Comment Letter C-1

April 5, 2009

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APR 06 2009
City of Goleta
Planning & Environmental Svcs.

Dan Nemechek
Planning and Environmental Services
130 Cremona Drive, Suite B
Goleta, CA 93117

Dear Dan,

C-1.1 The Draft Supplemental EIR for the General Plan Track 3 Amendments is flawed. The
C-1.2 environmental review indicates that the new alternatives are not the environmentally superior
C-1.3 choice. Only CE 1.2, CE1.5, CE 5.1, and CE-1A-4 should be considered for change. There is no justification for the other changes and they will weaken the General Plan standards.

C-1.4 A Citywide Habitat Management Plan is not sufficient to protect the environment. It would be easier to further weaken than elements in the General Plan. There will also be a delay in preparing the Habitat Plan and the City will be left with no real protection.

C-1.5 The public has been very vocal about wanting the General Plan kept as originally prepared and approved. Alternative 1, the no change alternative, is preferred.

C-1.6 The statement "No new potentially significant effects on the environment were identified as a result of the proposed General Plan Amendments" is not accurate. Most of the changes will weaken environmental protection and is growth inducing.

Barbara S. Massey
7912 Winchester Circle
Goleta, CA 93117
685-5968

Response to Comment No. C-1.1

The commentator has expressed an opinion that the Draft Supplemental EIR for the proposed Track 3 General Plan amendments is flawed. Comment noted.

Response to Comments Nos. C-1.2 and C-1.3

The commentator recommends that only changes to CE 1.2, CE 1.5, CE 5.1, and CE-IA-4 should be considered. Comment noted.

Response to Comment No. C-1.4

The commentator is opposed to preparation of a citywide HMP because policies under the HMP could be weakened more easily than under the existing GP/CLUP and protections would be lost in the interim. See Response to Comment No. A-6.3.

Response to Comment No. C-1.5

The commentator states that the public has been very vocal about wanting the General Plan kept as originally prepared and approved, and that Alternative 1 (the No Change Alternative) is preferred. Comment noted.

Response to Comment No. C-1.6

The commentator states that the statement “no new potentially significant effects on the environment were identified as a result of the proposed General Plan Amendments” is not accurate. The commentator believes that most of the changes will weaken environmental protection and are growth inducing. Comment noted.

Comment Letter C-2

April 5, 2009

Comments from Ingeborg E. Cox, MD

Track 3 General Plan Amendments

It appears that the City of Goleta's General Plan (GP) will have a lot of Policy amendments dictated by what the developers need for the changes they want in their projects.
Just to cite a few:
Reducing the quantities and size of lands designated as ESHA. The beneficiaries will be: Haskell's Landing, Bacara and the Bishop Ranch.
Allow for the construction of bridge abutments within creeks and riparian corridors which benefits Haskell's Landing.
Why are we letting the developers dictate the changes for the General Plan? We have a plan that took a lot of Goletans' time and effort that should be respected. Such changes should be project specific and not extended to the whole GP.

C-2.1 Section 3.3 page 7: It needs to be noted that Venoco's Ellwood processing plant has also hydrogen sulfide (H2S) in their facilities. The sour crudes that come from Platform Holly to the Elwood contain H2S.
The Ellwood Onshore Facility (EOF) and the Elwood Marine Terminal (EMT) are the last two remaining non-conforming oil and gas facilities on the South Coast. (2-2) Draft EIR Venoco Full Field Development. (DEIRVFFD)
Releases from the EOF could impact the public by exposing the public to flammable vapors that could burn or explode, by exposing the public to toxic vapors (hydrogen sulfide), exposing the public to thermal radiation from fires, or exposing the public to overpressures from explosions.(4.2-41 DEIRVFFD)

C-2.2 Regarding Table 3.3-2: There is no explanation on why the maximum 24-hr concentration for suspended particulates (PM10) has jumped to 233.7 in 2007 from 39.9 in 2006. I strongly hope this is a typographical error as otherwise this has health consequences and should be explained.

