evidence of carcinogenicity* in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of *limited evidence of carcinogenicity* in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

- **Group 2B:** The agent is *possibly carcinogenic to humans*.
  This category is used for agents for which there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals. It may also be used when there is *inadequate evidence of carcinogenicity* in humans but there is *sufficient evidence of carcinogenicity* in experimental animals. In some instances, an agent for which there is *inadequate evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 3: The agent is *not classifiable as to its carcinogenicity to humans*.
This category is used most commonly for agents for which the evidence of carcinogenicity is *inadequate* in humans and *inadequate* or *limited* in experimental animals.
Exceptionally, agents for which the evidence of carcinogenicity is *inadequate* in humans but *sufficient* in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans.
Agents that do not fall into any other group are also placed in this category.

An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

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Group 4: The agent is *probably not carcinogenic to humans*.
This category is used for agents for which there is *evidence suggesting lack of carcinogenicity* in humans and in experimental animals. In some instances, agents for which there is *inadequate evidence of carcinogenicity* in humans but *evidence suggesting lack of carcinogenicity* in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group.

Evidence for studies in humans - Definition

As shown previously, the evidence relevant to carcinogenicity is evaluated using standard terms. For studies in humans, evidence is defined into one of the following categories:

Sufficient evidence of carcinogenicity: The Working Group considers that a causal relationship has been established between exposure to the agent and human cancer. That is, a positive relationship has been observed between the exposure and cancer in studies in which chance, bias and confounding could be ruled out with reasonable confidence. A statement that there is *sufficient evidence* is followed by a separate sentence that identifies the target organ(s) or tissue(s) where an increased risk of cancer was observed in humans. Identification of a specific target organ or tissue does not preclude the possibility that the agent may cause cancer at other sites.

Limited evidence of carcinogenicity: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence.

Inadequate evidence of carcinogenicity: The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available.

Evidence suggesting lack of carcinogenicity: There are several adequate studies covering the full range of levels of exposure that humans are known to encounter, which are mutually consistent in not showing a positive association between exposure to the agent and any studied cancer at any observed level of exposure. The results from these studies alone or combined should have narrow confidence intervals with an upper limit close to the null value (e.g. a relative risk of 1.0). Bias and confounding should be ruled out with reasonable confidence, and the studies should have an adequate length of follow-up. A conclusion of *evidence suggesting lack of carcinogenicity* is inevitably limited to the cancer sites, conditions and levels of exposure, and length of observation covered by the available studies. In addition, the possibility of a very small risk at the levels of exposure studied can never be excluded.

In some instances, the above categories may be used to classify the degree of evidence related to carcinogenicity in specific organs or tissues.

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Traffic-related Air Pollution near Busy Roads
The East Bay Children's Respiratory Health Study

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Recent studies, primarily in Europe, have reported associations between respiratory symptoms and residential proximity to traffic; however, few have measured traffic pollutants or provided information about local air quality. We conducted a school-based, cross-sectional study in the San Francisco Bay Area in 2001. Information on current bronchitis symptoms and asthma, home environment, and demographics was obtained by parental questionnaire (n = 1,109). Concentrations of traffic pollutants (particulate matter, black carbon, total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) were measured at 10 school sites during several seasons. Although pollutant concentrations were relatively low, we observed differences in concentrations between schools nearby versus those more distant (or upwind) from major roads. Using a two-stage multiple-logistic regression model, we found associations between respiratory symptoms and traffic-related pollutants. Among those living at their current residence for at least 1 year, the adjusted odds ratio for asthma in relationship to an interquartile difference in NO_X was 1.07 (95% confidence interval, 1.00–1.14). Thus, we found spatial variability in traffic pollutants and associated differences in respiratory symptoms in a region with good air quality. Our findings support the hypothesis that traffic-related pollution is associated with respiratory symptoms in children.

Keywords: air pollution; asthma; bronchitis; epidemiology; vehicle emissions

Numerous epidemiologic studies have documented adverse effects of air pollution on health (1). The majority of these population-based studies have used pollutant concentrations measured at central monitoring sites to estimate exposures and have not, in general, considered local spatial variability in pollutant levels. However, motor vehicle emissions, the principal source of ambient air pollution in most urban areas, are likely to vary substantially within a given community, and researchers have begun to document differences in traffic-related pollutants on a neighborhood scale (2, 3).

