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September 12, 2008

Mr. Charlie Knox
Community Development Manager
Community Development Department
City of Benicia
250 East L Street
Benicia, CA 94510

Re: Addendum to Final Environmental Impact Report
Benicia Business Park
Comments by Benicia Unified School District
Our File No.: 1375.10308

Dear Mr. Knox:

The Benicia Unified School District ("District") has asked this office to provide the District's comments to the Addendum to the Final Environmental Impact Report ("Addendum" and "FEIR", respectively) for the proposed Benicia Business Park ("BBP") in the City of Benicia ("City"). The Addendum was prepared to analyze the environmental impacts of the changes proposed by the project sponsor (the "Mitigated Project") in contrast to the 2007 project ("2007 Project") that was analyzed in the FEIR.

GENERAL OBSERVATIONS

The District is concerned with the following issues that it has previously brought to the City's attention, which remain unaddressed in the Addendum. Additionally, the District raises issues that should have been addressed in the Addendum but were not:

- The District has previously communicated to the City its concerns regarding the BBP project. In or about March 4, 2007, Dirk Fulton, on behalf of the Board of Trustees provided extensive comments on the impacts to the District. We

ADVOCACY EXPERIENCE LEADERSHIP

reiterate those comments and provide an additional copy of that correspondence. The District believes that these issues have not been properly addressed in the FEIR or the Addendum.

- The Mitigated Project's impact on air quality for the District's students and staff in general, and most particularly those attending at working at the Robert Semple Elementary School ("Semple School"), 2015 East Third Street, Benicia, was not addressed in the Addendum.

- The proposed traffic calming measures and the potential impacts of the increased vehicular traffic created by the Mitigated Project, although reduced from the 2007 Project, remain at a significant threshold for the students and staff at Semple School and have not been addressed in any meaningful way in the Addendum. The District will provide more extensive comments on the traffic safety issues presented by the BBP in separate correspondence.

- The Mitigated Project's impact of increased noise level at Semple School was not addressed in the Addendum and as a result, it is unclear as to whether the City is asserting that the Mitigated Project no longer has noise level impacts at this location.

- The FEIR and the Addendum conclude that the Mitigated Project's impacts have been reduced to a less than significant level based in part on vaguely defined site uses. The generalized approach in the FEIR and the Addendum, which is appropriate for a "program" or "tiered" EIR, is inadequate for a project EIR. These documents do not rise to the level of analysis required under CEQA for a project EIR. Since there has been inadequate analysis of a "project" and its impacts, there cannot be defensible conclusions as to whether mitigation of impacts has been achieved until specific project data is available.

COMMENTS ON SPECIFIC SECTIONS OF THE ADDENDUM

A. The Addendum fails to analyze the cumulative impacts of increased vehicle traffic on air quality at Semple School. (Addendum Section 8.)

1. Section 8 c. Toxic Air Contaminants. The Addendum reaches the conclusion that no new sources of toxic air contaminants will be present as a result of the Mitigated Project. No evidence is provided by which this conclusion is reached. The District requests that this issue be addressed in a more comprehensive manner.

2. Section 8 d. Operational Emissions – CO Analysis. The Addendum concludes that since the Mitigated Project contains fewer square feet of industrial development that it will be expected to produce less CO and

not contribute cumulatively to CO concentrations. There is no reduction in the square footage of commercial development and the fact that the mix of industrial and commercial occupants has not been determined precludes the conclusion that less CO will be produced.

The District again provides the City a copy of the "Traffic–Related Air Pollution Near Busy Roads Study" (May 31, 2004), attached to the letter from Dirk Fulton, which indicates that children attending schools that are located within 500 feet of a road with more than 25,000 cars will suffer a 7% increase in respiratory problems associated with asthma and asthma precursor type symptoms. (American Thoracic Society, Traffic-related Air Pollution near Busy Roads (2004) American Journal of Respiratory and Critical Care Medicine, Vol. 170, pp. 520-526.)

The District reiterates its request, made March 9, 2007, for long term health assessments to ensure student health is maintained and not negatively impacted while attending Semple School.

3. Section 8 e. Demolition and Construction Emissions. The Addendum concludes that due to the lesser amount of grading (from 9 million cubic yards down to 4 million cubic yards of grading) proposed under the Mitigated Project that construction-period air quality impacts would be reduced to a less than significant level.

   The District asserts that stating that the reduction in size of the project in conjunction with the implementation of the mitigation measures is not sufficient analysis to support the conclusion reached in the Addendum.

4. Section 8 f. Long-term Emissions Impacts. The Addendum concludes that the Mitigated Project's size, not its design features, results in significant unavoidable emissions of ozone precursors.

   The District requests that the City refrain from certifying the Addendum based on the finding made in the FEIR that there will be a significant and unavoidable impact to the regional air quality. The FEIR states that "the potential [is small] for an individual project to significantly deteriorate regional air quality or contribute to significant health risk..." (FEIR at 269.) However, the Addendum states that it is the project's size that is the reason for its result in "significant unavoidable emissions of ozone precursors." (Addendum at 39.) The Addendum, in essence, contradicts the FEIR's finding that it is unlikely that regional air quality or health risk would worsen from the current condition due to emissions from an individual project. (FEIR at 269.) That statement is not analyzed in the FEIR and no supporting documentation is provided. While the

Addendum reaches the same result (approval of the Mitigated Project) as the FEIR does for the 2007 Project, the Addendum makes a contradictory assertion. The similarity between the two environmental documents is that neither contains appropriate analysis to support the conclusion reached.

B. The Addendum fails to analyze the impacts of increased noise on the learning environment at the Semple School. (Addendum Section 9.)

1. Section 9 c. Construction Period Impacts. The Addendum reaches the conclusion that the Mitigated Project will result in similar construction period noise impacts as would the 2007 Project. The FEIR has determined that the 2007 Project could have a significant impact for a short term along 2nd Street.

   The District reiterates its previous request (March 9, 2007) that further analysis of potential noise is conducted and that all proposed mitigations be required of the project sponsor.

2. Section 9 d. Operation Impacts. The Addendum concludes without analysis that the reduction in the size of the Mitigated Project and the implementation of Mitigation Measure NOI-2a, 2b, and 2c will ensure that noise impacts are reduced to a less than significant level. The Addendum acknowledges this while stating that transportation modeling data was unavailable at the time the Addendum was written. The District asserts that the Addendum, like the FEIR before it, has failed to properly analyze the cumulative impacts of the increased traffic noise on the students and staff at Semple School.

   The proposed Mitigation Measures (FEIR at 285), for the siting of a hotel with proposed outdoor activity (FEIR at 284) may be minimally adequate for a hotel however, the District believes that additional noise mitigation measures may be necessary for students and staff at the Semple School. The proposed and minimally acceptable mitigation measures include construction of a noise barrier, sound wall or sound wall/berm combination around all outdoor activity areas (FEIR at 285). Further, the City standards for "office/industrial facilities with areas that require good speech intelligibility... must be constructed to maintain an interior noise level of 45 dBA CNEL." (FEIR at 285.) To achieve this interior noise level, installation of noise-attenuated ventilation systems should be required of the project applicant to attain mitigation of the impacts of the Mitigated Project on the students and staff at Semple School.

The FEIR acknowledges that the City's General Plan prohibits noise levels in excess of 65dBA CNEL for schools (among other uses). However, no measurement is made of the location of the Semple School despite being recognized as a sensitive receptor under the General Plan. The Addendum concludes, without analysis, that implementation of the four-part Mitigation Measure will ensure that the Semple School is not negatively impacted by the increase in noise from vehicular traffic.

C. FEIR and Addendum include elements of a Program EIR while reaching conclusions as a Project EIR.

1. The CEQA Guidelines define a "program" as "a series of actions that can be characterized as one large project" and related, in pertinent part and applicable here, geographically and as logical parts in the chain of contemplated actions. (CEQA Guidelines, § 15168, subd. (a).) A program EIR is intended to be supplemented with later, more specific, detailed analysis.

    a. The District asserts that the City has not made clear whether the FEIR is intended to be a program EIR or a project EIR and as a result fails to provide the reader with sufficient information with which to read and evaluate the document.

    b. Several examples of ambiguous nature of the analysis in the Addendum are: 1) the variety of types and sizes of specific land uses that are assumed for inclusion in the Mitigated Project (Addendum at 6); 2) the lack of specific site plans for the proposed 857,000 square feet of commercial and industrial construction (Addendum at 7); and, 3) infrastructure, such as interior streets, that would not be connected until final phase of development adjacent to the road (Addendum at 9-10).

    c. CEQA Guidelines, § 15168, subd. (c)(5), states that "A program EIR will be most helpful in dealing with subsequent activities if it deals with the effects of the program as specifically and comprehensively as possible."

