

AGENDA ITEM #8
March 24, 2009
Discussion

MEMORANDUM

March 20, 2009

TO: County Council
FROM: ^{GO} Glenn Orlin, Deputy Council Staff Director
SUBJECT: **Discussion**—health effects of air quality near major highways

At the suggestion of Councilmember Trachtenberg, the Council President has scheduled convening the Council as the Board of Health to gain better understanding of the effects of traffic on major highways (such as the Capital Beltway, I-270, and the Intercounty Connector) on public health, and what measures local jurisdictions are taking to mitigate the effects. Ron White, Deputy Director of the Risk Sciences and Public Policy Institute at Johns Hopkins University's Bloomberg School of Public Health (©A), has been invited to give about a 30-minute presentation on these topics, leaving another half-hour for discussion.

Background. Several Councilmembers raised this issue in early 2007 after the publication of an article by Gauderman, et. al., which shows a positive correlation between proximity to freeways and the respiratory problems in children between the ages of 10-18 (©1-7). On February 1 they requested that the Planning Board provide information as to how many residents live within 500m of the ICC; the Board responded on February 7 with information about the ICC, but also I-270 and I-495 (©8-10). The chart on ©10 notes that those exposed to the ICC will be comparable to Beltway (both in total and per-mile), but considerably less than to I-270. Not noted is that exposure is also a function of traffic volume and, in particular, the volume of heavy truck traffic. In 2030 the traffic volume on the ICC will vary generally between 60-100,000 vehicles per day (vpd), while the projection for I-495 is 250-275,000 vpd. I-270 is projected to carry volumes between these two extremes. Heavy truck traffic volume is and will continue to be a much higher percentage of the traffic flow on the Beltway and I-270, since they are key interregional traffic corridors.

On October 8, 2007 then-Council President Marilyn Praisner posed several questions to the State Highway Administration on a wide range of environmental issues regarding the ICC; an excerpt from SHA's reply regarding public health issues is on ©11-14. In 2008 several members of the House of Delegates sponsored a bill that would have required a public health impacts assessment to be completed before spending funds to build the ICC, but the bill died in Committee (©15-17).

Further reading. Attached are several other technical articles examining the relationships between traffic exposure and public health (©18-52).

Ronald H. White, M.S.T.
Johns Hopkins University
Bloomberg School of Public Health
Baltimore, Maryland

Ronald H. White is an Associate Scientist in the Department of Health Policy and Management, with joint appointments in the Departments of Epidemiology and Environmental Health Sciences, at the Johns Hopkins Bloomberg School of Public Health in Baltimore, Maryland. Mr. White also serves as Deputy Director of the Risk Sciences and Public Policy Institute at the Johns Hopkins Bloomberg School of Public Health.

He previously served as Assistant Executive Director, Education, Research, and Community Affairs at the National Osteoporosis Foundation and in several positions at the National Office of the American Lung Association, culminating as Assistant Vice President, National Policy. Prior to joining the American Lung Association, he was senior transportation/air quality planner and then public participation coordinator for air quality planning at the Tri-State Regional Planning Commission in New York, N.Y. He earned his Master of Science in Teaching degree in environmental studies from Antioch University, and a Bachelor of Arts degree in environmental science from Clark University.

Mr. White currently serves as a member of the External Science Advisory Committee for the National Environmental Respiratory Center of the Lovelace Respiratory Research Institute. He was a member of the National Research Council Committee on Research Priorities for Airborne Particulate Matter, the Integrated Human Exposure Committee of the EPA Science Advisory Board, and the EPA Blue Ribbon Panel on the Use of Oxygenates in Gasoline. He also served as a consultant member of the EPA Clean Air Scientific Advisory Committee for the 1999-2006 Particulate Matter National Ambient Air Quality Standards Review. Mr. White has published several articles in the peer-reviewed literature, as well as contributing to book chapters.

A

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 (FEV₁, -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 FEV₁ ($p=0.013$, 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.

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.^{16–28} Cross-sectional studies in Europe have shown that deficits in lung function are related to residential exposure to traffic.^{27,29–32} 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.^{7,15} 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

Published Online
January 26, 2007
DOI:10.1016/S0140-6736(07)60037-3

See also Online/Comment
DOI:10.1016/S0140-6736(07)60038-5

Department of Preventive Medicine, University of Southern California, 1540 Alcazar Street, Suite 220, Los Angeles, CA 90033, USA (W J Gauderman PhD, H Vora MS, Prof R McConnell MD, K Berhane PhD, Prof F Gilliland MD, Prof D Thomas PhD, E Avol MS, Prof J Peters MD); Sonoma Technology Inc., #C, Petaluma, CA 94954, USA (F Lurmann MS); Respiratory and Environmental Research Unit, Institut Municipal d'Investigació Mèdica, C. Doctor Aiguader, 80, 08003 Barcelona, Spain (N Kunzli MD); and Division of Environmental Health Sciences, School of Public Health, University of California, Berkeley, CA 94720-7360, USA (M Jerrett PhD)
Correspondence to: Dr W James Gauderman jimg@usc.edu

income greater than US\$100 000 per year, or an income over US\$15 000 per year and at least 4 years of college education. The middle category (36%, n=1283) included children with a parental income between US\$15 000 and US\$100 000 and some (less than 4 years) college or technical school education, and low socioeconomic status (41%, n=1483) included all remaining children.

The study protocol was approved by the institutional review board for human studies at the University of Southern California, and written consent was provided by a parent or legal guardian for every study participant.

Exposure data

We characterised exposure of every study participant to traffic-related pollutants by two types of measures—proximity of the child's residence to the nearest freeway or to the nearest major non-freeway road, and model-based estimates of traffic-related air pollution at the residence, derived from dispersion models that incorporated distance to roadways, vehicle counts, vehicle emission rates, and meteorological conditions.⁴¹ Regional air pollution was continuously monitored at one central site location within each study community over the course of the investigation. Further details of exposure assessment are available in the webappendix.

See Online for webappendix

Statistical methods

The outcome data consisted of 22 686 pulmonary-function tests recorded from 3677 participants during 8 years in both cohorts. We focused on three pulmonary-function measures: forced vital capacity (FVC), forced expiratory volume in 1 s (FEV₁), and maximum midexpiratory flow rate (MMEF, also known as FEF₂₅₋₇₅). The exposures of primary interest were the traffic measures described above.

We used a hierarchical mixed-effects model to relate 8-year growth in each lung-function measure to traffic exposure, with basic structure that has been previously described.⁴² To account for the growth pattern in lung function during this period, we used a linear spline model,⁴³ constructed so that 8-year growth in lung function was estimated jointly with other model parameters. We estimated and tested the effect of traffic exposure on 8-year growth, and in some analyses on mean lung function at 10 and 18 years of age. The model allowed for separate growth curves for each sex, race, ethnic origin, cohort, and baseline-asthma subgroup. The model also included adjustments for height, height squared, body-mass index (BMI), BMI squared, present asthma status, exercise or respiratory illness on the day of the test, any tobacco smoking by the child in the previous year, and indicator variables for field technician. Random effects for the intercept and 8-year growth parameters were included at the level of participant and community.

To keep the potential effect of outliers to a minimum and to examine possible non-linear exposure-response relations, we used categorical forms of each traffic

indicator in our models. For distance to the freeway, we formed four categories—less than 500 m, 500–1000 m, 1000–1500 m, and more than 1500 m. Distances to non-freeway major roads were similarly categorised based on distances of 75 m, 150 m, and 300 m. Model-based estimates of pollution from freeways and non-freeways were categorised into quartiles on the basis of their respective distributions (see webappendix). The categories for all traffic indicators were fixed before any health analyses were done. Traffic effects are reported as the difference in 8-year growth for each category relative to the least exposed category, so that negative estimates signify reduced lung-function growth with increased exposure.

We also considered joint estimation of traffic effects within the community and pollution between communities, which was based on the long-term average pollutant concentrations measured at the central sites (see webappendix). Pollutant effects are reported as the difference in 8-year growth in lung function from the least to the most polluted community, with negative differences indicating growth deficits with increased exposure. Possible modification of a traffic effect by community-average ambient pollutant concentration was tested by inclusion of the appropriate interaction term in the model.

To examine attained lung function, we computed percent-predicted lung function for participants who were measured in 12th grade, our last year of follow-up (n=1497, mean age 17.9 years [SD 0.41]). To estimate predicted FEV₁ values, we first fitted a regression model for observed FEV₁ (log transformed) with predictors log height, BMI, BMI squared, sex, asthma status, race or ethnic origin, field technician, and sex-by-log height, sex-by-BMI, sex-by-BMI squared, sex-by-asthma, and sex-by-race or ethnic origin interactions. We calculated predicted FEV₁ on the basis of this model and percent-predicted as observed divided by predicted FEV₁. We used a regression model to calculate the mean percent-predicted value for each category of distance to the freeway, with adjustment for community. To aid in interpretation, we scaled percent-predicted values so that children who lived furthest (>1500 m) from a freeway had a mean of 100%, and we give means for the remaining distance groups relative to this benchmark. Analogous calculations were used to obtain the percent-predicted mean for FVC and MMEF.

Regression procedures in SAS (version 9.0) were used to fit all models. Associations denoted as significant were those with a p value less than 0.05, assuming a two-sided alternative hypothesis.