Recently, a number of epidemiologic studies have reported associations between residential proximity to busy roads and a variety of adverse respiratory health outcomes in children, including respiratory symptoms, asthma exacerbations, and decrements in lung function (4–12). In some reports, truck traffic has been more strongly associated with these adverse outcomes than total vehicular traffic (6, 7, 10, 11).

Most studies have used metrics of proximity to traffic as surrogates of exposure to traffic pollution (e.g., residential proximity to major roads, traffic volume at the nearest road, or modeled levels of traffic pollution). Few have measured pollutant concentrations as part of the exposure assessment or provided information on local air quality (7, 10–12). The majority of studies have been conducted in Europe and Japan, where fleet composition (diesel versus gasoline), emissions factors, fuel specifications, land use, and population distributions near busy roads differ from those in the United States. Regional and microenvironmental concentrations of particulate matter (PM) may be higher in European cities compared with many parts of the United States (13). Therefore, it is important to evaluate the extent to which proximity to traffic may be associated with health impacts in the United States. Previous studies in the United States were conducted in areas of Southern California and the Northeast with significant local air-quality problems; both used metrics of proximity to traffic, not measured pollutant concentrations (8, 14).

The objective of this study was to explore associations between respiratory symptoms and exposures to traffic-related air pollutants among children living and attending schools near busy roads in an urban area with high traffic density but good regional air quality. Some of the results of this study have been previously reported in the form of abstracts (15).

METHODS

Study Design and Health Assessment

We conducted a school-based, cross-sectional study in the San Francisco metropolitan area (Alameda County, CA) in 2001. The study area was comprised of 10 neighborhoods that span a busy traffic corridor. School sites were selected to represent a range of locations upwind and downwind of major roads (Figure 1).

In spring 2001, we enrolled children (grades 3–5) in participating classes (n = 64) using methods similar to those used in other school-based studies (16–18). We obtained information on health outcomes (bronchitis symptoms in the past 12 months and physician-confirmed asthma in the past 12 months), demographics, home environmental factors, and activity factors using parental questionnaires (English and Spanish) (for additional information on the study design and health assessment, *see* the online supplement). The study protocol was approved by the Committee for the Protection of Human Subjects, California Health and Human Services Agency.

Air Pollution from Traffic

We measured concentrations of traffic pollutants (particulate matter [PM_{10}, $PM_{2.5}$], black carbon [BC], total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) at the school sites. PM_{10} and $PM_{2.5}$ mass concentrations were measured using filter-based samples, whereas BC concentrations were determined on the PM_{10} filter samples using an established light attenuation method that we validated for fiberfilm filters (19, 20). NO_X and NO_2 concentrations were determined with passive diffusion samplers (Ogawa, Inc., Pompano Beach, FL). Nitric oxide (NO) concentrations were calculated as the difference between NO_X and NO_2.

Pollutant monitoring was conducted simultaneously at all school sites for 11 1-week intervals in the spring (March–June) and for 8 weeks

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Figure 1. East Bay Children's Respiratory Health Study area. The study region is to the east and across the bay from the city of San Francisco.

in the fall (September–November) of 2001. NO_X and NO_2 were sampled during all weeks at each school. PM_{10} and $PM_{2.5}$ and the BC concentrations were not measured every week. Study-averaged air pollution concentrations were calculated at each school by first normalizing the data to account for occasional missing values. Additional details are described in the online supplement and elsewhere (21). In preliminary analyses, we also used school location in relationship to prevailing winds and proximity to busy roads as an additional traffic metric.

Data Analysis

We examined associations between pollutants and health outcomes using a two-stage hierarchical modeling strategy. This method has been used in other epidemiologic studies of air pollution when pollutants were measured at the group level (18, 22). In our study, the exposure groups were represented by the neighborhood schools. In the first stage, we initially identified potential confounders (demographic, host, or home environmental variables) associated with health outcomes in this dataset. We then performed exploratory stepwise logistic regressions to develop a model in which individual-level characteristics best predicted the odds of each health outcome. Explanatory variables that remained significant at $p < 0.15$ were retained in the model. We then fit a logistic regression model that included an indicator variable for each school in addition to the individual-level covariates.