2. CEQA Guidelines, §15378, subd.(c) states that the term "project" refers to the activity which is being approved and which may be subject to several discretionary approvals by governmental agencies. Based on this, it is unclear exactly what has been approved by the City due to the

vague nature of the uses proposed and the wide-ranging possible uses that could be constructed within the BBP. Therefore, the District must reiterate its prior comments as to the lack of in-depth analysis of the impacts of noise, air pollution, and traffic on the students and staff at the Semple School.

D. Conclusion

For the foregoing reasons, we believe that the FEIR and the Addendum have failed to meet the requirements of CEQA in that they have not properly considered the impacts of the BBP on the students and staff of the District and in particular, the Semple School. In addition, the FEIR and the Addendum fail to provide adequate mitigation for the impacts on an elementary school, which is recognized as a sensitive receptor to both noise and air pollutants.

Very truly yours,

MILLER BROWN & DANNIS

Mark W. Kelley

Enclosure

cc: Board of Trustees, Benicia Unified School District
Elizabeth Patterson, Mayor, City of Benicia

Letter
A7

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March 9, 2007

Mr. Charles Knox
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Benicia, CA 94510

Re: Comments on Draft EIR for Seeno Project @ E. 2nd Street and Lake Herman Road (the "Project") –

Dear Mr. Knox,

I write as President of the Board of Trustees for the Benicia Unified School District ("BUSD") in connection with the Project as referenced above.

This letter addresses several student and community health safety issues raised by the draft Environmental Impact Report ("EIR") for the Project. These health and safety concerns are as follows:

Traffic-Generated Air Pollution. The EIR shows that 35% of the traffic generated by the Project will use E. 2nd Street, which abuts the Robert Semple Elementary School Health effects of traffic-generated air pollution are not thoroughly reviewed in the EIR (pages 265-268). This is so despite the fact that traffic flows on East 2nd Street greatly increase from 11,100 existing ADT to 37,900 ADT at build-out (See EIR Appendix D). The attached health study ("Traffic-Related Air Pollution Near Busy Roads" – see Attachment) shows that children attending schools that are located within 500 feet of a road carrying more than 25,000 cars will suffer a 7% increase in respiratory problems associated to asthma and asthma precursor type symptoms. Lung development in children who show no acute symptoms or respiratory distress can also be affected, with chronic daily exposure to such high levels of air pollutants: PM 2.5 and PM 10, e.g., tiny particle material tailpipe emissions, especially diesel, that can penetrate lung tissue. 1

Such impacts will directly and immediately affect our young elementary students playing on fields and working in classrooms. These impacts are material and possibly unmitigatable. Extensive long term health assessments are needed to assure student safety.

Traffic Control Issues. During the peak commute hours for the Project, BUSD parents will be dropping-off and delivering their children to and from Robert Semple. The Project, by itself, will generate 2,300 new trips during the morning school "drop-off" 2

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cont.

hour. (See EIR Figure IV 6-7). Yet, no changes are proposed for the intersections at East Second Street and merging arterials of Hillcrest, Seaview, East Tennys and Rankin Way. This adverse and potentially dangerous situation also occurs during the afternoon "pick-up" hour. Accordingly, further traffic studies are needed to guarantee that our safe routes to school are maintained. 2 cont.

Noise Impacts. Robert Semple is already impacted from noise by the 55,000 cars per day on I-780 and the existing 11,000 on East 2nd Street. No mitigations are proposed to protect the children from additional noise generated by this Project (EIR pages 279-282.). Noise studies and mitigations are required to protect the Robert Semple School site as a safe learning community. 3

Cumulative Impacts. The Project presents significant and material cumulative environmental impacts as to Robert Semple and the health and safety of its students. The school is fronted by I-780 (55,000 ADT) and is within ¼ mile of the Benicia Valero Refinery. Neither the air quality impacts of the adjacent freeway or of the refinery have ever been adequately studied by any government entity, including the City of Benicia. In combination with the impacts of the Project, these cumulative air, noise, and general safety impacts are weighty. Such impacts must be closely studied over extensive time periods to determine whether or not said impacts may be mitigated. Only then can the community be assured that the health of our children will remain protected. 4

Thank you for the opportunity to provide these comments. I look forward to your detailed response.

Very truly yours,

Dirk A. Fulton, President
Benicia Unified School District

DAF:bt

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cont.

ATTACHMENT

Traffic-related Air Pollution near Busy Roads

The East Bay Children's Respiratory Health Study

Janice J. Kim, Svetlana Smorodinsky, Michael Lipsett, Brett C. Singer, Alfred T. Hodgson, and Bart Ostro

Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland; and Atmospheric Sciences Department and Indoor Environment Department, Environmental Energy Technologies Division, Lawrence Berkeley National Laboratory, Berkeley, California

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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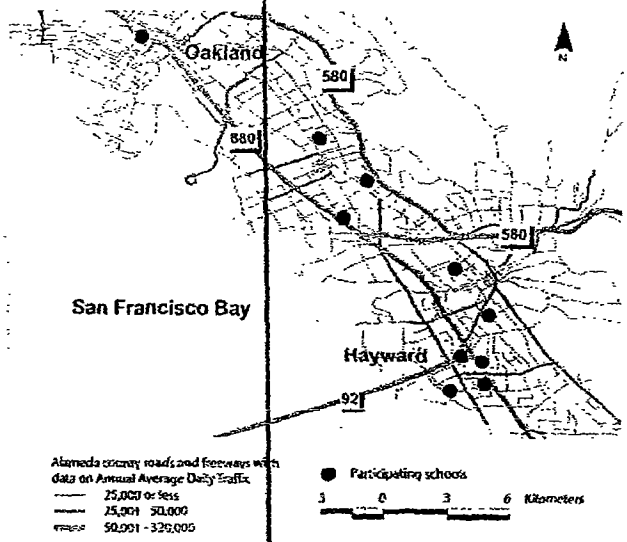

Figure 1. East Bay Children's Respiratory Health Study area. The study region is to the east and across the bay from the city of San Francisco.

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

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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} (μg/m³)	$PM_{2.5}$ (μg/m)	BC (μg/m³)	NO_X (ppb)	NO_2 (ppb)	NO§ (ppb)
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

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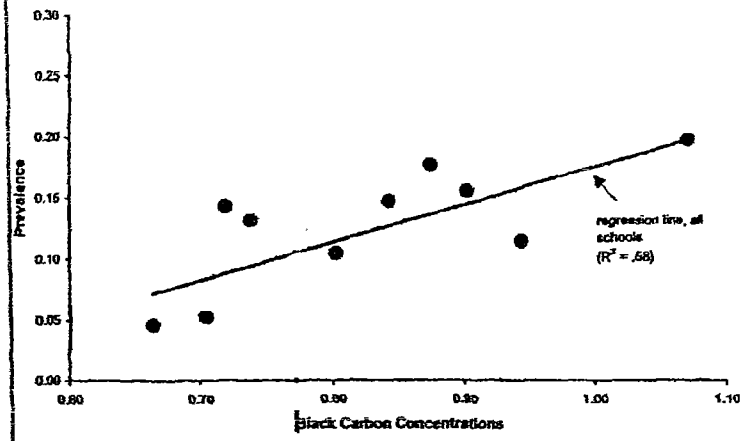

Figure 2. Adjusted school-specific bronchitis prevalence rates versus black carbon, long-term residents.

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.