Role of the funding source

The funding sources of this study had no role in the study design, collection, analysis, or interpretation of data, in the writing of the report, or in the decision to submit the paper for publication. The corresponding

author had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Results

An average of 6.2 pulmonary function tests were done per child. There were equal proportions of male and female participants (webtable 1). Most children were of non-Hispanic white or Hispanic ethnic origin. 440 (12%) children lived within 500 m of a freeway, with most of these children residing in six of the 12 communities (webtable 2 and webfigure). Model-based estimates of pollution from a freeway were skewed toward either high or low values within most study communities.

8-year growth in FVC, FEV₁, and MMEF averaged 1512 mL, 1316 mL, and 1402 mL/s, respectively, in girls, and 2808 mL, 2406 mL, and 2476 mL/s, respectively, in boys. Closer residential distance to a freeway was associated with reduced growth in lung function (table 1). In children who lived within 500 m of a freeway, 8-year growth was significantly reduced compared with those who lived at least 1500 m from a freeway. Large deficits in FEV₁ and MMEF growth were also estimated for the two highest-exposure quartiles of model-based pollution from a freeway, although neither deficit was statistically significant. Indicators of traffic from non-freeway roads, including both distance and model-based pollution estimates, were not associated with reduced growth.

The association between FEV₁ growth and distance to a freeway was significant in various sensitivity analyses (table 2). Compared with the results shown in table 1 (base model), distance-effect estimates were larger with additional adjustment for socioeconomic status. Further investigation showed that low socioeconomic status was associated with increased traffic exposure, with mean residential distance to freeways of 1.8 km (SD 1.32), 2.0 km (1.65), and 2.5 km (1.91) for low, middle, and high groups respectively. However, socioeconomic status was not significantly associated with FEV₁ growth, and therefore adjustment for this variable induced only a modest change. Adjustment for indoor sources of air pollution including gas stoves, pets, and exposure to environmental tobacco smoke also resulted in little change in the estimated freeway-distance effects.

Significant distance effects were seen in the subset of children who reported never having had asthma, and in the subset of children who reported no active tobacco smoking. The relation between FEV₁ growth and distance was noticeably larger in boys than in girls, although a test of effect modification by sex was non-significant ($p=0.10$). Only six of the 12 communities had substantial numbers of children living within 500 m of a freeway. The estimated effects of freeway distance on lung development were more pronounced in these six higher-traffic communities than in the other communities. There was no significant evidence of heterogeneity in the local distance effects in these six communities (data not shown). Furthermore,

	FVC (mL) difference (95% CI)	FEV ₁ (mL) difference (95% CI)	MMEF (mL/sec) difference (95% CI)
Freeway distance*			
<500 m	-63 (-131 to 5)	-81 (-143 to -18)	-127 (-243 to -11)
500-1000 m	-31 (-93 to 32)	-41 (-99 to 17)	-35 (-142 to 73)
1000-1500 m	-19 (-84 to 46)	-33 (-93 to 26)	-94 (-204 to 16)
Model-based pollution from freeway†			
4th quartile (high)	-66 (-186 to 54)	-69 (-179 to 42)	-147 (-352 to 58)
3rd quartile	-61 (-151 to 29)	-78 (-161 to 5)	-144 (-298 to 9)
2nd quartile	-27 (-90 to 36)	-22 (-80 to 36)	-37 (-144 to 71)
Non-freeway distance‡			
<75 m	5 (-63 to 72)	-35 (-97 to 27)	-66 (-181 to 49)
75-150 m	4 (-59 to 68)	22 (-37 to 80)	35 (-74 to 144)
150-300 m	-10 (-63 to 42)	-8 (-56 to 40)	-16 (-105 to 73)
Model-based pollution from non-freeway†			
4th quartile (high)	13 (-70 to 96)	3 (-74 to 80)	2 (-140 to 144)
3rd quartile	42 (-27 to 111)	16 (-47 to 80)	-23 (-141 to 95)
2nd quartile	6 (-54 to 66)	2 (-53 to 57)	11 (-91 to 113)

*Difference in 8-year lung-function growth relative to children living at least 1500 m from a freeway. †Difference in 8-year lung-function growth relative to children in the first (lowest) quartile of exposure. ‡Difference in 8-year lung-function growth relative to children living at least 300 m from a non-freeway road.

Table 1. Association between 8-year lung-function growth and several indicators of residential traffic exposure

	Freeway distance (m)					
	<500	p	500-1000	p	1000-1500	p
Base model*	-81	0.012	-41	0.165	-33	0.275
Additional covariates						
Base+socioeconomic status	-92	0.005	-50	0.092	-37	0.228
Base+gas stove in the home	-86	0.008	-42	0.160	-33	0.281
Base+pets in the home	-80	0.013	-41	0.165	-33	0.275
Base+in-utero exposure to maternal smoking	-83	0.011	-33	0.269	-36	0.245
Base+second-hand smoke exposure	-86	0.008	-41	0.163	-37	0.230
Subgroups						
Non-asthmatics only	-83	0.025	-70	0.042	-61	0.091
Non-smokers only	-99	0.006	-49	0.154	-48	0.182
Boys only	-158	0.003	-54	0.264	-77	0.123
Girls only	-12	0.750	-39	0.254	3	0.932
Six communities with closest freeway proximity†	-105	0.003	-56	0.101	-40	0.260
Deleting observations after a residence change‡	-86	0.030	-73	0.042	-53	0.148

*Base model results are the same as those in table 1. All models include adjustment for the covariates listed in the Methods section. Values are the difference in 8-year FEV₁ growth relative to those living >1500 m from a freeway. †Including only children from the six communities with the largest number of children living near a freeway (Riverside, Atascadero, Alpine, San Dimas, Long Beach, and Santa Maria). ‡Censoring any pulmonary function tests recorded after a participant left his or her baseline address.

Table 2. Sensitivity analysis of freeway-distance effects on 8-year FEV₁ growth

around 34% (1267) of children moved from their baseline residence during follow-up but remained in one of the 12 study communities and thus continued to participate. If we omitted post-move lung-function measurements from the analysis, the estimated effects of freeway-distance on FEV₁ growth were more pronounced.

See Online for webtables 1 and 2 and webfigure

	Regional pollutant effect*	p	Local freeway distance (m)				p for interaction†		
			<500	p	500-1000	p		1000-1500	p
1000-1800 h ozone	-13	0.821	-81	0.012	-41	0.165	-33	0.275	0.51
Nitrogen dioxide	-109	0.003	-80	0.012	-41	0.166	-33	0.279	0.81
Acid	-111	0.002	-80	0.013	-41	0.164	-33	0.285	0.54
PM ₁₀	-111	0.013	-81	0.012	-42	0.158	-32	0.287	0.24
PM _{2.5}	-100	0.009	-80	0.012	-41	0.160	-33	0.285	0.40
Elemental carbon	-101	0.001	-80	0.012	-42	0.156	-33	0.282	0.63

* Pollutant effects are the difference in 8-year FEV₁ growth from lowest to highest observed community-average concentration of the pollutant, specifically: per increase of 37.5 ppb ozone (1000-1800 h), 34.5 ppb of nitrogen dioxide, 9.6 ppb of acid vapour, 51.4 µg/m³ of PM₁₀, 22.8 µg/m³ of PM_{2.5}, and 1.2 µg/m³ elemental carbon. Distance effects are the difference in 8-year growth relative to those living >1500 m from a freeway. † A test of whether freeway-distance effect is modified by regional concentration of the pollutant. PM₁₀, particulate matter <10 µm aerodynamic diameter; PM_{2.5}, particulate matter <2.5 µm aerodynamic diameter.

Table 3: Joint effect of regional pollution and local distance to a freeway on 8-year FEV₁ growth

Reduced lung-function growth was independently associated with both freeway distance and with regional air pollution (table 3). Statistically significant joint models of regional pollution with distance to freeway were seen for nitrogen dioxide, acid vapour, elemental carbon, and particulate matter with aerodynamic diameter less than 10 µm and less than 2.5 µm. Ozone was not associated with reduced lung-function growth. There was no significant evidence of effect modification (interaction) of local traffic effects with any of the regional pollutants.

A subset of 1445 children were observed over the full 8 years of the study, from age 10 to 18 years. In this group, we noted significant deficits in 8-year FEV₁ growth and MMEF growth for those who lived within 500 m of a freeway (table 4). At 10 years of age, there was some evidence of reduced lung function for those who lived closer to a freeway than those who did not, although none of the differences between distance categories was statistically significant. However, by 18 years of age, participants who lived closest to a freeway had

substantially lower attained FEV₁ and MMEF than those who lived at least 1500 m from a freeway.

These deficits in average FEV₁ and MMEF translated into pronounced deficits in percent-predicted lung function at 18 years of age (figure). There was a trend of lower percent-predicted lung function for children who lived closer to a freeway than for those who lived further away. The effect was most pronounced for those who lived less than 500 m from a freeway, with average percent predicted values of 97.0% (95% CI 94.6-99.4) for FEV₁ (p=0.013 relative to >1500 m) and 93.4% (89.1-97.7) for MMEF (p=0.006).

Discussion

This study shows that residential proximity to freeway traffic is associated with substantial deficits in lung-function development in children. 8-year increases in both FEV₁ and MMEF were smaller for children who lived within 500 m of a freeway, than for those who lived at least 1500 m from a freeway. Freeway effects were seen in subsets of non-asthmatic and non-smoking participants, which is an indication that traffic exposure has adverse effects on otherwise healthy children. Deficits in 8-year growth resulted in lower attained FEV₁ and MMEF at 18 years of age for participants who lived within 500 m of a freeway than for those who lived further away. Since lung development is nearly complete by age 18 years, an individual with a deficit at this time will probably continue to have less than healthy lung function for the remainder of his or her life.