C-2.3 The city of Goleta should formalize Green House Gases (GHG) thresholds within its Environmental thresholds and Guidelines Manual.

C-2.4 Constructions Emissions need to be considered as it appears that in the Hollister Corridor multiple construction projects will occur simultaneously in this part of the city.

C-2.5 Under the techniques to limit emissions of both ozone precursors and fugitive dust the phrases "whenever possible," "wherever feasible," "if available," and "if feasible" should be removed since otherwise no compliance is the result.

C-2.6 3.3-26 Table 3.3-6: Recommendations on siting new sensitive land uses. Avoid siting new sensitive land uses (residences) within 500 feet of a freeway. Even if the recommendations in the California Air Resources Board handbook are currently advisory, there are already enough medical studies that document the negative health effects of diesel exhaust and particulate matter 10 and 2.5. See enclosed five medical articles.

C-2.7 Also, a consideration to noise exposure which affects the residents health, has to be taken into con sideration. See enclosed synopsis of road traffic noise and high blood pressure.

(more)

C-2.8 3.5-1 Cultural resources. Do not remove the reference to the Coastal Development Permit requirement. Events should not take place in culturally sensitive areas. In the case of the Bacara, some of these culturally sensitive areas were Chumash sites.

C-2.9 Concerning the streamside protection area (SPA), leave the buffer width at 100 feet.

C-2.10 3.8-5 The middle-income household ($25,000-75,000). The traditional standard for determining housing affordability is when housing costs do not exceed 30 percent of the income of a household.

C-2.11 Regarding 3.9: There should be no alteration in existing drainage patterns by new development. If development is considered, before the development goes to the DRB a detailed hydraulic study shall be performed by the developer to determine the impacts of the construction buidout.

C-2.12 Alteration of an existing drainage pattern or creek can result in erosion, siltation or increased surface runoff. Housing should not be placed within a 100- year flood hazard area.

C-2.13 Keep policies CE2, 3, 6 and 10 in place as is. The buffer zone of 100 feet needs to be preserved to protect creeks. Buffer zones are important as they filter pollutants from runoff before these contaminants reach the creek.

Enclosed attachments of six medical articles on health effects and air pollution and noise

Comment Letter C-2
Attachment 1 of 6

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

(Received in original form March 4, 2004; accepted in final form May 31, 2004)

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This article has an online supplement, which is accessible from this issue's table of contents online at www.atsjournals.org

Am J Respir Crit Care Med Vol 170. pp 520–526, 2004
Originally Published in Press as DOI: 10.1164/rccm.200403-281OC on June 7, 2004
Internet address: www.atsjournals.org

Comment Letter C-2
Attachment 2 of 6

500 meters !!

Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study

W James Gauderman, Hita Vora, Rob McConnell, Kiros Berhane, Frank Gilliland, Duncan Thomas, Fred Lurmann, Edward Avol, Nino Kunzli, Michael Jerrett, John Peters

Summary

Background Whether local exposure to major roadways adversely affects lung-function growth during the period of rapid lung development that takes place between 10 and 18 years of age is unknown. This study investigated the association between residential exposure to traffic and 8-year lung-function growth.

Methods In this prospective study, 3677 children (mean age 10 years [SD 0·44]) participated from 12 southern California communities that represent a wide range in regional air quality. Children were followed up for 8 years, with yearly lung-function measurements recorded. For each child, we identified several indicators of residential exposure to traffic from large roads. Regression analysis was used to establish whether 8-year growth in lung function was associated with local traffic exposure, and whether local traffic effects were independent of regional air quality.

Findings Children who lived within 500 m of a freeway (motorway) had substantial deficits in 8-year growth of forced expiratory volume in 1 s (FEV_1, −81 mL, p=0·01 [95% CI −143 to −18]) and maximum midexpiratory flow rate (MMEF, −127 mL/s, p=0·03 [−243 to −11]), compared with children who lived at least 1500 m from a freeway. Joint models showed that both local exposure to freeways and regional air pollution had detrimental, and independent, effects on lung-function growth. Pronounced deficits in attained lung function at age 18 years were recorded for those living within 500 m of a freeway, with mean percent-predicted 97·0% for FEV_1 (p=0·013, relative to >1500 m [95% CI 94·6–99·4]) and 93·4% for MMEF (p=0·006 [95% CI 89·1–97·7]).