In the second stage, the adjusted school-level logits or prevalence rates determined in the first stage were regressed on the school-specific ambient pollutant concentrations. In this manner, we obtained the log odds ratios (ORs) relating asthma or bronchitis symptoms to air pollution, after adjusting for individual-level risk factors.

We calculated adjusted ORs for a change in measured pollutant concentration equal to the interquartile ranges of the pollutant distributions. Analyses were conducted using SAS version 8.2 for Windows (Cary, NC) and STATA, version 8 (College Park, TX).

RESULTS

We distributed 1,574 questionnaires in 64 participating classrooms in the 10 schools. Three children were excluded because their parents spoke neither English nor Spanish. Among the remaining students, there was a response rate of 70.7% (1,111/1,571). Participation rates across schools ranged from 61–83%. Approximately 30% completed the questionnaire in Spanish. Two children with reported cystic fibrosis were excluded from the analysis. The final analysis sample consisted of 1,109 questionnaires.

Table 1 summarizes the participants' demographic characteristics, prevalence of selected personal and home environmental characteristics, and respiratory health outcomes. Our study population was racially diverse. Approximately 30% of households had incomes below the federal poverty line. Fourteen percent of the parental respondents reported having been told by a doctor that their child had asthma in the preceding 12 months. This represents a measure of period prevalence of asthma and would include some incident cases. Twelve percent of children had bronchitis symptoms in the past year. Of those reporting bronchitis symptoms in the past 12 months, 43% also reported having asthma. Using a slightly different definition of asthma (physician-diagnosed ever, and asthma symptoms, including wheezing, in the past 12 months), 11% of our study population had current asthma.

The estimated pollutant concentrations at the schools are summarized in Table 2. Concentrations of several pollutants (i.e., BC, NO_X, NO, and, to a lesser extent, NO_2) were higher at schools located within 300 m downwind of a freeway compared with those at schools upwind or further from major traffic sources. There was less variation in $PM_{2.5}$ and PM_{10}. Concentrations of BC, NO_X, and NO were highly correlated (r^2 = approximately 0.9 for each interpollutant correlation). The study average $PM_{2.5}$ (12 $\mu g/m^3$) was similar to the annual average concentration of $PM_{2.5}$ at the central monitoring station, located approximately 15 km south of the study area. NO_X and NO_2 measurements at the school sites away from traffic were similar to levels measured at the regional site (21).

Table 3 summarizes the results of the two-stage hierarchical logistic regression models of the odds of asthma and bronchitis symptoms in the previous year in relationship to six different pollutants, each examined in separate regressions. Results are shown for all subjects, for long-term residents only (1 year or longer at the current address), and for the latter group stratified by sex. In addition to the traffic metric, explanatory variables

TABLE 1. DEMOGRAPHIC, FAMILY, AND HOME CHARACTERISTICS OF THE EAST BAY CHILDREN'S RESPIRATORY HEALTH STUDY RESPONDENTS

Characteristics	All Subjects (*n = 1,109*) (%)	Subjects Attending Schools: Near and Downwind of Major Roads (*Four Schools, n = 402*) (%)	Subjects Attending Schools: Far or Upwind of Major Roads (*Six Schools, n = 707*) (%)
Sex			
Female	52.6	51.8	53.1
Race/ethnicity			
White	12.6	11.0	13.5
Black, African American	11.1	7.0	13.4
Hispanic	43.5	47.6	41.2
Asian	14.0	15.5	13.1
Other/multiracial	18.9	18.8	18.9
SES indicators			
Household at/below federal poverty level	31.3	31.8	31.0
Parent's education: high school or less*	48.7	51.4	47.1
Family history			
Biological mother with asthma	12.2	9.5	13.7
Maternal smoking during pregnancy	10.3	7.8	11.7
Home indoor environment			
Smoker in the household, since child's birth	17.9	13.1	20.6
Smoker in the household, current	7.2	3.2	9.5
Furry pet	37.3	36.0	38.1
Pests, past 12 mo	63.1	65.4	61.8
Gas stove	63.1	63.6	62.9
Indicator of mold/mildew presence, past 12 mo	44.6	43.5	45.3
Health outcomes			
Chest illness before age 2	23.3	18.8	25.9
Asthma, past 12 mo	14.0	13.9	14.1
Bronchitis, past 12 mo	12.1	13.2	11.5

* Parent responding to the questionnaire.

retained in all of the final models for asthma and bronchitis included chest illness before age 2 years, household mold/moisture, and pests observed in the home in the preceding 12 months. The final models for asthma also included maternal history of asthma. The addition of other potential confounders such as race/ethnicity, socioeconomic variables, maternal smoking during pregnancy, current smoker in the home, air conditioning, and gas stove use yielded similar pollutant effect estimates.