We previously reported an association between community-average pollutant concentrations and 8-year lung-function growth.¹⁵ That result relied on comparisons in communities that had different concentrations of regional air pollution, and implicated many pollutants such as nitrogen dioxide, acid vapour, particulate matter with aerodynamic diameter less than 10 µm and 2.5 µm, and elemental carbon. Our present study builds on that result, and shows that in addition to regional pollution, local exposure to large roadways is associated with diminished lung-function development

	Lung function	8-year growth	
		Age 10 years	Age 18 years
	Difference* (95% CI)	Difference* (95% CI)	Difference* (95% CI)
FVC	Freeway distance		
	<500 m	-17 (-70 to 37)	-85 (-192 to 22)
	500-1000 m	-12 (-61 to 37)	-54 (-151 to 43)
FEV ₁	Freeway distance		
	<500 m	-23 (-73 to 28)	-121 (-219 to -23)
	500-1000 m	-32 (-78 to 14)	-93 (-183 to -4)
MMEF	Freeway distance		
	<500 m	-57 (-169 to 56)	-230 (-432 to -28)
	500-1000 m	-92 (-195 to 10)	-105 (-289 to 79)

* Difference in 8-year lung function or growth relative to children living >1500 m from a freeway.

Table 4: Cumulative effect of residential distance in the 1445 children with full 8-year follow-up

in children. We did not find any evidence that traffic effects varied depending on background air quality, which suggests that even in an area with low regional pollution, children living near a major roadway are at increased risk of health effects. Our results also suggest that children who live close to a freeway in a high pollution area experience a combination of adverse developmental effects because of both local and regional pollution.

We noted a larger freeway effect in boys than in girls, although the difference between sexes was not significant. By contrast, a cross-sectional European study²⁹ reported larger traffic effects on lung function in girls than in boys.²⁹ Several factors could explain this discrepancy in sex-specific effects between studies, from differences in specific air pollution mixtures and underlying population susceptibilities, to the general difficulty of comparisons between longitudinal and cross-sectional study effect estimates. In general, however, both studies show that lung function in children is adversely affected by exposure to traffic.

The concentrations of several pollutants are raised near major freeways. Daytime concentrations of black carbon, ultrafine particulate, and other exhaust pollutants have been reported to be high, but decline exponentially, within 500 m of a freeway,^{44,45} although night-time concentrations of ultrafine particulate remain above background concentrations for distances greater than 500 m from a freeway.⁴⁶ Some studies have reported increased traffic pollution, particularly nitrogen dioxide, at distances over 1000 m from a freeway.^{16,47-49} Elemental carbon, an indicator of pollution from diesel exhaust, varies with nearby high-traffic roads^{47,50,51} but can also be transported across large distances.⁵² Diesel exhaust is one of the primary contributors to particulate-matter concentrations in those communities most affected by traffic.⁵³ A pollutant such as elemental carbon could explain our reported health effects both locally and regionally.

Both regional ambient and ultrafine particulate matter present in high concentration in close proximity to roadways can elicit oxidative and nitrosative stress in the airways, which results in inflammation.^{54,55} Kulkarni and co-workers⁵² reported that traffic-related particulate matter was correlated with the amount of carbon in the airway macrophages of children, which in turn was associated with reductions in FEV₁, MMEF, and FVC. Chronic airway inflammation could produce our reported deficits in MMEF and FEV₁. Additional research is needed to identify the specific traffic pollutants that bring about health effects, and to elucidate the contribution of each pollutant to regional and local associations.

A strength of this study was the long-term, prospective follow-up of two large cohorts of children, with exposure and outcome data obtained consistently. However, as in any epidemiological study, our results could be confounded by one or more other factors related to both traffic and lung-function growth. Our results were robust

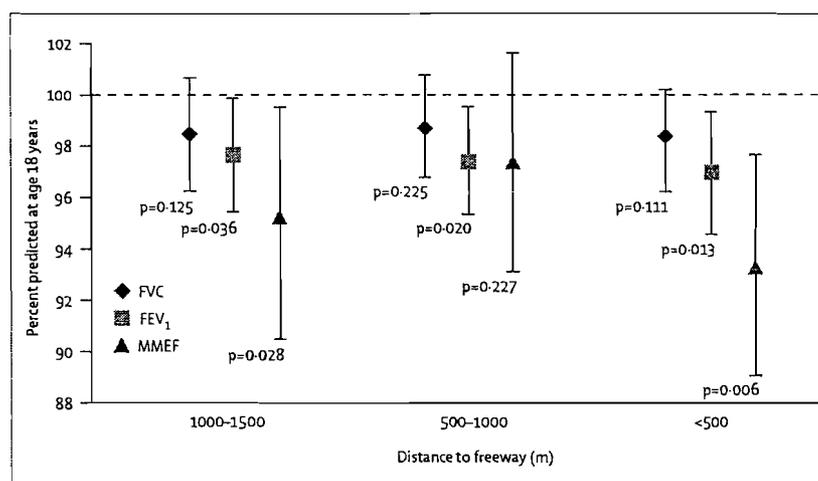


Figure: Percent-predicted lung function at age 18 years versus residential distance from a freeway. The horizontal line at 100% corresponds to the referent group, children living >1500 m from a freeway.

to adjustment for several factors, including socioeconomic status and indoor sources of air pollution, but the possibility of confounding by other factors still exists. Throughout the 8-year follow-up, we noted around an 11% loss of study participants per year. Participant attrition is a potential source of bias in cohort studies. We analysed the subset of children who were followed up for the full 8-year duration of the study and also noted significant traffic-effect estimates, which make participant loss an unlikely explanation for our results. We did not note a significant association between growth and model-based pollution from a freeway, despite large estimated deficits in the highest-exposure quartiles (table 1). However, we were restricted in detection of an association with model-based pollution from freeways because there was little variation in this measure within most of our study communities (webtable 2).

We have shown that residential distance from a freeway is associated with significant deficits in 8-year respiratory growth, which result in important deficits in lung function at age 18 years. This study adds to evidence that the present regulatory emphasis on regional air quality might need to be modified to include consideration of local variation in air pollution. In many urban areas, population growth is forcing the construction of housing tracts and schools near to busy roadways, with the result that many children live and attend school in close proximity to major sources of air pollution. In view of the magnitude of the reported effects and the importance of lung function as a determinant of adult morbidity and mortality, reduction of exposure to traffic-related air pollutants could lead to substantial public-health benefits.

Contributors

W J Gauderman, R McConnell, F Gilliland, E Avol, J Peters, M Jerrett, and N Kunzil participated in the writing of the manuscript. W J Gauderman, H Vora, K Berhane, D Thomas, and F Lurmann participated in the analysis of the data. All named authors took part in the interpretation of results, and approved the final version of the manuscript.

5



Conflict of interest statement

We declare that we have no conflict of interest.

Acknowledgments

This study was supported in part by the California Air Resources Board (contract A033-186), the National Institute of Environmental Health Sciences (P01ES11627 and P30ES07048), the US Environmental Protection Agency (R82735201), and the Hastings Foundation. We thank Cheryl Faucett for helpful discussions, members of our external advisory committee (Jonathan Sarnet, Nan Laird, Steve Rich, Petros Koutrakis, Gerhardt Coetzee) for important input, the school principals, teachers, students, and parents in each of the 12 study communities for their cooperation and the health testing field team.