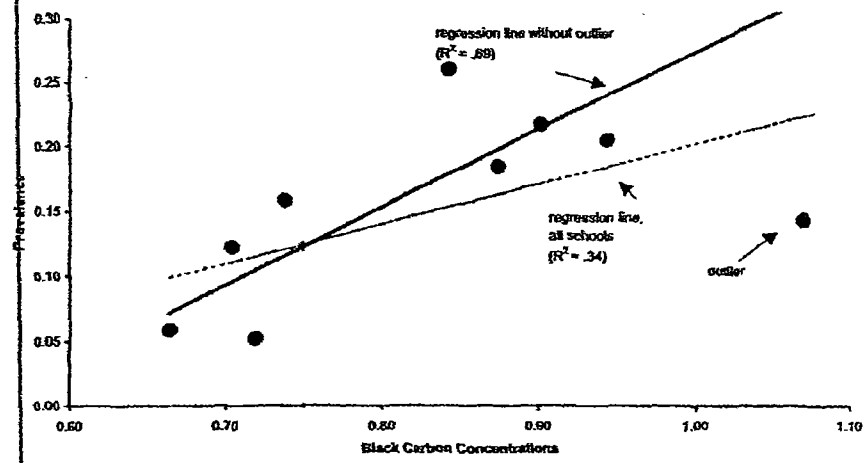

Figure 3. Adjusted school-specific asthma prevalence rates versus black carbon, long-term residents.

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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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COMMENTOR A7
Benicia Unified School District Board of Trustees
Dirk A. Fulton, President
March 9, 2007

A7-1: The Draft EIR addresses traffic emissions impacts in Section IV.H.2.b. (4) Operational Emissions – CO Analysis, and lists the 1-hour and 8-hour CO concentrations for Existing, Existing Plus Project, and Future (2025) Plus Project Conditions for 11 intersections in the project area in Tables IV.H-6, IV.H-7, and IV.H-8 (pages 261 to 264 of the Draft EIR).

The Future (2025) Plus Project peak hour CO concentrations shown in Table IV.H-8, show that the 1-hour CO concentrations at all analyzed intersections (i.e., the intersections that have the potential for the most severe project impacts) are well below State and federal standards. The Future Plus Project 1-hour CO concentrations at the East 2nd Street and Rose Drive and East 2nd Street and Military Street intersections would range from 4.2 parts per million (ppm) to 4.4 ppm, much lower than the State standard of 20 ppm. The 8-hour CO concentrations for these intersections on East 2nd Street would range from 4.3 ppm to 4.7 ppm for future plus project conditions. This too is well below the State standard of 9 ppm. Intersections in the vicinity of Robert Semple Elementary School would be expected to be exposed to similar or lower CO concentrations. The State standards used to evaluate impacts in the Draft EIR take into consideration all types of sensitive receptors, including schools and residences.

The *Air Quality and Land Use Handbook: A Community Health Perspective,* April 2005, by the California EPA and ARB, establishes guidelines for siting sensitive land uses near air pollutant sources. This handbook references the study *Traffic-Related Air Pollution and Respiratory Health: East Bay Children's Respiratory Health Study* by Kim, J., et al, 2004, for establishing these guidelines. State law restricts the siting of new schools within 500 feet of a freeway, urban roadways with 100,000 vehicles/day, or rural roadways with 50,000 vehicles/day, with some exceptions. The handbook recommends that new sensitive land uses not be sited within these boundaries. The modeled future plus project Average Daily Trips (ADT) would be 37,900 along East 2nd Street and 55,000 ADT on I-780. These traffic volumes are below the threshold set by the California EPA and ARB for locating schools near busy roadways. Therefore, the project would not be expected to expose sensitive receptors – including elementary school children at Robert Semple School – to hazardous levels of vehicle emissions, including emissions of CO and particulate matter.

A7-2: See Response to Comment A6-1.

A7-3: The Draft EIR addresses off-site traffic noise impacts on pages 279 to 283. As shown in Impact NOI-2 and in Tables IV.I-8 and IV.I-10, a significant traffic noise impact is identified for sensitive receptors along East 2nd Street under Existing Plus Project and Cumulative Plus Project conditions. The analysis shows that traffic noise levels 50 feet from the centerline of the outermost travel lane would increase by 3.5 dBA and 4.0 dBA respectively.

During review of the Draft EIR, minor typographical errors were identified in the traffic noise modeling values used in the analysis. However, after correction of these minor topographical errors, the noise modeling outputs, as well as the identified significant impacts, remain the same.

To further address the concerns for identified impacts for sensitive receptors located along the segment of East 2nd Street from I-780 to Rose Drive, the following changes will be made to pages 282 to 283 of the Draft EIR. The following revisions do not identify a new environmental impact of the project or introduce a new mitigation measure that would not be adopted by the project sponsor. Therefore, recirculation of the Draft EIR would not be required.

~~In summary, because there are no noise sensitive receptors within the noise contour areas for these roadway segments, the increased traffic noise levels would result in a less than significant impact for off-site receptors.~~

The segment of East 2nd Street from I-780 to Rose Drive could also experience a significant increase in traffic noise levels. The cumulative noise model for the project shows that traffic noise levels along East 2nd Street could increase to 71.6 dBA CNEL at 50 feet from the centerline of the outermost travel lane, a 4.0 dBA increase over the Cumulative Without Project scenario. Construction of a sound barrier at least 8 feet high along the property/right-of-way line would reduce the traffic noise impacts to sensitive receptors along this roadway segment to a less-than-significant level. The use of rubberized asphalt along the roadway segment, as an alternate mitigation measure, would also be effective in reducing traffic noise levels to a less-than-significant level. Rubberized asphalt reduces traffic noise through its porous and ductile qualities (refer to the following study for more information: Bollard and Brennan, Inc., 1999. *Report on the Status of Rubberized Asphalt., Traffic Noise Reduction in Sacramento County.* Prepared for Sacramento County Public Works Department.).

Mitigation Measure NOI-2c: For existing unprotected residential and school land uses along East 2nd Street from I-780 to Rose Drive, **one** (or more) of the following measures shall be implemented:

- A sound barrier at least 8 feet high shall be constructed along the property/right-of-way line of sensitive receptors along this roadway segment; or

- Rubberized asphalt shall be used to resurface the entire identified roadway segment. (LTS)

Tables IV.I-8, IV.I-9, and IV.I-10 are revised as follows:

Table IV.I-8: Existing Plus Project Traffic Noise Levels

Roadway Segment	ADT	Center line to 70 CNEL (feet)	Center line to 65 CNEL (feet)	Center line to 60 CNEL (feet)	CNEL (dBA) 50 feet from Centerline of Outermost Lane	Increase from Baseline Conditions
East 2nd St. - Lake Herman Rd. to Park Rd.	19,300	~~54~~ 56	116	248	~~69.4~~ 68.7	~~5.5~~ 4.8
East 2nd St. - Park Rd. to Industrial Way	11,700	< 50[a]	~~83~~ 84	178	~~67.3~~ 66.5	~~2.9~~ 2.1
East 2nd St. - Industrial Way to Rose Dr.	31,700	77	161	345	70.8	6.3
East 2nd St. - Rose Dr. to I-780 WB On Ramp	25,000	66	138	295	69.8	3.5
East 2nd St. - I-780 EB On Ramp to Military St.	13,300	< 50	~~60~~ 62	~~128~~ 129	~~65.1~~ 64.4	~~0.0~~ -0.7
Lake Herman Rd. - East 2nd St. to Reservoir Rd.	13,800	< 50	~~93~~ 94	199	~~68.0~~ 67.2	~~10.3~~ 9.5
Lake Herman Rd. - Reservoir Rd. to Sky Valley Rd.	15,900	< 50	102	218	68.6	7.2
Lake Herman Rd. - Sky Valley Rd. to Columbus Pkwy	16,000	< 50	102	219	68.6	7.0
Reservoir Rd. - Lake Herman Rd. & East 2nd St.	11,200	< 50	81	173	67.1	7.7
Industrial Way - East 2nd St. to Park Rd.	7,400	< 50	< 50	~~87~~ 88	~~62.6~~ 61.8	~~3.7~~ 2.9
Park Rd. – East 2nd St. to Industrial Way	2,400	< 50	< 50	< 50	57.7	5.4
Park Rd. - Industrial Way to Bayshore Rd.	13,300	< 50	60	128	65.1	2.5

[a] Traffic noise within 50 feet of the roadway centerline requires site-specific analysis.
Source: LSA Associates Inc., September 2006