For the full sample, associations were observed between both asthma in the past 12 months and bronchitis symptoms in the past 12 months and the pollutants, especially NO_X, NO, and BC. The effect estimates for $PM_{2.5}$ and PM_{10} were smaller, which may have been due in part to the smaller concentration ranges among the 10 sites for these pollutants. No multipollutant models were evaluated because of the high interpollutant correlations. Restricting the analysis based on duration of residence (i.e., at

TABLE 2. NEARBY TRAFFIC SOURCES AND AVERAGE POLLUTANT CONCENTRATIONS AT TEN SCHOOLS

School	Major Traffic Source†	AADT (*#/d*)	Distance‡ (*m*)	< 300 m Downwind	PM_{10} ($\mu g/m^3$)	$PM_{2.5}$ ($\mu g/m$)	BC ($\mu g/m^3$)	NO_X (*pbb*)	NO_2 (*pbb*)	NO§ (*pbb*)
1	No				30	12	0.7	42	22	19
2	Yes	90,000	230	Yes	29	13	0.9	55	24	31
3	Yes	210,000	360	No	32	12	0.8	49	21	29
4	No				30	12	0.8	41	19	22
5	Yes	210,000	130	Yes	30	12	0.9	62	26	36
6	No				30	12	0.7	39	21	17
7	No				29	11	0.7	33	20	11
8	Yes	130,000	350	No	29	12	0.7	45	23	21
9	Yes	210,000	200	Yes	30	12	0.9	57	26	31
10*	Yes	190,000	60	Yes	32	15	1.1	69	31	38
Study average					30	12	0.8	49	23	25

Definition of abbreviations: AADT = annual average daily traffic; BC = black carbon; NO = nitric oxide; NO_X = total nitrogen oxides; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter of aerodynamic diameter 2.5 μm or less; PM_{10} = particulate matter of aerodynamic diameter 10 μm or less.

Estimated average pollutant concentration at each school based on normalized concentrations (*see text*). Monitoring was conducted for 11 weeks in the spring (March–June) and 8 weeks in the fall (September–November of 2001). The number of weeks underlying our estimates of chronic exposure varied for each measured pollutant: NO_X (18), NO_2 (19), BC (11), $PM_{2.5}$ (10), and PM_{10} (9).

* There is also a shopping center and a parking lot abutting the school grounds to the south and a freeway off ramp less than 50 m to the northwest.

† Includes roads with AADT above 50,000 vehicles per day located within 1,000 m of school. AADT estimate provided by the California Department of Transportation (Cal Trans).

‡ Distances were estimated using a geographic information system. Latitude and longitude of the monitors were determined using a global-positioning system device (Garmin GPS II). In some cases, distances were estimated using aerial photographs or measured using a distance wheel.

§ $NO = NO_X - NO_2$

TABLE 3. ODDS RATIOS (95% CONFIDENCE INTERVAL) OF RESPIRATORY ILLNESS BY SCHOOL-BASED AMBIENT AIR POLLUTANT CONCENTRATIONS USING TWO-STAGE MODEL