References

- 1 Ware J, Ferris BJ, Dockery D, Spengler J, Stram D, Speizer F. Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. *Am Rev Respir Dis* 1986; 133: 834–42.
- 2 Dockery D, Speizer F, Stram D, Ware J, Spengler J, Ferris BJ. Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis* 1989; 139: 587–94.
- 3 Schwartz J. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. *Environ Res* 1989; 50: 309–21.
- 4 Raizenne M, Neas L, Damokosh A, et al. Health effects of acid aerosols on North American children: pulmonary function. *Am Rev Respir Dis* 1993; 147: A635.
- 5 Bascom R, Bromberg P, Costa D, et al. State of the art: health effects of outdoor air pollution, part 1. *Am J Respir Crit Care Med* 1996; 153: 3–50.
- 6 Bascom R, Bromberg P, Costa D, et al. State of the art: health effects of outdoor air pollution, part 2. *Am J Respir Crit Care Med* 1996; 153: 477–98.
- 7 Peters J, Avol E, Gauderman W, et al. A study of twelve southern California communities with differing levels and types of air pollution II. Effects on pulmonary function. *Am J Respir Crit Care Med* 1999; 159: 768–75.
- 8 Kunzli N, Lurmann F, Segal M, Ngo L, Balmes J, Tager IB. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen—results of a pilot study. *Environ Res* 1997; 72: 8–23.
- 9 Tager IB, Balmes J, Lurmann F, Ngo L, Alcorn S, Kunzli N. Chronic exposure to ambient ozone and lung function in young adults. *Epidemiology* 2005; 16: 751–59.
- 10 Frischer T, Studnicka M, Gartner C, et al. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med* 1999; 160: 390–96.
- 11 Jedrychowski W, Flak E, Mroz E. The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children. *Environ Health Perspect* 1999; 107: 669–74.
- 12 Horak FJ, Studnicka M, Gartner C, et al. Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian schoolchildren. *Eur Respir J* 2002; 19: 838–45.
- 13 Gauderman W, McConnell R, Gilliland F, et al. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med* 2000; 162: 1383–90.
- 14 Gauderman W, Gilliland F, Vora H, et al. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med* 2002; 166: 76–84.
- 15 Gauderman W, Avol E, Gilliland F, et al. The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med* 2004; 351: 1057–67.
- 16 Gauderman W, Avol E, Lurmann F, et al. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology* 2005; 16: 737–43.
- 17 Nicolai T, Carr D, Weiland SK, et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *Eur Respir J* 2003; 21: 956–63.
- 18 Venn A, Lewis S, Cooper M, et al. Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study. *Occup Environ Med* 2000; 57: 152–58.
- 19 Venn AJ, Lewis SA, Cooper M, Hubbard R, Britton J. Living near a main road and the risk of wheezing illness in children. *Am J Respir Crit Care Med* 2001; 164: 2177–80.
- 20 van Vliet P, Knappe M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res* 1997; 74: 122–32.
- 21 Hirsch T, Weiland SK, von Mutius E, et al. Inner city air pollution and respiratory health and atopy in children. *Eur Respir J* 1999; 14: 669–77.
- 22 Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. *Arch Environ Health* 1994; 49: 223–27.
- 23 Kim J, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B. Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am J Respir Crit Care Med* 2004; 170: 520–26.
- 24 Brauer M, Hoek G, Van Vliet P, et al. Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Respir Crit Care Med* 2002; 166: 1092–98.
- 25 Zmirou D, Gauvin S, Pin I, et al. Traffic related air pollution and incidence of childhood asthma: results of the Vesta case-control study. *J Epidemiol Community Health* 2004; 58: 18–23.
- 26 Delfino RJ. Epidemiologic evidence for asthma and exposure to air toxics: linkages between occupational, indoor, and community air pollution research. *Environ Health Perspect* 2002; 110 (suppl 4): 573–89.
- 27 Janssen NA, Brunekreef B, van Vliet P, et al. The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. *Environ Health Perspect* 2003; 111: 1512–18.
- 28 McConnell R, Berhane K, Yao L, et al. Traffic, susceptibility, and childhood asthma. *Env Health Perspect* 2006; 114: 766–72.
- 29 Brunekreef B, Janssen N, de Hartog J, Harssema H, Knappe M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. *Am J Epidemiol* 1997; 8: 298–303.
- 30 Wjst M, Reitmeir P, Dold S, et al. Road traffic and adverse effects on respiratory health in children. *BMJ* 1993; 307: 596–600.
- 31 Sugiri D, Ranft U, Schikowski T, Kramer U. The influence of large-scale airborne particle decline and traffic-related exposure on children's lung function. *Environ Health Perspect* 2006; 114: 282–88.
- 32 Kulkarni N, Pierse N, Rushton L, Grigg J. Carbon in airway macrophages and lung function in children. *N Engl J Med* 2006; 355: 21–30.
- 33 Hole D, Watt G, Davey Smith G, et al. Impaired lung function and mortality risk in men and women: findings from the Renfrow and Paisley prospective population study. *BMJ* 1996; 313: 711–15.
- 34 Knuiman M, James A, Davitini M, et al. Lung function, respiratory symptoms, and mortality: results from the Busselton Health Study. *Ann Epidemiol* 1999; 9: 297–306.
- 35 Schunemann H, Dorn J, Grant B, et al. Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. *Chest* 2000; 118: 656–64.
- 36 Schroeder E, Welch V, Couper D, et al. Lung function and incident coronary heart disease, the Atherosclerosis Risk in Communities Study. *Am J Epidemiol* 2003; 158: 1171–81.
- 37 Friedman G, Klatsky A, Siegelaub A, et al. Lung function and risk of myocardial infarction and sudden cardiac death. *N Engl J Med* 1976; 294: 1071–75.
- 38 Kannell W, Hubert H, Lew E, Jones J. Vital capacity as a predictor of cardiovascular disease: the Framingham Study. *Am Heart J* 1983; 105: 311–15.
- 39 Ashley F, Kannell W, Sorlie P, et al. Pulmonary function: relation to aging, cigarette habit, and mortality. The Framingham Study. *Ann Intern Med* 1975; 82: 739–45.
- 40 Peters J, Avol E, Navidi W, et al. A study of twelve southern California communities with differing levels and types of air pollution I. Prevalence of respiratory morbidity. *Am J Respir Crit Care Med* 1999; 159: 760–67.
- 41 Bensen P. CALINE4—A dispersion model for predicting air pollution concentrations near roadways. Sacramento: California Department of Transportation, 1989.
- 42 Berhane K, Gauderman W, Stram D, Thomas D. Statistical issues in studies of the long term effects of air pollution: the southern California Children's Health Study (with discussion). *Stat Sci* 2005; 19: 414–49.

6

- 43 Wang X, Dockery D, Wypij D, et al. Pulmonary function growth velocity in children 6 to 18 years of age. *Am Rev Respir Dis* 1993; 148: 1502-08.
- 44 Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc* 2002; 52: 1032-42.
- 45 Gilbert NL, Woodhouse S, Stieb DM, Brook JR. Ambient nitrogen dioxide and distance from a major highway. *Sci Total Environ* 2003; 312: 43-46.
- 46 Zhu Y, Kuhn T, Mayo P, Hinds WC. Comparison of daytime and nighttime concentration profiles and size distributions of ultrafine particles near a major highway. *Environ Sci Technol* 2006; 40: 2531-36.
- 47 Brauer M, Hoek G, van Vliet P, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. *Epidemiology* 2003; 14: 228-39.
- 48 Gilbert NL, Goldberg MS, Beckerman B, Brook JR, Jerrett M. Assessing spatial variability of ambient nitrogen dioxide in Montreal, Canada, with a land-use regression model. *J Air Waste Manag Assoc* 2005; 55: 1059-63.
- 49 Gonzales M, Qualls C, Hudgens E, Neas L. Characterization of a spatial gradient of nitrogen dioxide across a United States-Mexico border city during winter. *Sci Total Environ* 2005; 337: 163-73.
- 50 Fischer PH, Hoek G, Van Reeuwijka H, et al. Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam. *Atmos Environ* 2000; 34: 3713-22.
- 51 Roorda-Knape MC, Janssen NAH, de Hartog JJ, Van Vliet PHN, Harssema H, Brunekreef B. Air pollution from traffic in city districts near major motorways. *Atmos Environ* 1998; 32: 1921-30.
- 52 Khan A, Li J, Husain L. Atmospheric transport of elemental carbon. *J Geophys Res* 2006; 111: D04303.
- 53 Manchester-Neesvig J, Schauer J, Cass G. The distribution of particle-phase organic compounds in the atmosphere and their use for source apportionment during the southern California Children's Health Study. *J Air Waste Manag Assoc* 2003; 53: 1065-79.
- 54 Li N, Hao M, Phalen RF, Hinds WC, Nel AE. Particulate air pollutants and asthma. A paradigm for the role of oxidative stress in PM-induced adverse health effects. *Clin Immunol* 2003; 109: 250-65.
- 55 Nel A. Atmosphere. Air pollution-related illness: effects of particles. *Science* 2005; 308: 804-06.

TRAN



MONTGOMERY COUNTY PLANNING BOARD
THE MARYLAND-NATIONAL CAPITAL PARK AND PLANNING COMMISSION

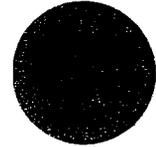
OFFICE OF THE CHAIRMAN

February 7, 2007

GO
CC
SBF
LL
MM
JZ

The Honorable Marilyn Praisner
The Honorable Philip Andrews
The Honorable Duchy Trachtenberg
The Honorable Mark Elrich
Montgomery County Council
Rockville, Maryland 20850

026674



Dear Council Members:

In your letter of February 1, 2007, you requested certain information about the numbers of residents living within 500 meters of the ICC, and requested that the Planning Board not transfer any additional land required for the ICC right-of-way until such time as the implications of a research article published in the *Lancet* can be discussed with the Council.

Before the Board agrees to postpone any decisions regarding the transfer of land to the State for the ICC project until the implications of the *Lancet* article can be analyzed and discussed with the Council, we request clear direction from the Council and Executive. Such action would involve a significant change in county policy from decisions reached by the Planning Board and the County Council in 2005 and 2006 regarding the Intercounty Connector.

As you know, the Board tabled some land transfers until it can be satisfied that every effort has been made to ensure proper water quality measures are taken in the Special Protection Areas of Upper Rock Creek and Paint Branch, and to place the ICC's western maintenance facility on Casey 6 instead of Casey 7 to implement better the Shady Grove Master Plan. Both issues were raised in the mandatory referral process. The agencies and builder involved have been engaged in intensive discussions to resolve both issues, which should soon enable the Board to remove the motion to transfer the land from the table, perhaps as early as this week.

The implications of the *Lancet* article and many other studies of the effects of exposure to air pollutants for residents living near highly trafficked roadways is of considerable interest as we develop the new growth policy for the county and a Master Plan for the Environment and Energy, as well as future area master plans. The *Lancet* study's prediction model diagrammed in the study figure indicates that the 3% predicted reduction in forced expiratory volume (FEV₁) for children living within 500 meters of a freeway is nearly identical to that of children living within 500 to 1,000 meters of a freeway, as well as those

RECEIVED
MONTGOMERY COUNTY
COUNCIL
2007 FEB -8 PM 1:36

8

The Honorable Marilyn Praisner
The Honorable Philip Andrews
The Honorable Duchy Trachtenberg
The Honorable Mark Elrich
February 7, 2007
Page Two

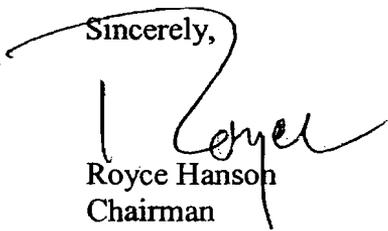
living within 1,000 to 1,500 meters of a freeway. These results indeed require further investigation. Adverse health consequences are also associated with the location of schools near busy highways, as pointed out by one of the commentators on the article; a point you made in your letter. There are several other public health issues associated with development patterns. Prominent among them is the issue of obesity. All of these issues involve trade-offs with other objectives that are best addressed in a broad growth policy or master plan context.