Interpretation Local exposure to traffic on a freeway has adverse effects on children's lung development, which are independent of regional air quality, and which could result in important deficits in attained lung function in later life.

Lancet 2007; 369: 571–77
Published Online January 26, 2007
DOI:10.1016/S0140-6736(07)60037-3
See Comment page 535

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Correspondence to: Dr W James Gauderman jimg@usc.edu

Introduction

Both cross-sectional[1–9] and longitudinal[10–15] studies have shown that lung function in children is adversely affected by exposure to urban, regional air pollution. Evidence has emerged that local exposure to traffic is related to adverse respiratory effects in children, including increased rates of asthma and other respiratory diseases.[16–21] Cross-sectional studies in Europe have shown that deficits in lung function are related to residential exposure to traffic.[22,23–32] However, does traffic exposure have an adverse effect on lung-function development in children? The answer to this question is important in view of the extent of traffic exposure in urban environments and the established relation between diminished lung function in adulthood and morbidity and mortality.[33–39]

We investigated the association between residential exposure to traffic and 8-year lung-function development on the basis of cohort data from the Children's Health Study. We also studied the joint effects of local traffic exposure and regional air quality on children's lung development.

Methods

Participants

The Children's Health Study recruited two cohorts of fourth-grade children (mean age 10 years [SD 0·44], one in 1993 (cohort 1, n=1718) and the other in 1996 (cohort 2, n=1959). All children were recruited from schools in 12 southern California communities as part of an investigation into the long-term effects of air pollution on children's respiratory health.[7,14,40] A consistent protocol was used in all communities to identify schools, and all students targeted for study were invited to participate.[40] Overall, 82% (3677) of available students agreed to participate. Pulmonary-function data were obtained yearly by trained field technicians, who travelled to study schools to undertake maximum effort spirometry on the children, using the same equipment and testing protocol throughout the study period. Details of the testing protocol have been previously reported.[7,25] Children in both cohorts were followed up for 8 years.

A baseline questionnaire, completed at study entry by each child's parent or legal guardian, was used to obtain information on race, Hispanic ethnic origin, parental income and education, history of doctor-diagnosed asthma, in-utero exposure to maternal smoking, and household exposure to gas stoves, pets, and environmental tobacco smoke.[40] A yearly questionnaire, with similar structure to that of the baseline questionnaire, was used to update information on asthma status, personal smoking, and exposure to environmental tobacco smoke. For statistical modelling, a three-category socioeconomic status variable was created on the basis of total household income and education of the parent or guardian who completed the questionnaire. High socioeconomic status (23% of children, n=823) was defined as a parental

Comment Letter C-2
Attachment 3 of 6

ENVIRONMENTAL EXPOSURE

Traffic exposure and lung function in adults: the Atherosclerosis Risk in Communities study

Haidong Kan, Gerardo Heiss, Kathryn M Rose, Eric Whitsel, Fred Lurmann, Stephanie J London

Thorax 2007;62:873–879. doi: 10.1136/thx.2006.073015

See end of article for authors' affiliations

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Received 9 October 2006
Accepted 31 March 2007
Published Online First 18 April 2007