Table IV.I-9: Cumulative Without Project Traffic Noise Levels

Roadway Segment	ADT	Center line to 70 CNEL (feet)	Center line to 65 CNEL (feet)	Center line to 60 CNEL (feet)	CNEL (dBA) 50 feet from Centerline of Outermost Lane
East 2nd St. - Lake Herman Rd. to Park Rd.	7,800	< 50[a]	64	136	~~67.7~~ 65.5
East 2nd St. - Park Rd. to Industrial Way	8,300	< 50	66	142	~~67.7~~ 65.8
East 2nd St. - Industrial Way to Rose Dr.	10,100	< 50	77	162	~~67.8~~ 65.9
East 2nd St. - Rose Dr. to I-780 WB On Ramp	15,000	< 50	99	210	~~69.5~~ 67.6
East 2nd St. - I-780 EB On Ramp to Military St.	18,000	< 50	73	157	~~68.4~~ 66.4
Lake Herman Rd. - East 2nd St. to Reservoir Rd.	1,800	< 50	< 50	52	~~61.4~~ 59.1
Lake Herman Rd. - Reservoir Rd. to Sky Valley Rd.	4,300	< 50	< 50	92	~~65.2~~ 62.9
Lake Herman Rd. - Sky Valley Rd. to Columbus Pkwy	4,500	< 50	< 50	94	~~65.3~~ 63.1
Reservoir Rd. - Lake Herman Rd. & East 2nd St.	2,700	< 50	< 50	67	~~63.0~~ 60.9
Industrial Way - East 2nd St. to Park Rd.	4,500	< 50	< 50	63	~~62.4~~ 60.4
Park Rd. – East 2nd St. to Industrial Way	900	< 50	< 50	<50	~~55.4~~ 53.4
Park Rd. - Industrial Way to Bayshore Rd.	10,500	< 50	52	~~11~~ 110	~~66.1~~ 64.1

[a] Traffic noise within 50 feet of the roadway centerline requires site-specific analysis.
Source: LSA Associates Inc., September 2006

Table IV.I-10: Cumulative Plus Project Traffic Noise Levels

Roadway Segment	ADT	Center line to 70 CNEL (feet)	Center line to 65 CNEL (feet)	Center line to 60 CNEL (feet)	CNEL (dBA) 50 feet from Centerline of Outermost Lane	Increase from Cumulative Conditions
East 2nd St. - Lake Herman Rd. to Park Rd.	~~26,800~~ 21,000	~~57~~ 59	~~122~~ 123	263	~~69.8~~ 69.0	~~4.5~~ 3.5
East 2nd St. - Park Rd. to Industrial Way	~~18,500~~ 16,500	< 50[a]	~~104~~ 105	224	~~68.8~~ 68.0	~~3.0~~ 2.2
East 2nd St. - Industrial Way to Rose Dr.	~~40,200~~ 36,100	83	176	377	71.4	5.5
East 2nd St. - Rose Dr. to I-780 WB On Ramp	~~37,300~~ 37, 900	86	181	389	71.6	4.0
East 2nd St. - I-780 EB On Ramp to Military St.	~~28,100~~ 18,000	< 50	~~73~~ 75	~~157~~ 158	~~66.4~~ 65.7	~~0.0~~ -0.7
Lake Herman Rd. - East 2nd St. to Reservoir Rd.	~~15,500~~ 13,800	< 50	~~93~~ 94	199	~~68.0~~ 67.2	~~8.9~~ 8.1
Lake Herman Rd. - Reservoir Rd. to Sky Valley Rd.	~~20,000~~ 17,100	< 50	107	229	68.9	6.0
Lake Herman Rd. - Sky Valley Rd. to Columbus Pkwy	~~20,300~~ 15,000	< 50	98	210	68.3	5.2
Reservoir Rd. - Lake Herman Rd. & East 2nd St.	~~2,700~~ 12,000	< 50	84	181	67.4	6.5
Industrial Way - East 2nd St. to Park Rd.	~~11,300~~ 8,700	< 50	< 50	~~97~~ 98	~~63.3~~ 62.5	~~2.9~~ 2.1
Park Rd. – East 2nd St. to Industrial Way	~~3,100~~ 2,100	< 50	< 50	< 50	57.1	3.7
Park Rd. - Industrial Way to Bayshore Rd.	~~22,700~~ 13,800	< 50	62	132	65.3	1.2

[a] Traffic noise within 50 feet of the roadway centerline requires site-specific analysis.
Source: LSA Associates Inc., September 2006

A7-4: See Responses to Comments A7-1 and A7-3. The project would not result in significant cumulative traffic, noise, and air quality-related impacts beyond those identified in the Draft EIR.

From: Anne Cardwell
To: Charlie Knox; Council; Heather McLaughlin
Date: 9/28/2008 12:35:47 PM
Subject: Fwd: For The Record: to Deny Seeno Project

>>> Martin Duvall <martyduvall@sbcglobal.net> 9/27/2008 9:21 AM >>>
Dear City Leaders

Benicia, my lifelong family home, has always benefited from a small town charm.

Somehow, I feel a bit less charmed when trying to get in or out of downtown Benicia at certain peak traffic times at East Second Street - sometimes waiting for 2 or even 3 street light changes to get off of Military East toward the 780 Freeway and Industrial Park. Gridlock? In Benicia?

It truly exists and is soon to get worse when Seeno cavalierly chooses to ignore traffic mitigation on both East second Street and Interstate 780. Imagine the residents on that street that feel this effect all day long? Imagine that corridor when Seeno's proposed business park increases traffic by it's projected numbers?

I am not a traffic crusader, but I point this out as just one glaring issue that effects the everyday quality of life in our town with respect to the Seeno project.

Quality of Life. Far and away, this is the reason most often cited by citizens and visitors alike for appreciating Benicia's many attributes. Quality of Life is a promise many if not all of our elected officials promised to promote and protect.

I believe in property rights and free enterprise, but City's do have domain over what types of developments they allow. I'm no expert on this, but our City has a responsibility to direct developers to accommodate Benicia's requirements. Seeno has a right to develop their property. But to further encroach on the already conspicuous traffic concerns on one of the most crucial traffic arteries in town should be concern enough all on its' own to deny Seeno's project, regardless of all the other reasons that can and have been cited.

There has been much written about the Green Gateway Group's proposal for an alternative project. This citizen's initiative is a first step to aid and entice our City's leaders in doing what is right and required in this case.

Martin E Duvall
9A McKay Way
Benicia, CA 94510
707.344.7096

CC: Jayne York

September 30, 2008

Dear City Council Members,

In the Statement of Overriding Conditions that LSA Associates prepared for the Benicia Business Park EIR, they state the following:

4.1 AIR QUALITY
Impact AIR-2: Long-term project-related "regional emissions" would exceed the BAAQMD thresholds of significance for ozone precursors.
[I might add, there is nothing "regional" about the direct impact those ozone precursors would have on increased levels of ozone here in Benicia.]

They go on to propose Mitigation Measure AIR-2:
"The *BAAQMD CEQA Guidelines* identifies potential mitigation measures for various types of projects. The following are considered to be feasible and effective in further reducing vehicle trip generation and resulting emissions from the project."
They then list several mitigations that they will implement which are suggested by the *BAAQMD CEQA Guidelines*, such as benches and shelters for transit facilities, bicycle lanes, sidewalks, and a Trip Demand Management measure such as a ride-matching program.

Following that list of mitigations, they state that those measures will not be sufficient to reduce ozone precursors to levels below BAAQMD significance threshold. They claim "there is <u>no mitigation available</u> with currently feasible technology to reduce the project's regional air quality impacts to a less than significant level." And based on that claim of no other mitigation available, they are asking you to this extremely harmful air quality impact by signing the Statement of Overriding Considerations.

But an important mitigation measure which is suggested by the *BAAQMD CEQA Guidelines* is missing from their list of measures that they will implement. That is the provision of a "shuttle service to transit stations" and the establishment of "mid-day shuttle service from worksite to food service establishments." This is a mitigation that is available and feasible and very effective. Lawrence Berkeley Lab has been using this measure for years in Berkeley (ALL of it paid for by the Lab, including the wages for the bus drivers). It has dramatically reduced vehicle traffic at the Lab because it its, free, frequent, convenient, and because parking space for private cars is extremely limited.

Please note that our General Plan program 4.10.B states that the City shall <u>require</u> that projects with significant air quality impacts must include <u>all</u> feasible mitigation measures <u>needed</u> to reduce impact to less than significant levels.

Since Discovery Builders is not willing to include this mitigation measure, please do not sign the Statement of Overriding Considerations !