With regard to air quality issues related to the ICC, the microscale air quality analysis conducted in 2004 for the Intercounty Connector environmental impact statement predicted that localized carbon monoxide concentrations would increase at residential receptors along the highway, but not to levels that would exceed the State and National Ambient Air Quality Standards (S/NAAQS), the operating standards for such decisions.

We will develop the information you have requested on the numbers of residential units within both 0.5 and 1.5 kilometers on either side of our entire master planned network of freeways and controlled access highways in the county. The most efficient way to produce this information will be to prepare it in conjunction with the new county growth policy recommendations. This will avoid need for an adjustment in the work program, slowing production of the growth policy. As soon as the data are produced, we will transmit them to you and be prepared to discuss their implications in the work sessions on growth policy.

I note that you plan to ask the Health Department for comment. It might be useful for the Director to inquire whether the National Institute of Environmental Health might be persuaded to fund a county project through an appropriate research institution. We would be interested in receiving a copy of any comments from the Health Department.

Sincerely,



Royce Hanson
Chairman

Attachment A
Health Effects of Residences Located Proximate to Freeways

(excerpted from ICC Project Status Report #10 to the Planning Board)

On two occasions, the County Council has expressed concern that the ICC will introduce adverse localized air quality effects, citing medical journal studies identifying a link between residential proximity to a freeway and impaired lung function. In spring 2007, the Council requested information regarding the number of residences within 0.5 KM or 1.5 KM of the ICC. Staff has completed this analysis, as summarized in Table 1.

Table 1. Number of Dwelling Units (DU) Located Proximate to County Freeways

Freeway	Length (Miles)	Number of DU within 0.5 KM	Number of DU within 1.5 KM
I-370/ICC	16	12,000	29,000
I-270	24	39,000	50,000
I-495	14	11,000	36,000

As indicated in Table 1, the density of residential development near the ICC is generally about the same or a little lower than that near the other freeways in the County. The correlation between residence/freeway proximity and health effects requires further study. Staff proposes to consider this further during our review of sustainability measures in FY 08.

In the meantime, however, we will continue to implement master plan recommendations regarding both careful introduction of the ICC into the communities through which it has been planned, as well as the careful development of residential properties adjacent to existing freeways, such as recommended in the Shady Grove Sector Plan.

TRAN



Martin O'Malley, Governor
Anthony G. Brown, Lt. Governor

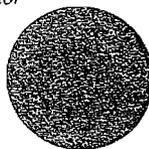
John D. Porcari, Secretary
Neil J. Pedersen, Administrator

Maryland Department of Transportation

GO
CC
SEF
L

November 30, 2007

032120



2007 DEC -5 AM 9:27

RECEIVED
MONTGOMERY COUNTY
COUNCIL

The Honorable Marilyn J. Praisner
President, Montgomery County Council
100 Maryland Avenue
Rockville MD 20850

Dear Council President Praisner:

Thank you for your October 8 memorandum to State Highway Administrator Neil J. Pedersen regarding the Intercounty Connector (ICC). Mr. Pedersen received your memo and asked me to respond to the questions, as I am directly involved in the specific details on this project. Questions directed to Maryland-National Capital Park and Planning Commission (MNCPPC) staff will be answered by them in a separate letter. For your convenience, your questions (in bold) are followed by our answers.

Public Health Issues

- **What modeling of vehicle emissions and their impact on air quality and health was done to assess the likely impact on people who live within 500 feet of the ICC right-of-way? 1,000 feet? 1,500 feet?**

The ICC study team conducted local air quality analyses in accordance with U.S. Environmental Protection Agency (EPA), Federal Highway Administration (FHWA), and SHA guidelines, to protect public health, especially sensitive populations such as asthmatics, children, and the elderly. The results of these analyses are included in the Air Quality Technical Report (AQTR) and summarized in the Final Environmental Impact Statement (FEIS). Although these analyses did not specifically address 500 feet, 1,000 feet, or 1,500 feet limits, the locations selected for these studies included residences, schools, and other sites with sensitive populations within these limits.

- **If any modeling was done, please be specific about when and where.**

Air quality modeling was performed as part of the National Environmental Policy Act (NEPA) studies. The studies included the analysis of carbon monoxide (CO), fine particulate matter (PM_{2.5}), and mobile source air toxics (MSAT).

(11)

The CO analysis, set forth in detail in the AQTR, was performed to determine concentrations at residences, schools, daycare centers, and other locations where there are sensitive populations immediately adjacent to the ICC's right-of-way. In addition, areas immediately adjacent to high traffic intersections were studied. The analyses performed demonstrated that CO impacts resulting from the ICC Selected Alternative will not result in a violation of the National Ambient Air Quality Standards (NAAQS). As discussed on page III-123 of the Record of Responses to Public Comments, which was released with the Record of Decision (ROD) and is available on the project website at www.iccproject.com, "these standards [NAAQS] are set by EPA based on the requirements of the Clean Air Act. The Clean Air Act requires these limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly." The CO analysis demonstrated that the ICC project conforms to the Clean Air Act since it will not cause or contribute to any new localized violations of the CO NAAQS nor will it increase the frequency or severity of any existing violations.

For the PM_{2.5} analysis, a qualitative hot-spot analysis was conducted. For the comparison approach, the anticipated traffic volumes on the ICC were compared to those on other major roadways near existing air quality monitors. The Muirkirk PM_{2.5} monitoring site was selected as the most appropriate surrogate monitor because the traffic volume and truck percentage near the Muirkirk monitor was similar to that expected on the ICC in 2010. This monitoring site is also close to the ICC study area and has similar land use and terrain characteristic. A PM_{2.5} trends analysis for the Washington D.C./Maryland/Virginia non-attainment area showed significant anticipated future decreases from direct on-road mobile sources through 2010 and thereafter. Accordingly, the project-level hot-spot analysis found that the ICC project would not cause or contribute to a new violation of the PM_{2.5} NAAQS, or increase the frequency or severity of a violation. The manner in which the PM_{2.5} hot-spot analysis was carried out and the conclusions from that analysis are detailed in Attachment H of the ROD. A sensitivity analysis was performed by FHWA to evaluate whether new 2006 standards for PM_{2.5} can be met. These studies concluded that new, more conservative standards will be met.

A qualitative project-level MSAT analysis was performed for the ICC no-action and build alternatives. This analysis was a qualitative analysis because modeling techniques are not currently available to perform a quantitative analysis and determine concentrations at specific locations. The results of this analysis were included in the FEIS and AQTR. For the MSAT analysis, total emissions were estimated for the six priority MSATs in the affected area of the ICC. The pollutants studied are Benzene, Acrolein, Formaldehyde, 1,3-Butadiene, Acetaldehyde, and diesel exhaust (diesel exhaust gases and diesel particulate matter). The ICC study area considered in this MSAT study is located in Montgomery County and Prince George's County, north of Washington, D.C. It extends from I-270 to US 1 and from I-495 (Capital Beltway) to the Patuxent River. The analysis showed that MSAT emissions in the design year (2030) will decrease greatly from current conditions for both the no-action and build alternative. The amount of the decrease varies from 67 percent to 92.5 percent, depending on MSAT considered.

In addition to the localized pollutant analyses discussed above, the Metropolitan Washington Council of Governments (MWCOCG), the local planning organization (MPO) has performed a regional air quality analysis which included the ICC. As discussed on page III-123 of the Record of Responses to Public Comments, "the regional air quality analysis performed by MWCOCG shows that the region with the ICC conforms to the region's air quality implementation plan's purpose of eliminating or reducing the severity and number of violations of the NAAQS and achieving expeditious attainment of such standards, including the NAAQS for CO, ozone, and PM_{2.5}."

The NAAQS for CO and PM_{2.5} were set to protect the public health and the ICC will not cause a violation of these standards. There are no NAAQS for MSATs. As discussed on page III-123 of the Record of Responses to Public Comments, "documentation associated with the MSAT analysis did include a discussion of and citation for health related information, as available from EPA at the time the analysis was completed. As stated in the analysis [and referred to by commenters], FHWA relies on EPA for the review and interpretation of air pollution research and associated health effects. The FHWA believes that EPA, and its established procedures and use of panels of national scientific experts, is in the position to prepare and present credible scientific evidence. In the FEIS, FHWA provided a summary of the scientific consensus of the health effects of the six pollutants that EPA has identified as the priority mobile source air toxics, and conducted an emissions analysis for these six pollutants. This discussion begins on page IV-318 of the FEIS."

- **If no modeling was done, why not and do you intend to do any? If not, why not?**

Please see above for discussion of modeling.