Background: Traffic exposure is a major contributor to ambient air pollution for people living close to busy roads. The relationship between traffic exposure and lung function remains inconclusive in adults.
Methods: A cross-sectional study was conducted to investigate the association between traffic exposure and lung function in the Atherosclerosis Risk in Communities (ARIC) study, a community based cohort of 15 792 middle aged men and women. Traffic density and distance to major roads were used as measures of traffic exposure.
Results: After controlling for potential confounders including demographic factors, personal and neighbourhood level socioeconomic characteristics, cigarette smoking and background air pollution, higher traffic density was significantly associated with lower forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) in women. Relative to the lowest quartile of traffic density, the adjusted differences across increasing quartiles were 5.1, −15.4 and −21.5 ml for FEV_1 (p value of linear trend across the quartiles = 0.041) and 1.2, −23.4 and −34.8 ml for FVC (p trend = 0.010). Using distance from major roads as a simpler index of traffic related air pollution exposure, the FEV_1 was −15.7 ml (95% CI −34.4 to 2.9) lower and the FVC was −24.2 ml (95% CI −46.2 to −2.3) lower for women living within 150 m compared with subjects living further away. There was no significant effect of traffic density or distance to major roads on lung function in men. The FEV_1/FVC ratio was not significantly associated with traffic exposure in either men or women.
Conclusions: This is the largest published study of traffic exposure and pulmonary function in adults to date. These results add to growing evidence that chronic exposure to traffic related air pollution may adversely affect respiratory health.

Road traffic is a major factor in ambient air pollution in industrialised countries, contributing pollutants including fine particulate matter, carbon monoxide and oxides of nitrogen. An expanding body of epidemiological research suggests that traffic related exposure is associated with acute and chronic respiratory effects.[1–7] For example, residential proximity to busy roads is associated with a variety of adverse respiratory health outcomes including symptoms[1–3] and asthma exacerbation.[4–7] The effect of traffic air pollution on adult lung function remains inconclusive; exposure to automobile exhaust was associated with lower lung function in adults in some studies[8–12] but not others.[13 14]

Traffic emissions result in small scale spatial variations and higher concentrations within short distances from major roads.[15 16] Air pollution data from fixed monitoring stations may be inadequate to study traffic related air pollution and health outcomes, especially for those living near busy roads. For example, Hoek and colleagues identified a consistent association between cardiopulmonary mortality and living near a major road, but not with estimated ambient background concentration of the traffic indicator pollutants black smoke and nitrogen dioxide.[17] Assessment of traffic exposure can enhance studies of health effects of ambient air pollution because local sources are important, and because few people live close to the monitoring stations which are purposefully located away from local sources like busy roads. For people living close to busy roads, air pollution from traffic sources may be more important than the area background measured at the closest monitoring station.

We examined the relation between traffic exposure and lung function in a population based cohort of 15 792 middle-aged men and women, the Atherosclerosis Risk in Communities (ARIC) study.

METHODS

Study population

Participants were from the ARIC study which is designed to investigate the natural history and aetiology of atherosclerosis and its sequelae. The design, objectives and quality control activities of the ARIC study have been reported in detail elsewhere.[18 19] Participants were sampled from four US communities: Forsyth County, North Carolina; Jackson, Mississippi; northwest suburbs of Minneapolis, Minnesota; and Washington County, Maryland. The lung function variables collected during visit 1 (1987–9) were used with the traffic and background air pollution data in a cross-sectional analysis. Participants of an ethnicity other than African American or white were excluded from the current analysis (n = 48). Also, African-Americans from Minnesota and Maryland field centres were excluded (n = 55) because of their small number.

We also repeated this analysis using visit 2 (1990–2) and conducted a longitudinal analysis on the change in lung function between visit 1 and visit 2.

Pulmonary function measurements

The main measurements of lung function were forced expiratory volume in 1 s (FEV_1), volume of gas (in litres) exhaled in the first second of expiration; forced vital capacity (FVC), total volume of gas exhaled; and the ratio of FEV_1/FVC.

Abbreviations: ARIC, Atherosclerosis Risk in Communities; BMI, body mass index; ETS, environmental tobacco smoke; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; GIS, geographical information system; PM_{10}, particulate matter <10 μm

Comment Letter C-2
Attachment 4 of 6

ORIGINAL CONTRIBUTION

Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

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Michael J. Thun, MD
Eugenia E. Calle, PhD
Daniel Krewski, PhD
Kazuhiko Ito, PhD
George D. Thurston, ScD

Context Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive.

Objective To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality.

Design, Setting, and Participants Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998.

Main Outcome Measure All-cause, lung cancer, and cardiopulmonary mortality.