Thank you,
Norma Fox

[You can see the *BAAQMD CEQA Guidelines* at the following link; of particular note is Table 15, on pg.65… http://www.baaqmd.gov/pln/ceqa/ceqa_guide.pdf]

Sept. 30, 2008

Dear City Council Members,

The EIR Addendum to the proposed Benicia Business Park states that the project will cause "significant unavoidable air quality impacts that cannot be reduced to a less-than-significant level" by any of the proposed mitigation measures. Specifically, it will cause "long-term project-related emissions from traffic which would exceed the BAAQMD thresholds of significance for ozone precursors." (Ozone is a serious public health risk, and Benicia already has higher levels of ozone than most communities.)

Because the applicant claims they cannot provide mitigations to reduce those emissions below BAAQMD thresholds, CEQA law will require you to sign a Statement of Overriding Considerations if you choose to approve the project. You must affirm in this statement that the economic benefit of the project to the city's tax base and job supply (75% jobs for out-of-town employees) is so great that it "outweighs the unavoidable adverse environmental effects."

In addition, CEQA law requires that your claims of significant economic benefit "must be based on substantial evidence in the administrative record." But the problem is, there has been NO economic analysis performed on the revised 2008 version of this project that would provide that substantial evidence and reliably quantify those glowing economic claims.

I know that all of you are deeply committed to your fiduciary responsibilities and to preserving Benicia's health and quality of life, so I can't understand why any of you would consider signing off on that Statement, and permanently impairing our air quality and public health, when its claim of significant economic benefit is based on sheer speculation.

In fact I was surprised and dismayed that the Council has never once spent any time reviewing the now outdated (2005) economic analysis (which was based on a former and much larger version of the project), and asking the developer to explain and defend its sources of data, assumptions, and projections in the light of today's dire economic climate.

Before signing that Statement, I hope that you will require Seeno/Discovery Builders to produce a new economic analysis to support its glowing revenue claims, one based on their current (smaller) version of the project and on current economic data and projections. Or better yet, since the developer negligently omitted an updated economic analysis from the documentation for the current project, I hope you will simply refuse to sign the unsupported Statement of Overriding Considerations, and thereby deny approval of this poorly designed project.

Sincerely,
Norma Fox

PS. Project denial would not be the end of the project. Instead, it would finally open the door and allow a fresh start for the developer to work with the City and the community on a new vision for the project, clearly outlined in a detailed Specific Plan. Many details of the community's vision have already been thoroughly described throughout the approval process by many individuals and groups (most recently in a detailed document posted at www.greengatewaygroup.org), so the developer would not have to start over with no ideas and a blank slate.

Submitted by Marilyn Bardet on September 30, 2008

Air Pollution and Children's Health

A fact sheet by
Cal/EPA's Office of Environmental Health Hazard Assessment and
The American Lung Association of California.

In the past 30 years, state and federal air-quality programs have made great progress in reducing air pollution. However, there has been an increasing awareness in recent years that children may be more susceptible than adults to the harmful effects of air pollutants.

The California Environmental Protection Agency's Office of Environmental Health Hazard Assessment (OEHHA) assesses health risks from environmental contaminants. OEHHA currently is reviewing whether the state's ambient air quality standards are adequate to protect the health of infants and children and is working to identify toxic air contaminants that may cause infants and children to be especially susceptible to illnesses. 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 air pollution and children's health.

Why may children be at greater risk than adults from air pollution?

In many cases, children may have greater exposure than adults to airborne pollutants. Infants and children generally breathe more rapidly than adults, which increases their exposure to any pollutants in the air. Infants and children often breathe through their mouths, bypassing the filtering effect of the nose and allowing more pollutants to be inhaled. Children generally spend significantly more time outdoors than adults, especially during summer months when smog levels are highest.

Children are often more susceptible to the health effects of air pollution because their immune systems and developing organs are still immature. For example, lead that is inhaled is more easily deposited in the fast-growing bones of children. Irritation or inflammation caused by air pollution is more likely to obstruct their narrower airways. It may also take less exposure to a pollutant to trigger an asthma attack or other breathing ailment due to the sensitivity of a child's developing respiratory system. Exposure to toxic air contaminants during infancy or childhood could affect the development of the respiratory, nervous, endocrine and immune systems, and could increase the risk of cancer later in life.

What are the major kinds of air pollutants and their impacts on children?

"Criteria" Air Pollutants

Several common air pollutants are regulated under the state and federal Clean Air Acts and are known as "criteria" air pollutants. Two of the most widespread criteria pollutants are particulate matter (PM) and ozone. PM consists of microscopic particles less than one-seventh the width of a human hair. These particles come from a variety of both manmade and natural sources, such as diesel engines, smoke from fireplaces as well as forest and agricultural fires, and dust from tilled farmland. PM can bypass the body's natural defenses and penetrate deep into the lungs. The elderly, children and people with existing respiratory or cardiac diseases are considered to be especially sensitive to the harmful effects of PM. Recent studies suggest that PM may exacerbate asthma and cause coughs and other respiratory symptoms in children. Recent studies also suggest that prolonged exposure to PM may also affect the growth and functioning of children's lungs. Researchers found that as children grow up in smoggier areas, there is a notable lag in lung function growth.

Ozone is the major component of urban smog. It is formed by chemical reactions in the atmosphere involving sunlight and various gases in motor vehicle exhaust and industrial emissions. Ozone is a powerful respiratory irritant that can cause lung inflammation, transient decreases in lung function, shortness of breath, chest pain, wheezing, coughing and exacerbation of respiratory illnesses such as asthma. Long-term and repeated ozone exposures may lead to chronically reduced lung function.

OEHHA provides detailed analyses of health information on PM, ozone and other common pollutants to the California Air Resources Board (ARB), which sets ambient air quality standards for those pollutants. These air quality standards have been established at levels that are intended to protect the health of all Californians. Unfortunately, PM and ozone levels in most urban areas of California frequently exceed the ambient air quality standards. ARB and local air districts operate regulatory programs under state and federal requirements to reduce airborne levels of these pollutants to the ambient air quality standards.

Other "criteria pollutants" include nitrogen dioxide, carbon monoxide, lead, sulfur dioxide, sulfates and hydrogen sulfide.

ARB is sponsoring a major 10-year study of the effects of air pollution on children's health. Information on this study is available at ARB's web site at http://www.arb.ca.gov/research/abstracts/98-320.htm.

Toxic Air Contaminants

California also regulates the emissions of other pollutants, known as toxic air contaminants, which may pose a present or potential hazard to human health or contribute to an increase in deaths or serious illnesses. OEHHA provides assessments of the health risks from various toxic air contaminants to ARB, which can enact control measures designed to reduce the exposure of Californians to these contaminants. More than 200 chemicals are currently listed as toxic air contaminants in California.

Many toxic air contaminants are present in motor vehicle exhaust and industrial emissions, and are formed from the combustion of other chemicals. Among these byproducts of combustion are dioxins and polycyclic organic matter (POM), which may

affect the development of the fetus and increase cancer risks later in life. Particles found in diesel exhaust may make children more susceptible to allergies and asthma. Other toxic contaminants have numerous sources. Acrolein, which may exacerbate asthma, is found in motor vehicle exhaust, tobacco and wood smoke, some industrial emissions, and is also formed in the atmosphere from chemical reactions involving other pollutants. Some toxic air contaminants, such as lead, are naturally occurring in the environment. Lead has been banned as an additive in gasoline and household paint but is present in some industrial emissions. It can cause developmental problems and harm the central nervous system.

What is California doing to improve health protection for children?

The Legislature in 1999 approved the Children's Environmental Health Protection Act (Senate Bill 25), authored by Senator Martha Escutia, which seeks to ensure that California's air quality programs protect the health of infants and children. The Act requires ARB, in consultation with OEHHA, to review all ambient air quality standards to determine whether they adequately protect the health of the public, including children. The Act also requires OEHHA to identify toxic air contaminants that may cause infants and children to be especially susceptible to illness, and it requires ARB to determine the adequacy of existing control measures for toxic air contaminants or the need for new control measures to protect the health of the public, particularly infants and children.

The initial stage of the ambient air quality standards review was completed in December 2000. ARB and OEHHA concluded that PM and ozone may cause health effects in children even at levels meeting the state's ambient air quality standards. The amount of time children play outdoors and their higher breathing rates are some of the reasons why children may be more sensitive to these pollutants than adults. The review also found evidence that levels of nitrogen dioxide (a pollutant in motor vehicle exhaust and many kinds of industrial emissions) that meet the ambient air quality standard may harm asthmatic children.