- **What, if any, requirements is SHA subject to regarding the establishment of baseline measures for current air quality and current emissions near the ICC right-of-way in order to measure ICC impact?**

The subject of air quality monitoring was addressed on page III-256 of the Record of Responses to Public Comments as follows: "The lead agencies [FHWA, SHA, etc.] are not required to site monitors specifically for measuring the impacts of highway emissions for conformity purposes. The lead agencies are also not required to place new monitors specifically to monitor suspected hot spot locations along a proposed roadway. Air quality monitoring stations are established by state air agencies, with oversight from EPA. The FHWA and SHA have no authority to enforce or review the compliance of any individual monitoring site with the criteria spelled out in 40 CFR Part 58."

- **What monitoring was done of the current levels of air quality within 500 feet of the ICC right-of-way? 1,000 feet? 1,500 feet? Please be specific about any monitoring that was done? If none was done, why not? Does SHA intend to do any? If not, why not?**

Monitoring in addition to that provided by Maryland Department of the Environment (MDE) is not required as part of the environmental and conformity of air quality analyses. Please see responses to previous questions as well.

- **What is the age distribution and number of people at respiratory risk adjacent to the proposed route of the ICC?**
- **Using those population numbers (from census or other reliable data), in conjunction with the increased morbidity and mortality risk, does SHA have epidemiologically justifiable estimates of how many additional deaths and cases of heart and lung disease might be expected among Montgomery County residents from their exposure to the ICC-related air pollution from living, working, or going to school along the proposed route of the ICC?**
- **How many people who already have existing heart or lung problems are currently living in the closest zones of highest risk?**
- **Additionally, what is the estimate of how many pregnancies can be expected each year in the ICC risk zones?**

Health-related impacts such as air, water, and noise pollution were considered during the ICC's study phase (see FEIS Chapter IV for summaries of each of these studies). There is no methodology that exists to quantitatively evaluate the adverse health impacts as described above. A person's exposure to elements varies significantly and is dependent on many factors, including their travel patterns, habits, and locations where they spend most of their time.

Environmental Issues

- **What is the specific cost of each environmental mitigation action that SHA is taking? Please provide a list of each action and its cost.**

Please find attached a list of the ICC's mitigation projects with the cost estimates for each.

- **Which projects are considered environmental stewardship and what are the criteria for that designation?**

Please find attached a list of the ICC's environmental stewardship (ES) projects. The ES package for the ICC was developed through analyses of the local natural, community, and cultural needs and a substantial level of coordination with local, State, and federal resource agencies, individuals, the public, and community groups. The ES program provides a comprehensive collection of improvement projects that meet the natural and community/cultural needs of the ICC study area.

 <p style="text-align: center;">2008 Regular Session bill information current as of November 23, 2008 - 0:20 a.m.</p>									
Sponsors	Title	Synopsis	History	Sponsor List	Subjects	Statutes	Documents	Another Session	Another Bill

HOUSE BILL 1595

File Code: Environment

NO SIMILAR bill in
2009

Sponsored By:

Delegates Mizeur, Kaiser, Ali, Barnes, Beidle, Bobo, Bromwell, Carr, Carter, V. Clagett, Frush, Gutierrez, Guzzone, Healey, Heller, Hixson, Holmes, Hubbard, Hucker, Ivey, Kullen, Lafferty, Montgomery, Nathan-Pulliam, Niemann, Oaks, Pena-Melnyk, Ramirez, Rice, Ross, Schuh, Schuler, Stein, Stukes, F. Turner, V. Turner, Valderrama, and Vaughn

Entitled:

Intercounty Connector - Public Health Impacts from Air Pollution - Assessment

Synopsis:

Prohibiting the State and the Maryland Transportation Authority from expending specified funds for the Intercounty Connector until a specified assessment is completed; specifying parameters for the assessment; requiring the Department of Transportation and the Department of the Environment jointly to create models and to quantify emissions, impacts, and risks, as specified; requiring an assessment of impacts, an analysis, publication of results, public hearings, public comments, and written responses, as specified; etc.

History by Legislative and Calendar Date

Legislative date is used to record history occurring in the Chambers otherwise Calendar date is used. ?

House Action

- 3/3 First Reading House Rules and Executive Nominations
- 3/7 Re-referred Environmental Matters
- 3/10 Hearing 3/19 at 1:00 p.m.
- 3/22 Unfavorable Report by Environmental Matters

Senate Action

No Action

(15)

Sponsored by:

Delegate Heather R. Mizeur, District 20
Delegate Saqib Ali, District 39
Delegate Ben Barnes, District 21
Delegate Pamela Beidle, District 32
Delegate Elizabeth Bobo, District 12B
Delegate Eric M. Bromwell, District 8
Delegate Alfred C. Carr, Jr., District 18
Delegate Jill P. Carter, District 41
Delegate Virginia P. Clagett, District 30
Delegate Barbara Frush, District 21
Delegate Ana Sol Gutierrez, District 18
Delegate Guy Guzzone, District 13
Delegate Anne Healey, District 22
Delegate Henry B. Heller, District 19
Delegate Sheila E. Hixson, District 20
Delegate Marvin E. Holmes, Jr., District 23B
Delegate James W. Hubbard, District 23A
Delegate Tom Hucker, District 20
Delegate Jolene Ivey, District 47
Delegate Anne R. Kaiser, District 14
Delegate Sue Kullen, District 27B
Delegate Stephen W. Lafferty, District 42
Delegate Karen S. Montgomery, District 14
Delegate Shirley Nathan-Pulliam, District 10
Delegate Doyle L. Niemann, District 47
Delegate Nathaniel T. Oaks, District 41
Delegate Joseline A. Pena-Melnyk, District 21
Delegate Victor R. Ramirez, District 47
Delegate Craig L. Rice, District 15
Delegate Justin D. Ross, District 22
Delegate Steven R. Schuh, District 31
Delegate Todd Schuler, District 8
Delegate Dana Stein, District 11
Delegate Melvin L. Stukes, District 44
Delegate Frank S. Turner, District 13
Delegate Veronica Turner, District 26
Delegate Kriselda Valderrama, District 26
Delegate Michael L. Vaughn, District 24

Bill indexed under the following Subjects:

Administrative Agencies -see also- Electronic Government
Environment, Department of
Environmental Matters -see also- Conserv; Nat Resrce; Pollut
Health -see also- Mental Health
Hearings
Highways
Montgomery County
Motor Vehicles -see also- Aband Veh; Ambulances; Buses; etc.
Pollution -see also- Sediment Control
Prince George's County
Publications -see also- Maryland Register
Public Information
Reports
State Bonds
Transportation, Department of

(16)

Transportation-see also- Aircraft; Airports; Boats; etc

Bill affects the following Statutes:

Transportation
(4-321 , 4-321.1)

Documents:

Bill Text (Displayed in PDF Format): [First Reading](#), Third Reading, Enrolled

Fiscal and Policy Note (Displayed in PDF Format): [Available](#)

Amendments: None offered

Top	Sponsors	Title	Synopsis	History	Sponsor List	Subjects	Statutes	Documents	Another Session	Another Bill
---------------------	--------------------------	-----------------------	--------------------------	-------------------------	------------------------------	--------------------------	--------------------------	---------------------------	---------------------------------	------------------------------

(17)

Potential Health Effects Associated with Residential Proximity to Freeways and Primary Roads: Review of Scientific Literature, 1999–2006

Vickie L. Boothe
Derek G. Shendell, D.Env, M.P.H.

Abstract

This review presents epidemiologic evidence of adverse health effects associated with residential proximity to traffic.

Of the 29 peer-reviewed studies that met the authors' defined criteria, 25 reported statistically significant associations with at least one adverse health effect across a broad range of exposure metrics and diverse geographical locations. Specific pollutants contributing to the associated health effects could not, however, be identified, and uncertainties existed because of the lack of individual exposure assessments that could rule out confounding by other factors. Improved exposure assessments and future studies should be considered for better identification of contributing pollutants and mechanisms of action. In the meantime, additional policies, additional regulations, and improved land use and urban planning can better protect the public and limit exposure, especially for vulnerable populations such as pregnant women, children, and the elderly.

Introduction

During the 1970s and 1980s, environmental regulations substantially reduced emissions from industry and stationary sources. Automobiles and other road traffic (mobile sources) became the most prominent contributors to urban air pollution in many areas of the United States (U.S. Environmental Protection Agency, 1994). Traffic emissions include nitrogen oxides (NO_x), carbon monoxide (CO), volatile organic compounds (VOCs) including benzene and 1,3 butadiene, and particulate matter (PM). In addition, toxic air pollutants including aldehydes, polycyclic aromatic hydrocarbons

(PAHs), and metals can adhere to traffic-generated particulate matter (Oberdörster, 2001).

Studies characterizing distributions of fresh vehicular exhaust documented that concentrations were higher near roadways but diminished to near background levels within 150–300 meters (m) (Gilbert, Goldberg, Beckerman, Brook & Jerrett 2005; Zhu, Hinds, Kim, & Sioutas, 2002; Zhu, Kuhn, Mayo, & Hinds, 2006). The steep declines in concentrations were attributed to evaporation of volatile constituents, atmospheric dispersion, and coagulation (Zhu, Hinds, Kim, Shen, & Sioutas, 2002).