Results Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-μg/m³ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

Conclusion Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

JAMA. 2002;287:1132-1141 www.jama.com

BASED ON SEVERAL SEVERE AIR pollution events,[1-3] a temporal correlation between extremely high concentrations of particulate and sulfur oxide air pollution and acute increases in mortality was well established by the 1970s. Subsequently, epidemiological studies published between 1989 and 1996 reported health effects at unexpectedly low concentrations of particulate air pollution.[4] The convergence of data from these studies, while controversial,[5] prompted serious reconsideration of standards and health guidelines[6-10] and led to a long-term research program designed to analyze health-related effects due to particulate pollution.[11-13] In 1997, the Environmental Protection Agency adopted new ambient air quality standards that would impose regulatory limits on fine particles measuring less than 2.5 μm in diameter ($PM_{2.5}$). These new standards were challenged by industry groups, blocked by a federal appeals court, but ultimately upheld by the US Supreme Court.[14]

Although most of the recent epidemiological research has focused on effects of short-term exposures, several studies suggest that long-term exposure may be more important in terms of overall public health.[4] The new standards for long-term exposure to $PM_{2.5}$ were originally based primarily on 2 prospective cohort studies,[15,16] which evaluated the effects of long-term pollution exposure on mortality. Both of these studies have been subjected to much scrutiny,[5] including an extensive independent audit and reanalysis of the original data.[17] The larger of these 2 studies linked individual risk factor and vital status data with national ambient air pollution data.[16] Our analysis uses data from the larger study and

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Santa Barbara County Air Quality News • Issue 91 • Summer 2007

Santa Barbara County
Air Pollution Control District

On the Air

Comment Letter C-2
Attachment 5 of 6

Living Near Freeways Harms Children's Lungs

Children who live near busy roads experience slower lung development than children who don't—whether they live in Los Angeles, Santa Maria or Santa Barbara. This is one of the key findings from the Children's Health Study, a 15-year University of Southern California (USC) family of studies of more than 11,000 children from sixteen communities.

Researchers found that children who live within 500 meters, or approximately a third of a mile, of a freeway or busy roadway have substantial deficits in lung function and lung development when compared with children living 1,500 meters, or about a mile away from the roadways.

"The interesting thing about this is that the relationship between busy roadways and slower lung growth holds true regardless of the air quality of the surrounding area, whether it's a community like Los Angeles or Riverside with higher pollution levels, or one like Santa Barbara or Santa Maria where the air is relatively much cleaner," notes Ed Avol, Professor at the Keck School of Medicine at USC. He explains that children who both live near freeways and live in a high-pollution community have a double challenge, because they are affected by both the regional and local pollution.

He comments, "When you think about just how many children in Southern California live near freeways, this is a pretty significant finding." In Santa Barbara and Santa Maria, impacts were primarily related to living near freeways such as Highway 101, he explains, as these communities do not have many high-traffic four-lane roads considered busy roadways.

The Children's Health Study began in 1993 to assess the impacts of air pollution on the growth and development of kids' lungs. Children—primarily fourth graders—were recruited from schools in twelve communities from San Diego to San Luis Obispo counties, ranging from Southern California locations with more polluted air, to Santa Maria and Lompoc. Communities in Santa Barbara and San Luis Obispo counties were selected for their cleaner air, compared to Los Angeles area counties.

The children were studied each year as their lungs developed until high school graduation, and results were compared to air quality measurements in the communities. The research team collected extensive information about each child's home, socioeconomic status and other factors that might affect health.

Then the team visited the schools and measured the children's lungs, assessing how much air could be expelled in one breath and how quickly it could be expelled.

Early findings from the studies showed that the lungs of children in more polluted areas were growing more slowly than the lungs of children in cleaner-air communities. Higher levels of ozone, a key component of smog, were associated with more school absences and more cases of newly diagnosed asthma. The study also showed that when children moved from more polluted to less polluted areas, their lungs began to grow at rates similar to other children in that cleaner community, although they seemed to remain permanently affected by the period of slower growth.