		All Subjects		LTR Subjects		LTR Females		LTR Males	
		(*n* = 1,109)		(*n* = 871)		(*n* = 462)		(*n* = 403)	
	Exposure	OR	CI	OR	CI	OR	CI	OR	CI
Bronchitis*		n = 93/797		n = 79/635		n = 38/341		n = 41/291	
	NO_X	1.05	(1.01, 1.08)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.03	(0.98, 1.09)
	NO_2	1.02	(0.99, 1.06)	1.03	(1.00, 1.06)	1.04	(1.01, 1.08)	1.02	(0.98, 1.06)
	NO	1.05	(1.02, 1.09)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.04	(0.98, 1.10)
	PM_{10}	1.03	(0.99, 1.07)	1.02	(0.98, 1.07)	1.04	(1.01, 1.09)	1.01	(0.95, 1.06)
	$PM_{2.5}$	1.02	(1.00, 1.05)	1.03	(1.01, 1.05)	1.04	(1.02, 1.05)	1.02	(0.99, 1.05)
	BC	1.04	(1.00, 1.08)	1.05	(1.01, 1.08)	1.06	(1.02, 1.10)	1.03	(0.98, 1.08)
Asthma†		n = 101/705		n = 78/562		n = 42/297		n = 36/263	
	NO_X	1.04	(0.97, 1.11)	1.07	(1.00, 1.14)	1.17	(1.06, 1.29)	1.02	(0.93, 1.11)
	NO_2	1.02	(0.97, 1.07)	1.04	(0.98, 1.10)	1.09	(1.03, 1.15)	1.00	(0.94, 1.07)
	NO	1.05	(0.98, 1.12)	1.08	(1.00, 1.15)	1.19	(1.03, 1.36)	1.02	(0.94, 1.12)
	PM_{10}	1.02	(0.96, 1.09)	1.04	(0.97, 1.12)	1.09	(0.92, 1.29)	1.02	(0.94, 1.10)
	$PM_{2.5}$	1.00	(0.97, 1.04)	1.01	(0.97, 1.06)	1.06	(0.99, 1.15)	0.99	(0.95, 1.04)
	BC	1.02	(0.96, 1.09)	1.05	(0.99, 1.13)	1.12	(0.95, 1.33)	1.00	(0.93, 1.09)
Asthma (no outlier, school 5)†		n = 96/641		n = 73/507		n = 38/271		n = 35/233	
	NO_X	1.08	(1.00, 1.17)	1.10	(1.00, 1.20)	1.14	(1.02, 1.28)	1.07	(0.96, 1.19)
	NO_2	1.06	(0.99, 1.13)	1.07	(0.98, 1.17)	1.09	(0.97, 1.22)	1.05	(0.96, 1.16)
	NO	1.08	(1.00, 1.17)	1.09	(1.00, 1.19)	1.14	(1.03, 1.26)	1.07	(0.96, 1.18)
	PM_{10}	1.06	(0.97, 1.16)	1.08	(0.98, 1.19)	1.09	(0.96, 1.24)	1.08	(0.97, 1.19)
	$PM_{2.5}$	1.04	(0.96, 1.12)	1.03	(0.94, 1.13)	1.03	(0.91, 1.17)	1.03	(0.94, 1.14)
	BC	1.07	(0.98, 1.17)	1.09	(0.99, 1.19)	1.14	(1.02, 1.27)	1.06	(0.95, 1.18)

Definition of abbreviations: BC = black carbon; CI = confidence interval; LTR = long-term resident; OR = odds ratio; NO = nitric oxide; NO_X = total nitrogen oxides; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter of aerodynamic diameter 2.5 μm or less; PM_{10} = particulate matter of aerodynamic diameter 10 μm or less.

Odds ratios are calculated per IQR of average pollutant concentrations as follows: NO_X =14.9 ppb; NO_2 = 3.6 ppb; NO = 11.6 ppb; PM_{10} = 1.4 μg/m³; $PM_{2.5}$ = 0.7 μg/m³; BC = 0.15 μg/m³.

For hierarchical analyses of asthma in long-term residents (current address for 1 year or more), only 9 schools were included in the analysis; one school had no cases (due to low numbers and missing values).

* First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence.

† First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence; maternal history of asthma.

least 1 year at current residence) tended to increase the effect estimates slightly in relationship to asthma, especially when the sample was restricted to girls. Stratification by duration of residence or sex did not change the results for bronchitis. Results were similar when nonnormalized pollution values were used (data not shown).

We conducted additional sensitivity analyses, including (*1*) dropping the one school that was an outlier with respect to the proportion of Hispanic students (89% vs. 21–53% at other schools), (*2*) using a different definition for current asthma, and (*3*) stratifying bronchitis by a reported history of asthma. When the "outlier" school was dropped, the magnitude of the ORs for bronchitis did not change much, but the confidence intervals were wider. In the asthma analyses, dropping the outlier school resulted in similar or slightly greater effect estimates. Applying different questionnaire-based asthma definitions showed little change but slightly larger confidence intervals. After stratifying students by whether they also "ever" had asthma, the results suggested that those with a history of asthma were driving the results for bronchitis, but the sample size became too small to make clear inferences. Figures 2 and 3 depict the associations between BC and bronchitis and asthma.