OEHHA completed a detailed review of the PM standards in May 2002. Based on this review, OEHHA recommended, and ARB adopted, stricter new PM standards in June 2002. (The report containing the results of the PM review and the new standards can be accessed at www.oehha.ca.gov/air/toxic_contaminants/PM10notice.html#may). OEHHA is conducting a detailed review of the ozone standard and is developing recommended revisions to the standard for ARB's consideration. After this is completed, OEHHA will review the nitrogen dioxide standard.

ARB and local air districts may have to develop new regulations to reduce emissions and ultimately reduce airborne levels of these pollutants to comply with the new PM standards and any future new standards for ozone and nitrogen dioxide.

The Act also requires OEHHA to identify toxic air contaminants that may cause infants and children to be especially susceptible to illnesses. ARB will review and, if necessary, revise or adopt any control measures needed to reduce the public's exposure to these contaminants. The Act required OEHHA to identify up to five contaminants in 2001. These contaminants - dioxin, lead, POM, diesel exhaust particles, and acrolein - are briefly discussed in the previous section. More detailed information is available at www.oehha.ca.gov/air/toxic_contaminants/SB25finalreport.htm.

OEHHA is developing guidelines for use in evaluating the adequacy of existing air toxics regulations. Beginning in 2004, OEHHA will annually evaluate selected toxic air contaminants and identify unhealthful levels of exposure to these contaminants. OEHHA will use these evaluations beginning in 2005 to annually update the list of toxic air contaminants that impact infants and children.

The Act also requires ARB to evaluate the adequacy of its current air-quality monitoring program to determine children's exposure to air pollutants, and it created the Children's Environmental Health Center within the California Environmental Protection Agency to coordinate Cal/EPA's activities and provide advice to the Governor and Cal/EPA Secretary on matters of children's environmental health.

In addition to complying with the requirements of the Children's Environmental Health Protection Act, OEHHA is taking two other steps in the area of children's health protection. OEHHA is developing improvements to scientific methods used to gauge cancer risks that children may face from exposure to contaminants in the environment. Also, OEHHA is refining methods to assess health risks from contaminants that may conceivably exist at proposed and existing school sites. These two projects are intended to improve the ability of scientists and regulators to make decisions that protect children from contaminants in the air, water, soil and food as well as elsewhere in the environment.

These efforts represent a commitment by the State of California to ensure that children have the opportunity to grow up in a healthy environment.

Revised November 2003

Submitted by Marilyn Bardet on September 30, 2008

Fact Sheet

April 2002

California Environmental Protection Agency
Air Resources Board

Fresno Asthmatic Children's Environment Study (F.A.C.E.S.)

The Fresno Asthmatic Children's Environment Study, which began in 2000, is a large epidemiological study of the effects of air pollution on children with asthma. About 300 asthmatic children who reside in the Fresno area of the Central Valley of California will be enrolled in the study. The overall goal of this study is to determine the effects of different components of particulate matter (PM), in combination with other ambient air pollutants, on the natural history of asthma in young children. The study is sponsored by the California Air Resources Board and conducted by investigators at the University of California, Berkeley.

Importance of the Fresno Asthmatic Children's Environment Study

- The information provided by the study will help the Air Resources Board (ARB) protect public health. The ARB sets California's ambient air quality standards to protect people who are most sensitive to air pollution.
- Children may be more strongly affected by air pollution because their lungs and bodies are still developing. Understanding the effects of air pollution on children with asthma is essential for setting health standards protective of sensitive populations.

The Pollutants Studied

- Particulate matter
- Criteria air pollutants: NO_x, SO_2, CO, ozone
- Polycyclic Aromatic Hydrocarbons (PAHs)
- Volatile Organic Compounds (VOCs)
- Environmental Tobacco Smoke
- Allergens

The Information Gathered by the Study

The study consists of a variety of measurements taken over the course of five years. Measurements taken include skin testing for allergies, lung function testing, and extensive questions about the child's health and home environment. Research staff will also visit the child's home to collect indoor air and dust samples. Children will keep a journal of activities and time spent in different locations throughout the day, as well as symptoms and medication use. Portable spirometers will be used to measure lung function at home.

For More Information

Please contact the ARB's Public Information Office at (916) 322-2990, or visit our web site at http://www.arb.ca.gov/research/faces/faces.htm. You may obtain this document in an alternative format by contacting our ADA coordinator at (916) 322-4505 (voice); (916) 324-9531 (TDD, Sacramento area only); or (800) 700-8326 (TDD, outside Sacramento).

Submitted by Marilyn Bardet on September 30, 2008

This is the html version of the file
http://www.co.missoula.mt.us/ENVHealth/AirQ/AirQualityTopics/TransportationAndBuiltEnv/Idling%20Fact%20Sheet%2012-17-04.doc.
Google automatically generates html versions of documents as we crawl the web.

Idling Fact Sheet

Compiled by the Missoula City-County Air Quality Advisory Council

Reducing idling reduces air pollution. Vehicle exhaust contains at least 21 air toxics which, by definition, are hazardous to human health. Major pollutants from automobiles include hydrocarbons, nitrogen oxides, carbon monoxide, and particulate matter, all of which have significant health and environmental impacts. Emissions from **idling** vehicles can be as much as 20 times greater than those from one traveling at 32 mph. Many communities in the United States and Canada have or are considering ordinances that restrict excessive vehicle **idling** in order to improve air quality and protect citizens' health.

Use the 30-second rule to save gas and reduce emissions. Contrary to the commonly held misconception, frequent restarting has little impact on engine components such as the battery and starter motor. Your savings in fuel will easily offset the cost of what little wear does result. Remember, when you are idling, you are getting zero miles per gallon. The U.S. EPA's website states, "You will save gas by turning the engine off and restarting it again if you expect to idle for more than 30 seconds. You will also prevent pollution by preventing long idles. Try parking your car and going into restaurants, banks, and the like instead of idling in drive-up lanes." When you must wait in a drive thru, turn your engine off.

The best way to warm up your vehicle is to drive it. It's a common misconception that **idling** for several minutes is the best way to warm up a vehicle. Not only is extended **idling** unnecessary, but many parts of the vehicle—including wheel bearings, tires, and the suspension system—only warm up once the vehicle is moving. You only need to idle long enough to get the oil circulating—about 30 seconds—before driving away, and it's a good idea to avoid high speeds and fast acceleration until the engine temperature rises. Modern diesel engines also need only a short engine warm up times. To keep windows from fogging up, clear snow from the

air intake on top of your hood (before you start the engine) and open a window slightly as soon as you get in the car. When temperatures are in the teens or colder, use an engine block heater for 2-4 hours to help your car start more easily, get your defroster working faster, improve your winter gas mileage as well as reduce air pollution (see Engine Block Heater Fact Sheet for more details).

Reducing idling reduces wear and tear on your engine and saves money. Idling creates wear and tear on your engine because fuel doesn't combust completely, and some fuel residue can condense on cylinder walls. Also, excessive idling can cause condensation to form in the exhaust, which may result in corrosion and reduced lifespan of the exhaust system. Idling for 10 minutes a day uses an average of 26 gallons of gas a year. At $2 a gallon, a driver could save over $50 a year in gasoline costs just by turning off the engine.

Unattended idling vehicles are unsafe, illegal, and vulnerable to theft. Not only does common sense tell us that leaving a running vehicle unattended can be dangerous, but it is also illegal to do so. Specifically, Missoula's Municipal Code on Unattended Motor Vehicles (10.14.050) states that, "No person driving or in charge of any motor vehicle except a licensed delivery truck or other delivery vehicle, shall permit it to stand unattended without first stopping the engine, locking the ignition and removing the key." Unattended **idling** vehicles are not only unsafe and illegal, they are an open invitation to easy theft!

Some other things you can do to improve air quality: (1) Plan ahead to combine errands or avoid the trip altogether; (2) bike, walk, ride the bus, or carpool; (3) maintain your car regularly; (4) drive smoothly and avoid sudden throttling.

For more information, visit the following websites or call the Health Department at 258-4755.