(continued on page two)

Wildfire Smoke

In July and August, APCD and the County Public Health Department issued advisories calling on residents to stay indoors and avoid outdoor exercise when smoke from the Zaca Fire was present in the air. The Zaca Fire started July 4, and burned over 240,000 acres, prompting evacuation orders, and creating ash and plumes of smoke.

APCD Public Information Officer Bobbie Bratz remarked, "The

(continued on page two)

Smoke plumes from the Zaca Fire

Inside...

Comment Letter C-2
Attachment 6 of 6

ORIGINAL ARTICLE

Road traffic noise and hypertension

Gösta Leon Bluhm, Niklas Berglind, Emma Nordling, Mats Rosenlund

Occup Environ Med 2007;64:122–126. doi: 10.1136/oem.2005.025866

See end of article for authors' affiliations

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Accepted 6 October 2006
Published Online First 19 October 2006

Background: It has been suggested that noise exposure increases the risk of hypertension. Road traffic is the dominant source of community noise exposure.
Objective: To study the association between exposure to residential road traffic noise and hypertension in an urban municipality.
Methods: The study population comprised randomly selected subjects aged 19–80 years. A postal questionnaire provided information on individual characteristics, including diagnosis of hypertension. The response rate was 77%, resulting in a study population of 667 subjects. The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB(A), from <45 dB(A) to >65 dB(A).
Results: The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB(A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for >10 years (OR 1.93; 95% CI 1.29 to 2.83). Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Conclusion: The results of our study suggest an association between exposure to residential road traffic noise and hypertension.

Noise acts as a ubiquitous stress-mediating factor in the physical environment. General annoyance, disturbances in psychosocial well-being and reduction in sleep quality are commonly reported effects of noise exposure.[1 2] An increased risk of non-auditory physiological effects due to noise, such as hypertension and ischaemic heart disease, have also been suggested.[3–9] Most previous studies have been performed in occupational settings with high noise levels.[3 4 6–10] Community noise is less well studied.

Road traffic is the dominating source of community noise in the urban environment. Few studies have investigated an association between exposure to road traffic noise and hypertension, and the results are conflicting.[3 4 11] Studies in this field have low precision and validity problems, including crude exposure assessments, selection bias and limited control of important confounding factors. Exposure has usually been assessed either from subjective reports or without consideration of important factors that may influence the individual exposure level—for example, window type, bedroom window orientation and type of residence.

The suggested biological mechanism for an association between exposure to community noise and hypertension is that noise induces stress by disturbing sleep and interfering with relaxation and concentration and many other cognitive effects that activate the sympathetic nervous system and the endocrine system.[12] The primary physiological effects of noise exposure are vegetative reactions such as increase in blood pressure, heart rate and finger pulse amplitude, cardiac arrhythmia, and changes in respiration and body movements.[13] Therefore, a hypothesis has emerged that stress due to persistent exposure to environmental noise could result in permanent vascular changes, with increased blood pressure and ischaemic heart disease as potential outcomes.[14–16]

Our objective was to study a possible association between exposure to residential road traffic noise and hypertension among adults in an urban municipality. To better characterise individual noise exposure, we aimed at investigating factors that may influence the true exposure level, such as window type, bedroom window orientation and type of residence.

METHODS

Study population

The study was performed in a municipality with 55 000 inhabitants located 15 km north of Stockholm City. A questionnaire designed for a countywide investigation of health effects related to various environmental factors was distributed in April 1997 to 1000 individuals aged 19–80 years living in the municipality.[17] A stratified random sampling procedure was applied to ensure a sufficient number of subjects exposed to traffic noise, consisting of two strata with 500 residents in each. The noise-exposed group was drawn from those living within 100 m on each side of the highway, main roads or the railway. The other sample was drawn from the remaining parts of the municipality. Statistics Sweden performed the sampling by combining the National Population Register (containing background information for the study population) with the Real Estate Register (containing geographical coordinates for the residence of each individual). The response rate was 77% in both samples. This study focused on exposure to road traffic noise; thus, subjects who were residing close to the railway (n = 91) were not included. One subject who had removed the identification sticker from the returned questionnaire was excluded, as we did not have the address of that subject. In total, the study comprised 667 subjects.