DISCUSSION

To our knowledge, this is the first epidemiologic study in the United States to evaluate relationships between measured traffic-related pollutants and respiratory symptoms. For children residing at their current address for at least 1 year, we found modest but significant increases in the odds of bronchitis symptoms and physician-diagnosed asthma in neighborhoods with higher concentrations of traffic pollutants. These results are consistent with previous reports of positive associations between proximity to traffic and various respiratory outcomes (4–12). Furthermore, our findings were observed in a region with relatively clean air (low concentrations of ozone and PM) (*see* the online supplement for details). Although previous epidemiologic studies in the United States exploring chronic respiratory effects of air pollution in children have shown inconsistent results, this might be due in part to exposure misclassification, as these studies used air quality measurements conducted at single fixed-site monitors in each city (17, 18, 22, 23).

Our findings were robust to multiple sensitivity analyses using different questionnaire-based definitions of current asthma and wheezing in the past 12 months. The slight increase in effect estimates for associations between asthma after restricting the analysis to those with longer duration at current residence may be due to a reduction in exposure measurement error. Our study population was very mobile (23% had moved in the preceding 12 months, and only 32% had lived at the same address since before the age of 2 years).

We considered whether there might be bias due to nonresponse or self-reporting. We saw no significant difference in proportions of questionnaires returned in Spanish versus English by school, but there was a modest inverse correlation between pollution concentrations measured at each school and response rate. However, the response rate for individual classrooms within each school varied as well and appeared to depend on the willingness of teachers to encourage participation. Dropping the school closest to a freeway (which also had the highest measured pollutant concentrations, a high percentage of Hispanic students, and the lowest response rate) did not change the effect estimates for

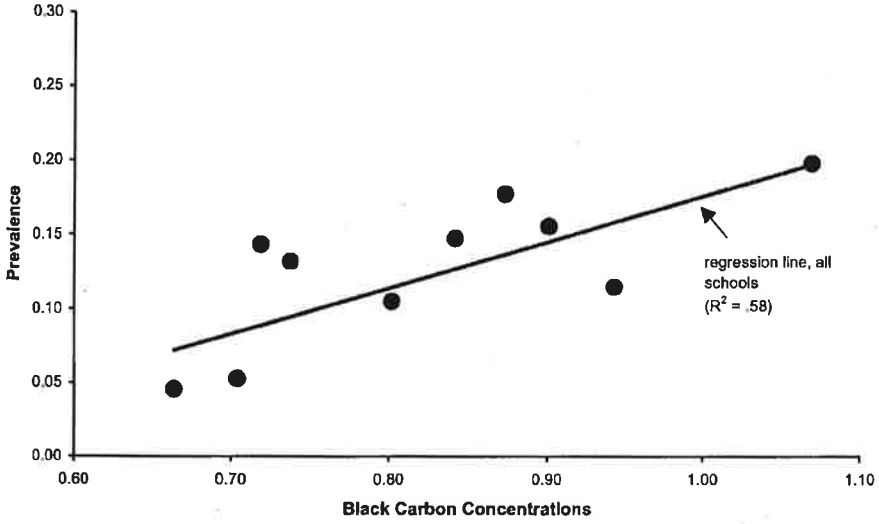

Figure 2. Adjusted school-specific bronchitis prevalence rates versus black carbon, long-term residents.

bronchitis and increased the estimates for asthma. This would suggest that knowledge of potential high traffic exposure probably did not affect parental reporting of the children's respiratory histories. This study was not undertaken in response to public concerns about traffic nor, at the time the study was conducted, was there much local interest in potential health hazards of proximity to traffic. Therefore, reporting and nonresponse biases were unlikely to have unduly influenced our results.

We found increased association with asthma (but not bronchitis) with exposure to traffic air pollutants for girls who had lived at their current addresses at least 1 year compared with boys (Table 3). Several investigators have also reported greater traffic-associated effect estimates for girls versus boys (7, 8, 10, 24, 25). Previous air pollution studies examining the sex-specific effects of air pollution on lung function and lung function growth have been mixed (26, 27). The reasons for the observations in our study are unclear and deserve attention in future studies.