Adverse health effects have been associated with residential proximity to traffic. Traffic constituents potentially affecting health include ultrafine (PM_{0.1}) and fine (PM_{2.5}) particles which can penetrate deep into the lungs (Oberdörster, 2001) and have been associated with respiratory, pulmonary, and cardiovascular morbidity and mortality (Brunekreef & Holgate, 2002; Dockery 2001; Pope, Burnett, Thun, Calle, & Kerwsky, 2002). Exposures to traffic-related PM and CO have been associated with adverse birth outcomes (Ritz, Yu, Fruin, Chapa, Shaw, & Harris, 2002; Ritz & Yu, 1999; Ritz, Yu, & Fruin, 2000), which may lead to increased childhood morbidity and mortality and increased risk of hypertension and coronary heart disease in adulthood (Barker, 1995; Osmond & Baker, 2000). Diesel emissions have been associated with effects including lung cancer (Lloyd & Cockette, 2001; Mauderly, 1990; Sydbom, Blomberg, Parnia, Stenfors, Sandstrom, & Dahlen, 2001) and pulmonary/respiratory disorders (Nel, Diaz-Sanchez, & Li 2001). In addition, traffic emissions contain many known and suspected carcinogens. Because of their potency and high concentrations, most of the cancer risk has been attributed to benzene, 1,3 butadiene, and particle-bound PAHs (Rosenbaum, Axelrad, Woodruff, Wei, Ligocki, & Cohen 1999). Chronic benzene exposures have been linked to both structural and chromo-

TABLE 1a

Research on Traffic Proximity and Parent-Reported Adverse Respiratory Effects—Key Results

Reference	Location	Study Population (N)	Health Effects Assessed	Exposure Metric	Health Effects with Significant Associations	Distance to Traffic	Traffic Density	Adjusted Odds Ratio	95% CI
Venn et al., 2001	Nottingham, England	4–11 year olds (6,147)	Parent-reported wheeze prevalence	Postal code distance and daily traffic vehicles/day	Parent-reported wheeze prevalence	150 m	10,000–100,000 vehicles/day	1.08	1.00–1.16
		11–15 year olds (3,709)	Parent-reported wheeze prevalence	Postal code distance and vehicles/day	Parent-reported wheeze prevalence	150 m	10,000–100,000 vehicles/day	1.16	1.02–1.32
Janssen et al., 2003	20 city districts, Netherlands	7–12 year olds (2,083)	Parent-reported wheeze, nasal symptoms, lung function, conjunctivitis, itchy rash, bronchitis asthma, hay fever, eczema, allergy	Residential distance to truck traffic, trucks/day	Parent-reported conjunctivitis	500 m	5,190–22,326 trucks/day	2.57	1.00–6.58
					Parent-reported itchy rash	500 m	5,190–22,326 trucks/day	2.08	1.20–5.58
					Parent-reported current wheeze	50 m	>99,500 vehicles/day	1.67	1.07–2.58
					Parent-reported cough prevalence	50 m	>99,500 vehicles/day	1.62	1.62–2.27
Nicolai et al., 2003	Munich, Germany	4–6 year olds & 9–11 year olds (7,508)	Cough, current asthma wheeze, lung function, bronchial hyper-reactivity	Average daily traffic count & distance to residence	Parent-reported asthma prevalence	50 m	>99,500 vehicles/day	1.79	1.05–3.05
					Parent-reported current wheeze	50 m	>99,500 vehicles/day	1.67	1.07–2.58
					Parent-reported cough prevalence				
Lewis et al., 2004	United Kingdom	4–6 yr olds (11,562)	Self-reported wheeze, asthma prevalence, medication use	Residential distance to main road	None	50 m	>99,500 vehicles/day	1.62	1.62–2.27
						150 m	N/A	No statistically significant results	
Gauderman et al., 2005	10 of 12 Southern California Communities in the Children's Health Study	4th graders (average age = 10 years) (208)	Self-reported wheeze, asthma prevalence, asthma medication use	Residential distance to freeway	Parent-reported asthma prevalence	150 m	N/A	1.89	1.19–3.02
					Parent-reported wheeze	150 m	N/A	1.59	1.06–2.36
					Parent-reported wheeze with exercise	150 m			
Ryan et al., 2005	Cincinnati, Ohio	≤1 year old (622)	Parent-reported wheeze	Residential distance to freeway, state route with speed >50 mph; bus or state route with speed <50 mph	None	400 m	N/A	2.57	1.50–4.38
					None	100 m	Freeway	No statistically significant results	
					Parent-reported wheeze	100 m	State route, speed >50 mph	No statistically significant results	
							Bus/state route, speed <50 mph	2.50	1.15–5.42

Notes:
Relative risk (RR) by comparison with low- or no-exposure categories.
N/A = not available (i.e., not reported).

somal anomalies in humans and increased incidence of leukemia in individuals occupationally exposed (Finkelstein, 2000; Paxton, 1996; Rinsky, Smith, et al., 1987; Rinsky, Young, & Smith, 1981).

This paper summarizes available scientific findings published in peer-reviewed journals in English on health effects associated with defined residential proximity to traffic from January 1999 through June 2006.

Methods

An electronic search was performed on PubMed. Search terms included “traffic” and “traffic emissions” in combination with any of the following terms: “asthma,” “adverse birth outcomes,” “birth weight,” “childhood cancer,” “mortality,” and “health effects.” A total of 139 unique records were returned. Twenty-nine epidemiological studies defined residential proximity to traffic met specific inclusion criteria.

Fifty-four records were excluded because they were exposure characterizations or assessments and did not address health effects; 26 were based on exposures in settings other than residences, such as workplaces or schools; 16 were based on air pollution monitoring or modeling; eight were risk assessments; three were animal studies; and three were not in English. It should be noted these criteria excluded some important studies conducted since 1999, such

TABLE 1b**Research on Traffic Proximity and Self-Reported Adverse Respiratory Effects—Key Results**

Reference	Location	Study Population (N)	Health Effects Assessed	Exposure Metric	Health Effects with Significant Associations	Distance to Traffic	Traffic Density	Adjusted Odds Ratio	95% CI
Garshick et al., 2003	Southeastern Massachusetts	U.S. veterans 60.6 ± 12.8 years old 2,628	Self-reported persistent wheeze, chronic cough, chronic phlegm	Average daily traffic count & distance to residence	Self-reported persistent wheeze	50m	9,351 vehicles/day median	1.31	1.00–1.71
							>10,000 vehicles/day	1.71	1.22–2.40
Heinrich et al., 2005	East Germany	18–79 year olds (6,896)	Self-reported wheeze, nocturnal coughing, current asthma, hay fever, chronic, bronchitis, allergic sensation	Self-reported residential proximity to traffic intensity	Self-reported chronic bronchitis	Street of residence	Self-reported extremely or considerably busy roads	1.36	1.01–1.83
Schikowski et al., 2005	Dortmund, Duisburg, Essen, Gelsenkirchen, & Herne, Germany	Women 54–55 years old (4,757)	Frequent cough, chronic bronchitis, COPD, forced expiratory volume, forced vital capacity	Average daily traffic count & distance to residence	Self-reported COPD	100 m	>10,000 vehicles/day	1.79	1.06–3.02
					Self-reported frequent cough	100 m	>10,000 vehicles/day	1.24	1.03–1.49
Venn et al., 2005	21 districts of Jimma, Ethiopia	Children and adults	Self-reported wheeze, rhinitis, eczema, dust mite sensitivity	Residential distance to road and density of vehicles/12 hours	Self-reported wheeze	150 m	653 vehicles per 12 hours median	1.17 per 30 m	1.01–1.36
McConnell et al., 2006	13 California communities	5–7 year olds (4,762)	Self-reported asthma prevalence, asthma medication use, wheeze	Residential distance to freeways, highways, & arterial roads	Lifetime asthma	75 m	N/A	1.29	1.01–1.86
					Asthma prevalence	75 m	N/A	1.50	1.16–1.95
						75–150 m	N/A	1.33	1.02–1.72
					Asthma prevalence with no family asthma history	75 m	N/A	2.46	1.48–4.09
Current wheeze with no family asthma history	75 m	N/A	2.74	1.71–4.39					

Notes:
Relative risk (RR) by comparison with low- or no-exposure categories.
N/A = not available (i.e., not reported).

as that by Wilhelm and Ritz (2005), which used residential proximity to central-site ambient-air-monitoring stations to evaluate potential associations between adverse birth outcomes and traffic-related pollutants.

Literature Review Results

Respiratory Effects

Nineteen studies evaluated residential proximity to traffic and respiratory effects. The results for studies of parent-reported respiratory effects are summarized in Table 1a, those for self-reported respiratory effects are summarized in Table 1b, and those for physician-diagnosed respiratory effects are summarized in Table 1c. Of the 19 studies, 10 examined respiratory symptoms such as wheeze, frequent coughs, and chronic phlegm; four examined indicators of asthma severity including hos-

pitalizations and doctor visits; and six investigated the relationship between residential proximity to traffic and prevalence of asthma.

Seven of the 10 studies examining self- or parent-reported respiratory symptoms reported statistically significant associations between proximity to traffic and wheeze (McConnell et al., 2006; Garschick, Laden, Hart, & Caron, 2003; Gauderman et al., 2005; Nicolai et al., 2003; Ryan et al., 2005; Venn, Lewis, Cooper, Hubbard, & Britton, 2001; Venn, Yemaneberhan, Lewis, Parry, & Britton, 2005). Persistent or current wheeze was found to be associated with residential proximity within 50, 75, and 150 m of busy roads (McConnell et al., 2006; Gauderman et al., 2005; Venn et al., 2005; Garschick et al. 2003; Janssen et al. 2003; Venn et al. 2001). McConnell and co-authors (2006) reported statistically significant associations for residential proximity within 75 m

and between 75 and 150 m but not for residences at distances of 150–300 m or greater. Heinrich and co-authors (2005) reported only marginal increases in wheeze, nocturnal coughing attacks, and hay fever in a group of East German adults with self-reported residential proximity to traffic. Lewis and co-authors (2004) reported no statistically significant associations between residential proximity to traffic within 150 m and self-reported asthma prevalence, medication usage, or wheeze in children in the United Kingdom.