Missoula County Environmental Health Division—http://www.co.missoula.mt.us/EnvHealth

Missoula Municipal Code—http://www.ci.missoula.mt.us/cityclerk/city_code.htm

"Idling and climate change go hand in hand," Natural Resources Canada, Office of Energy Efficiency— http://oee.nrcan.gc.ca/transportation/**idling**/issues/why-**idling**-problem.cfm?attr=8

US EPA—http://www.epa.gov

"Your car and clean air: What YOU can do to reduce pollution" (**idling** info on p.3)—http://www.epa.gov/otaq/consumer/18-youdo.pdf

Mobile source emissions: Past, present, and future (Pollutants): http://www.epa.gov/otaq/invntory/overview/pollutants/index.htm

Mobile source air toxics: http://www.epa.gov/otaq/toxics.htm

National Safety Council, Environmental Health Center, "Environmentally friendly maintenance and repair"—http://www.nsc.org/ehc/mobile/mainrepa.htm

Submitted by Marilyn Bardet on September 30, 2008

Air and Breathing

We prefer Clean Air, Clean Water and Healthy Food

About Air and Breathing

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Air and Breathing Centers

The Way of Breath
Atmosphere
Air Quality
Cars and Trucks
Air Pollution
Climate Change
Airborne Diseases
Air Quality inside Buildings
Environment and Medicine
Allergy

Food and Lung Disease
The Alpha Nutrition Program

You are at Alpha Online, a service of
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Ads by Google
Chemical
Toxins Health
Human Toxins
Noise Pollution
Air Monitoring

Automotive Exhaust Chemicals: disease causing effects

A short list of the likely pathogens in car exhaust:

- Carbon Monoxide
- Nitrogen dioxide
- Sulphur dioxide
- Suspended particles including PM-10, particles less than 10 microns in size.
- Benzene
- Formaldehyde
- Polycyclic hydrocarbons

Background Information - Adverse Health Effects of Chronic Exposure to Petroleum Combustion Products. On November 18, 1994, the first-ever conference on "Air Pollution: Impacts on Body Organs and Systems" was held in Washington, D.C. by the National Association of Physicians for the Environment. An abridged version follows. The relevance of this perspective increases with increasing air pollution and climate change. 2008 Update.

Jaffe and Badman at the same conference summarized the effects of polluted air :

"Blood perfuses all of the body's organs and can carry toxic substances as well as beneficial substances, such as oxygen, to them. Air pollution is the source of many materials that may enter the human bloodstream through the nose, mouth, skin, and the digestive tract. Chemicals known to be harmful, such as benzene, lead and other heavy metals, carbon monoxide, volatile nitrites, pesticides, and herbicides. These substances have been shown to produce harmful effects on the blood, bone marrow, spleen, and lymph nodes. Blood cells are constantly undergoing turnover, with new blood cells entering the circulation as mature cells are lost, making the

blood system especially vulnerable to environmental poisoning. For example, lead interferes with normal red blood cell formation by inhibiting important enzymes. In addition, lead damages red blood cell membranes and interferes with cell metabolism in a way that shortens the survival of each individual cell. Each of these harmful effects can result in clinical anemia.

Benzene and other less known hydrocarbons are produced in petroleum refining, and are widely used as solvents and as materials in the production of various industrial products and pesticides. Benzene also is found in gasoline and in cigarette smoke. It has been shown that exposure to benzene is related to the development of leukemia and lymphoma. Benzene has a suppressive effect on bone marrow and it impairs blood cell maturation and amplification. Benzene exposure may result in a diminished number of blood cells or total bone marrow loss. A number of metabolites appear to be involved in this process, and there may be several targets of toxicity, including stem, progenitor, and some stromal cells.

Common air pollutants also have an affect on blood and thus on organs of the body. For example, carbon monoxide binds to hemoglobin two hundred times more avidly than oxygen and distorts the release to the tissues of any remaining oxygen. Thus, CO poisoning is a form of suffocation. Carbon monoxide can exacerbate cardiovascular disease in humans. Some airborne chemicals stimulate the immune system to activate leukocytes and macrophages that can produce tissue damage, especially to the cells that line human blood vessels. The combined effect of these events is to accelerate the changes that eventually lead to hypertension and ischemic heart disease.

Cory-Slechta and Lundberg discussed the adverse effects of pollution on the central nervous system: "The central nervous system (CNS) is the primary target for many serious air pollutants, such as lead, which is a major environmental hazard. Research provided evidence that levels of lead exposure associated with central nervous system effects, particularly as manifest in behavioral changes was lower than previously realized. Blood lead concentrations in children were not considered problematic until they exceeded 30 to 40 micrograms per deciliter ('g/dL); however, studies demonstrated

changes in cognitive function at blood concentrations as low as 10 to 15 'g/dL. While children are more susceptible to lead's CNS effects, adults exhibit similar deficits in learning and memory. Advanced aging increases vulnerability to the toxic effects of lead. In Germany, a large study documented an age-related decline in bone lead concentrations with advancing age. This effect was more pronounced in women than in men, reflecting post-menopausal processes in women which contribute to bone resorption and the release of lead back into the bloodstream. These results mean that brain lead exposure is actually increased during a period of already heightened susceptibility due to concurrent degeneration of other physiological functions, including both CNS and renal functions.

Although lead is the most studied of hundreds of known or suspected neurotoxic air pollutants, other heavy metals, pesticides, and organic solvents also cause neurobehavioral dysfunction. Expanded research in behavioral neurotoxicology is urgently needed. Changes in mood, cognition, and behavior are endpoints that need to be evaluated in addition to cancer rates or mortality data and may be more common. In various studies, increased levels of air pollutants are accompanied by increased psychiatric emergency calls and hospital admissions, behavior changes, and a lessened sense of well-being. Irritating odors and cigarette smoke have been found to increase aggressive behavior, and to decrease helping behavior and altruism, leading to a degradation of social interaction."

Goldstein and Albright discussed immune system effects: "The effects of airborne pollutants on the immune system have been most widely studied in the respiratory tract. An airborne pollutant may enter the respiratory tract as a volatile gas (e.g., ozone, benzene), as liquid droplets (e.g., sulfuric acid, nitrogen dioxide), or as particulate matter (e.g., components of diesel exhaust, aromatic hydrocarbons). These pollutants interact with the immune system and may cause local and systemic responses ranging from overactive immune responses to immunosuppression. Most airborne pollutants are small molecular weight chemicals that must be coupled with other substances (e.g., proteins or conjugates) before they can be recognized by the immune system and cause an effect. Some disorders which may occur because of pollutants in the

respiratory system are the following:

Immunosuppression can be demonstrated following exposure to polycyclic aromatic hydrocarbons (e.g.,tetrachlordibenzo-p-dioxin).

Hypersensitivity reactions (e.g., occupational asthma) can occur following exposure to toluene diisocyanate and other volatile chemicals.

There is clearly an underlying genetic basis for susceptibility to immunologic disease resulting from exposure to pollutants, but knowledge in this area is rudimentary at this time. For example, there is little understanding of genetically-determined susceptibility or resistance to pollutant-induced immune disorders. There is a lack of appropriate in vitro models, and it is difficult to identify specific, biologically-active substances that may be linked to immune disorders."

Workplace Exposure to Vehicle Exhaust and Chronic Illness

Here is a summary of a report on one patient with a known chemical exposure, as an example: Mr. A had been exposed to diesel and gasoline engine exhaust in the workplace from 1982 onward and had become too ill to work by Oct. 1993. Improvements to his work environment were not made until 1992, but apparently these were not adequate and he continued to be ill through 1993. He developed a chronic illness in the mid-80's which was slowly progressive and he beleived that chemical exposure at work had made him ill.

Mr. A manned a parts counter in a vehicle repair shop. The ventilation of this indoor work-space was judged to be inadequate and Mr A was exposed to significant concentrations of exhaust products which include a number of pathogenic possibilities.

His chief complaints were: chronic fatigue, recurrent flu-like illness, limited exertional tolerance, vertigo, dyspepsia with epigastric pain, chest pain with episodes of shortness of breath, cognitive dysfunction, anxiety. He described progressive dysfunction over several years and was concerned that exhaust exposure at work was making him ill. He described a progressive loss of tolerance for physical exertion - attempts at vigorous exertion left him exhausted, weak, and dizzy with cognitive

dysfunction; recovery from, for example, attempts to play ice-hockey would often take 48 hour or longer. He had trouble concentrating and suffered recent memory loss. He often felt severe fatigue with difficulty concentrating and memory drop outs at work. He withdrew from work Oct. 1993. He was more attentive to food choices and good nutrition and attempted to follow a graded exercise program. He reported benefit by staying away from the work environment and controlling his diet.