Exposures

We found spatial variability in exposure due specifically to roads with heavy traffic within a relatively small geographic area for BC, NO_X, NO, and to a lesser extent NO_2. There was less variation in $PM_{2.5}$ across schools; this is consistent with previous observations that $PM_{2.5}$ is more likely to reflect regional air quality (2). The higher effect estimates with BC, NO_X, and NO compared with NO_2 and $PM_{2.5}$ suggest that primary or fresh traffic emissions may play an etiologic role in these relationships. Although NO_X, NO, and BC may serve as indicators of exposure to traffic-related pollutant mixtures, they may also act as etiologic agents themselves (28).

We found that downwind direction was an important determinant of increased exposure to traffic pollutants and that a simple traffic indicator (school location downwind and < 300 m from a major road) gave estimates of ORs similar to or greater than pollutant measurements in preliminary analyses using a one-stage model (data not shown). Within a geographic area with flat terrain and low-rise buildings, the direction of wind in relationship to the traffic source is the most important weather parameter. Other parameters important in air dispersion of traffic pollutants (e.g., atmospheric stability, wind speed, and surface topography) would be relatively similar at the different school sites.

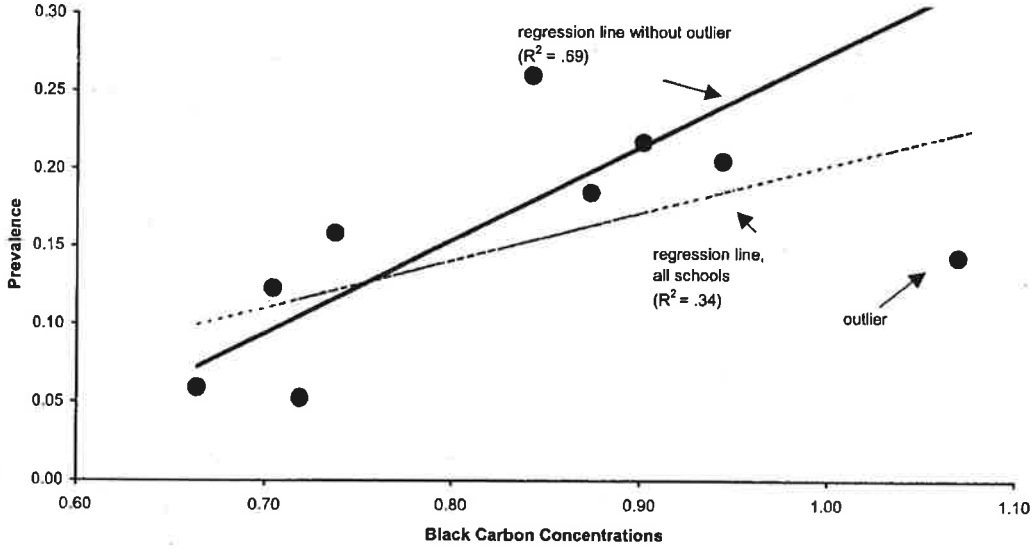

Figure 3. Adjusted school-specific asthma prevalence rates versus black carbon, long-term residents.

A simple single-stage logistic model using pollutant measurements also yielded positive associations between pollutants and symptoms with a much larger effect estimate and smaller confidence intervals.

We assumed that traffic-related pollutants measured at the neighborhood schools would be a good proxy for the children's overall exposure to such pollutants. Children attending the schools in this study generally lived within walking distance and did not use school buses. Therefore, pollutant concentrations in the children's neighborhoods probably tracked those at their schools. The most plausible exposure error in an urban setting would be that subjects who attend schools with very high traffic exposures from a nearby freeway would tend to have similar or lower home exposures, whereas children with low school exposures would tend to live in homes with similar or only slightly higher traffic exposures. This pattern of measurement error would tend to underestimate the association between exposure and outcome (29).

Alternatively, repeated daily exposures for 6–8 hours during the school year may themselves represent biologically important influences on some children's respiratory health, analogous to occupational exposures for susceptible adults. In a recent study of proximity to traffic and respiratory health, Janssen and colleagues found that effect estimates based on the school-to-highway distance were comparable or greater than those based on residence-to-highway distance (11).