Questionnaire

The survey included 87 questions and was mainly focused on prevalence of allergic diseases and environmental risk factors of regional importance. Information on educational level, employment status, general living conditions, and smoking habits was

Response to Comment No. C-2.1

The commentator states that it needs to be noted that Veneco's Ellwood Processing plant [Elwood Onshore Facility (EOF)] contains hydrogen sulfide (H_2S) in their facilities, and that releases from the EOF could impact the public by exposing the public to flammable vapors, by exposing the public to toxic vapors (hydrogen sulfide), exposing the public to thermal radiation, exposing the public to thermal radiation from fires, or exposing the public to overpressures from explosions.

The Track 3 Supplemental EIR incorporates the 2006 GP/CLUP EIR by reference. The comments listed above are addressed in Section 3.7.1.2, Oil and Gas Production, Processing, and Transport Hazards.

Response to Comment No. C-2.2

The commentator has inquired as to why the 24-hour concentration for suspended particulates (PM 10) has jumped to 233.7 in 2007 from 39.9 in 2006, as shown in Table 3.3-2.

The spike in 24-hour concentration for PM10 was attributable to an "Ash Event from the 2007 Zaca fire," as reported in CARB's 2007 Annual Air Quality Report, Table 8, posted on the SBAPCD website.

Response to Comment No. C-2.3

The commentator has expressed an opinion regarding the establishment of GHG thresholds, and does not comment on the environmental analysis presented in this Supplemental EIR. Comment noted. See Response to Comment No. A-5.27.

Response to Comment No. C-2.4

The commentator states that construction emissions need to be considered as it appears that in the Hollister Corridor multiple construction projects will occur simultaneously.

Constructions emissions are addressed in SEIR Section 3.3.3.3, Project Impacts, in the discussion for Impact 3.3-1 Construction Emissions.

Response to Comments Nos. C-2.5 through C-2.7

See Response to Comment No. C-2.3.

Response to Comment No. C-2.8

The commentator states that the reference to the Coastal Development Permit requirement should not be removed from the General Plan as events should not take place in culturally sensitive habitats.

Cultural Resources Section 3.5, Paragraph 2, addresses this comment and has been updated to reflect the following:

> Alternative 2a includes proposed policy amendment OS 1.10, which would involve removal of the reference to the Coastal Development Permit requirement for any temporary event that proposes to use a sandy beach area. Removal of this language

could potentially allow for temporary events to take place in culturally sensitive areas and remove the ability of the City to control methods of access or develop mitigation to reduce potential cultural resource impacts. However, future temporary events would still be required to undergo regulatory review by the City via a Coastal Development Permit (CDP) per the adopted Coastal Zoning Ordinance. In addition, any temporary event would be subject to the California Coastal Commission regulations and would be required to minimize and mitigate potential cultural resource impacts to public access and recreation along the shoreline. The California Coastal Commission currently requires a CDP for temporary events that occupy all or part of a sandy beach area. Removal of the reference to the CDP from the GP/CLUP would have no effect on the applicability of the City's CDP requirement per the Coastal Zoning Ordinance, nor the Coastal Commission Permit requirements that remain in effect. Alternatives 2b and 3 propose the same policy amendments as Alternative 2a.

Response to Comment No. C-2.9

The commentator supports keeping the SPA width at 100 feet.

Comment noted. See Attachment A for additional responses to comments on proposed changes to CE 2.2 and the amended CE 2.2 adopted by the City in May 2009 under a separate action.

Response to Comments Nos. C-2.10 through C-2.12

See Response to Comment No. C-2.3.

Response to Comment No. C-2.13

The commentator recommends that no change be made to existing CE policies 2, 3, 6, and 10.

Comment noted. See Attachment A for additional responses to comments on proposed changes to CE 2.2 and the amended CE 2.2 adopted by the City in May 2009 under a separate action.