Prolonged exposure to exhaust gases seem to induce allergy symptoms or hypersensitivity. Activated immune systems tend to start reacting to airborne and food antigens - exposed patients begin reacting to food materials following a typical pattern of chronic illness, and routinely report intolerance to even short exposures of low concentrations of airborne chemicals. Unfortunately there is very little research to help us understand this problem. Since chronic fatigue syndrome has become a common term, studies have been reported showing a scatter of immune and endocrine abnormalities but no simple diagnostic test has emerged. Tests of indoor air quality were conducted but were inadequate to access his exposure.

Index Chemicals Benzene and the polycyclic hydrocarbons are index chemicals - examples of the many chemicals present in combustion products and while the descriptions of the chemicals are very general, the reader should have no doubt that chronic exposure can produce profound and long-lasting changes in biological function. This information is taken directly from information bulletins issued by the U.S. Agency for Toxic Substances and Disease Registry Division of Toxicology 1600 Clifton Road, E-29Atlanta, Georgia 30333 USA.

Benzene The greatest possibility for high-level exposures is in the workplace... most people are exposed to benzene in tobacco smoke and automobile exhaust. Benzene has been found in at least 337 of 1177 National Priorities List hazardous waste sites. Other environmental sources of benzene include gasoline (filling) stations, vehicle exhaust fumes, tobacco smoke, underground storage tanks that leak, wastewater from industries that use benzene, chemical spills, groundwater next to landfills containing benzene, and possibly some food products that contain benzene naturally. Brief Exposure at High

Levels--Death may occur in humans and animals after brief oral or inhalation exposures to high levels of benzene; however, the main effects of these types of exposures are drowsiness, dizziness, and headaches. These symptoms disappear after exposure stops.

Long-Term Exposures at Various Levels--From overwhelming human evidence and supporting animal studies, the U.S. Department of Health and Human Services has determined that benzene is carcinogenic. Leukemia (cancer of the tissues that form the white blood cells) and subsequent death from cancer have occurred in some workers exposed to benzene for periods of less than 5 and up to 30 years. Long-term exposures to benzene may affect normal blood production, possibly resulting in severe anemia and internal bleeding. In addition, human and animal studies indicate that benzene is harmful to the immune system, increasing the chance for infections and perhaps lowering the body's defense against tumors. Exposure to benzene has also been linked with genetic changes in humans and animals.

Animal studies indicate that benzene has adverse effects on unborn animals. These effects include low birth weight, delayed bone formation, and bone marrow damage. Some of these effects occur at benzene levels as low as 10 parts of benzene per million parts of air (ppm). Although benzene has been reported to have harmful effects on animal reproduction, the evidence for human reproductive effects, such as spontaneous abortion or miscarriage, is too limited to form a clear link with benzene.

Benzene can be measured in the blood and the breath. The body changes benzene to phenol, which can be measured in the urine. Amounts of benzene in blood samples and phenol in urine samples cannot be used as yet to predict what degree of harmful health effects may occur. The meaning of benzene and phenol measurements in blood and urine should be viewed carefully for several reasons: 1) phenol occurs naturally in urine, and amounts of urinary phenol would have to be much higher than usual before any measurement was meaningful; 2) present test methods are limited and raise doubts about the blood level values found in some laboratories; 3) because smoking can raise the background level of benzene in the blood, smoking habits must be considered when evaluating exposure to benzene; 4)

benzene disappears rapidly from the blood and measurements may be accurate only for recent exposures; 5) average amounts of benzene found in the body have not been determined for the general population.

The Environmental Protection Agency (EPA) set the maximum permissible level in drinking water at 5 parts of benzene per billion parts of water (ppb). Because benzene can cause leukemia, EPA established an ultimate goal of 0 ppb for benzene in drinking water and in ambient water such as rivers and lakes. EPA realizes that this goal may be unattainable and has estimated how much benzene in ambient water would be associated with one additional cancer case for every 100,000 persons (6.6 ppb benzene), one case for every 1 million persons (0.66 ppb benzene), and one case for every 10 million persons (0.066 ppb benzene). The National Institute for Occupational Safety and Health (NIOSH) has recommended an occupational exposure limit in air of 0.1 part of benzene per million parts of air (ppm). The Occupational Safety and Health Administration's (OSHA) legally enforceable limit is an average of 1.0 ppm over the standard 8-hour workday, 40-hour workweek.

Polycyclic aromatic hydrocarbons (PAHs)

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances. PAHs can be man-made or occur naturally. There is no known use for most of these chemicals except for research purposes. A few of the PAHs are used in medicines and to make dyes, plastics, and pesticides. They are found throughout the environment in the air, water and soil. There are more than 100 different PAH compounds. Although the health effects of the individual PAHs vary, the following 15 PAHs are considered as a group with similar toxicity: acenaphthene, acenaphthylene, anthracene, benzanthracene, benzopyrene, benzofluoranthene, benzoperylene, benzofluoranthene, chrysene dibenzanthracene, fluoranthene, fluorene, indenopyrene, phenanthrene, pyrene.

Several factors will determine whether harmful health effects will occur and what the type and severity of those health effects will be. These factors include the dose (how much), the duration (how long), the route

by which you are exposed (breathing, eating, drinking, or skin contact), the other chemicals to which you are exposed and your individual characteristics such as age, sex, nutritional status, family traits, life style, and state of health. As pure chemicals, PAHs generally exist as colorless, white, or pale yellow-green solids. Most PAHs are found as mixtures of two or more PAHs. They can occur in the air either attached to dust particles, or in soil or sediment as solids. They can also be found in substances such as crude oil, coal, coal tar pitch, creosote, road and roofing tar. Most PAHs do not dissolve easily in water, but some PAHs evaporate into the air. PAHs generally do not burn easily and they will last in the environment for months to years.

PAHs that are attached to dust and other particles in the air and originate from vehicle exhausts, asphalt roads, coal, coal tar, wildfires, agricultural burning and hazardous waste sites. Background levels of PAHs in the air are reported to be 0.02-1.2 milligrams per cublic meter (mg/m3) in rural areas and 0.15-19.3 mg/m3 in urban areas. You may be exposed to PAHs in soil near areas where coal, wood, gasoline, or other products have been burned or from the soil on or near hazardous waste sites, such as former manufactured-gas sites and wood-preserving facilities. PAHs have been found in some drinking water supplies in the United States. The background level of PAHs in drinking water ranges from 4 to 24 nanograms per liter . For most people, the greatest exposure to PAHs occurs in the workplace.

PAHs can enter the body through the lungs. PAHs enter the body quickly and easily by all routes of exposure. The rate at which PAHs enter your body is increased when they are present in oily mixtures and tend to be stored in the kidneys, liver, and fat, with smaller amounts in the spleen, adrenal glands and ovaries. Results from animal studies show that PAHs do not tend to be stored in for a long time and are excreted within a few days in the feces and urine.

The U.S. Department of Health and Human Services has determined that PAHs may be carcinogens. Several of the PAHs, including benzanthracene, benzopyrene, benzofluoranthene, benzofluoranthene, chrysene, dibenzanthracene, indenopyrene have caused tumors in laboratory animals when they ate them, when they were applied to their skin and when they breathed them in the air for long periods of time.

Reports in humans show that individuals exposed by breathing or skin contact for long periods of time to mixtures of other compounds and PAHs can also develop cancer. Mice fed high levels of benzopyrene during pregnancy had difficulty reproducing and so did their offspring. The offspring from pregnant mice fed benzopyrene also showed other harmful effects, such as birth defects and decreased body weight. Similar effects could occur in humans, but we have no information to show that these effects do occur.

Studies in animals have also shown that PAHs can cause harmful effects on skin, body fluids, and the body's system for fighting disease after both short- and long-term exposure. These effects have not been reported in humans. PAHs are changed into chemicals that can attach to substances within the body. The presence of PAHs attached to these substances can then be measured in body tissues or blood after exposure to PAHs. However, this test is still being developed and it is not known yet how well it works. PAHs or their breakdown products can also be measured in urine. Although these tests can tell that you have been exposed to PAHs, it is not yet possible to use these tests to predict the severity of any health effects that might occur or to determine the extent of your exposure to the PAHs. These tests are not routinely available at a doctor's office because they require special equipment for sampling and detecting these chemicals.

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