The average measurements at each school were used to estimate long-term average traffic air pollutant concentrations. We measured pollutants at each of the 10 sites concurrently (to avoid concerns of week-to-week variability) in two different periods that reflect the major seasonal wind patterns for the area. We found that the rank order (relative values) of the schools did not vary from week to week or season to season, supporting the validity of this approach. Additionally, the NO_x and NO_2 concentrations at schools upwind or further from high traffic roads were similar to NO_X and NO_2 concentrations measured at the closest fixed-site monitor (21). Although there may have been some changes in the absolute traffic volume on major roads in recent years, the principal traffic patterns in the area have not changed. Thus, the relative values (rank order) of the site-specific pollutant concentrations measured in our study are likely to be representative of those in recent years.

The cross-sectional nature of our study design is a further limitation on causal inference, but we observed the same or modest increase in effect estimates for current asthma and bronchitis when we restricted our analysis to those who had lived at their present address for at least a year. Most studies on proximity to traffic and respiratory symptoms have been cross-sectional, and further longitudinal studies are needed to elucidate the role of traffic-related air pollution in the development and exacerbation of asthma and other respiratory symptoms.

Another limitation was that the exposures were assigned at the group level (n = 10); however, the multilevel analysis allows adjustment for individual confounders in the first stage of analysis. Moreover, in this respect, this study is comparable with other epidemiologic investigations (e.g., the Harvard Six Cities Study and the Children's Health Study in Southern California) (n = 12 communities). Another recent cross-sectional study of traffic-related air pollution and respiratory symptoms included 13 schools (18, 22, 23).

We also lacked information on indoor measurements of traffic-related pollutants. However, recent studies have found high correlations between personal exposures to NO_2 and traffic parameters (30). Others have found that indoor concentrations and exposure to soot (PM from diesel exhaust) is highly correlated with outdoor levels (2).

Other Covariates

Maternal asthma, household mold/moisture, pests, and chest illness before the age of 2 years were important explanatory variables in the final model for current asthma, consistent with previous studies (31–33). We explored whether current levels of traffic pollution could modify the risk of current asthma symptoms depending on past history of chest illness; however, there was not sufficient power to explore interactions based on early medical history. Race/ethnicity and indicators of socioeconomic status were not important predictors of health outcomes in our study. This may be due, in part, to our study design (i.e., the schools were selected to have relatively similar measures of socioeconomic status).

We did not find associations between exposure to environmental tobacco smoke and current asthma; the results of previous cross-sectional studies in school-aged children have been mixed (34). The prevalence of current household smokers in our study was small, however, limiting study power. It is possible that there is some underreporting of household smoking (7% in our study vs. 19% statewide). (35) Alternatively, a substantial portion of our study population was less acculturated Hispanics (30% of parents responded in Spanish), and only 3.6% of Hispanic households reported a history of maternal smoking. Other investigators have also observed very low smoking rates (less than 5%) among less acculturated Hispanics (B. Eskenazi, personal communication) (36). If underreporting does exist, it is possible that residual confounding might have affected our estimates of pollutant/respiratory health outcome relationships. However, the addition to the regression model of variables correlated with exposure to environmental tobacco smoke (e.g., socioeconomic status and race–ethnicity) did not change the pollutant effect estimates, suggesting that significant confounding by environmental tobacco smoke was not likely.

In summary, we found associations between traffic-related pollutants and asthma and bronchitis symptoms in the past 12 months in a highly urbanized region of the United States with good regional air quality, where local air pollution is dominated by vehicular sources. Although the cross-sectional study design, exposure assignment at the group level, small geographic area, and possible unmeasured covariates may limit the generalizability of the study, our findings are consistent with previous investigations in Europe and the United States (11, 14, 37). In addition, our results underscore the limitations of using central air monitoring stations for assigning population exposures. Concentrations of air toxics such as diesel exhaust particles or surrogates such as BC or soot should be more widely monitored. Measurement of personal exposures to traffic pollutants is not feasible in large population-based studies; the use of geographic modeling approaches to estimate exposures for individuals may be a good alternative (38). Future studies that can better characterize exposures to traffic pollutants, and their sources (i.e., diesel versus gasoline engines) will be important to understand better the public health impacts of motor vehicle emissions.

Conflict of Interest Statement: J.J.K. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; S.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; M.L. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.C.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; A.T.H. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.O. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

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