Results were mixed for five studies examining associations between proximity to traffic and respiratory-related doctor visits and hospitalizations. In a study of San Diego children, English and co-authors (1999) reported that doctor visits for asthma were associated with traffic density at the second quintile (5,500–9,000 cars/day) and 95th percentile (>41,000 cars/day) within

TABLE 1c**Research on Traffic Proximity and Physician-Diagnosed Respiratory Effects—Key Results**

Reference	Location	Study Population (N)	Health Effects Assessed	Exposure Metric	Health Effects with Significant Associations	Distance to Traffic	Traffic Density	Adjusted Odds Ratio	95% CI
English et al., 1999	San Diego, California	≤14 year olds w/ asthma (5,996)	Asthma prevalence and asthma doctor visits	Average daily traffic vehicles/day & distance to residence	Asthma doctor visits	550 ft	5,500–9,000 vehicles/day	2.14	1.10–4.16
							>41,000 vehicles/day	2.91	1.28–6.91
Wilkinson et al., 1999	North Thames London, England	5–14 year olds w/asthma & respiratory illnesses (9,214)	Asthma and respiratory illness hospitalizations 1992–1994	Postal code centroid distance to road and peak hourly traffic vehicles/hour	None	150 m	>1,000 vehicles/hour	No statistically significant results	
Lin et al., 2002	Erie County, New York	≤14 year olds w/ asthma (417)	Asthma hospital admissions	Average vehicle miles traveled & distance to residence	Asthma hospital admissions	200 m	>4,043 vehicle miles traveled	1.93	1.13–3.29
				Residential distance to heavy truck traffic	Asthma hospital admissions	200 m	≥1% heavy trucks	1.43	1.03–1.99
Lwebuga-Mukasa et al., 2004	16 ZIP codes near Peace Bridge on U.S./Canada Border	≥18 yr olds (13,910)	Asthma hospitalizations and outpatient visits 1991–1996	Pre- and post-NAFTA traffic volumes	None	N/A	N/A	No statistically significant results	
Zmirou et al., 2004	France	0–3 year olds (434)	Diagnosed asthma incidence, asthma prevalence	Lifetime average time weighted traffic density (vehicles/meter)	Diagnosed asthma incidence	300 m	>30 vehicles per day per meter	2.28	1.14–4.56
Gordian et al., 2006	Anchorage, Alaska	5–7 year olds (756)	Asthma prevalence	Average daily traffic per meter vehicle within buffer around residence	Asthma prevalence in children with no family asthma history	100 m	40,000–80,000 vehicle meters	2.43	1.23–5.28
							>80,000 vehicle meters	5.43	2.08–13.74
Smargiassi et al., 2006	Montreal, Canada	≥60 year olds (35,309)	Respiratory hospitalizations	Residences along roads and density of vehicles during 3-hour daily peak	Respiratory hospitalizations compared to other diagnostic groups	N/A	>3,160 vehicles/3-hour peak	1.18*	1.06–1.31
Sugiri et al., 2006	East & West Germany	5–7 yr olds (2,574)	Total lung capacity, airway resistance	Average daily traffic density kilometers/day & distance to highway	Total lung capacity	50 m	216–214 km/day mean	1.07	1.06–1.09
					Airway resistance	50 m	216–214 km/day mean	1.02	1.00–1.03

Notes:
Relative risk (RR) by comparison with low- or no-exposure categories.
N/A = not available (i.e., not reported).

550 feet (ft) of residences; this association was strongest for girls. Lin and co-authors (2002) reported increased risk of asthma hospitalization for children living in New York neighborhoods with heavy truck traffic and increased traffic density within 200 m of their homes. Smargiassi and co-authors (2006) reported increased risks of respiratory-related hospitalizations for older adults living in areas of Montreal with high traffic volumes. Wilkinson and co-authors (1999), however, using a postal-code centroid within 150 m of a busy road, reported

no association between respiratory-related hospital visits and residential proximity for school children in London. Lwebuga-Mukasa and co-authors (2004) also did not find a statistically significant association for respiratory-related children's hospital visits when they examined the change in traffic patterns before and after implementation of the North American Free Trade Agreement (NAFTA) near the Peace Bridge on the U.S./Canada Border.

Four of six studies examining asthma prevalence reported statistically significant findings

with residential proximity within 75, 100, 150, and 300 m of dense traffic across geographically diverse locations including Alaska, California, and France (Gordian, Haneuse, & Wakefield, 2006; McConnell et al., 2006; Gauderman et al., 2005; Zmirou et al., 2004).

Additional respiratory effects were reported to be associated with residential proximity to traffic. Schikowski and co-authors (2005) reported elevated risks of diagnosed chronic obstructive pulmonary disease, frequent cough, and reduced lung function associated

TABLE 2**Traffic Proximity and Childhood Cancer Research—Key Results**

Reference	Location	Study Population (N)	Health Effects Assessed	Exposure Metric	Health Effects with Significant Associations	Distance to Traffic	Traffic Density	Adjusted Odds Ratio	95% CI
Pearson et al., 2000	Denver, CO	≤14-yr-olds with cancer (579)	All cancers, leukemia	Daily distance-weighted traffic density—vehicles/day and distance to residence	All cancers	750 ft	5000–9999 vehicles/day	1.68	1.02–2.80
						750 ft	≥20,000 vehicles/day	5.90	1.69–20.56
					Leukemia	750 ft	5000–9999 vehicles/day	2.04	1.05–3.95
						750 ft	≥20,000 vehicles/day	8.28	2.09–32.80
Reynolds et al., 2002	California	≤15-yr-olds with cancer (6,988)	All cancers, leukemia, gliomas	Vehicles per square mile and miles of road per square mile within block group of residence	All cancers	N/A	6081–8530 vehicles/square mile	1.10*	1.01–1.19
							21.7–24.8 miles/square mile	1.11*	1.02–1.20
					Leukemia	N/A	6081–8530 v/m ²	1.18*	1.03–1.35
Crosignani et al., 2003	Varese Province, Italy	≤14-yr-olds with childhood leukemia (120)	Childhood leukemia	Distance to residence plus Caline model to estimate benzene concentration	Childhood leukemia	300 m	Benzene over 10 µg/m ³ estimated annual average.	3.91*	1.36–11.27
Steffen et al., 2004	Nancy, Lille, Lyon, & Paris, France	≤14-yr-olds with childhood leukemia (567)	Acute leukemia, acute non-lymphocytic leukemia, acute lymphocytic leukemia	Self-reported exposure to heavy-traffic roads & neighboring business	Acute leukemia	N/A	N/A	No statistically significant results	

Notes:

Relative risk (RR) by comparison with low- or no-exposure categories.

N/A = not available (i.e., not reported).

with residential exposure to traffic within 100 m for women 54–55 years of age in Germany. Sugiri and co-authors (2006) reported that residential proximity to a busy road of within 50 m was associated with reduced lung function in a group of German children.

Childhood Cancers

The studies that we examined augmented scientific evidence on potential associations between residential proximity to traffic and childhood cancers including acute non-lymphocytic leukemia and acute lymphocytic leukemia. Three of four studies (Table 2) reported a statistically significant association between childhood cancer and traffic exposure metrics and residential proximity within 750 ft (229 m), 200 m, and 300 m in Denver, Colorado; California; and Varese Province, Italy (Crosignani et al., 2003; Pearson, Wachtel, & Ebi, 2000; Reynolds et al., 2002). Steffen and co-authors (2004) reported an association for self-reported residential proximity to automobile repair stations and petrol stations but not for exposure to heavy traffic.

That study, however, was one of the few relying on the participant's perception of living near heavy traffic rather than more objective traffic and exposure metrics.

Adverse Birth Outcomes

Three studies examined the relationship between adverse birth outcomes and traffic exposure and reported statistically significant associations (Table 3). Maternal residence within 500 m of a major freeway in Taiwan was reported to be a significant risk factor for preterm birth (Yang et al., 2003). Wilhelm and Ritz (2003) reported that California mothers who lived within 750 ft (229 m) of the highest quintile of heavy-traffic roadways during pregnancy were more likely to have a preterm baby. These researchers reported higher risks of preterm and low-birth-weight babies being born in the fall and winter to mothers living nearer the highest-quintile traffic density. An extended analysis of these California births by Ponce and co-authors (2005) further confirmed the association between proximity to

dense traffic and low birth weight for births occurring in the winter.

Mortality Risks

Three studies examined the relationship between residential proximity to traffic and mortality. Associations were reported for cardiopulmonary, stroke, and cardiovascular mortality in the Netherlands, the United Kingdom, and Canada (Finkelstein, 2000; Maheswaran & Elliot, 2003; Hoek, Brunekreef, Goldbohm, Fischer, & van den Brandt, 2002). Both Finkelstein (2000) and Hoek and co-authors (2002) reported statistically significant mortality risks for residences within 100 m of a highway (freeway) and 50 m of an urban road. Maheswaran and Elliot (2003) reported elevated mortality risks at a distance of up to 1000 m from the centroid of the residential enumeration district. These findings were consistent with those of previous studies establishing PM as a well-defined risk factor for premature mortality; more than 50 percent of total PM emissions in urban

TABLE 3**Traffic Proximity and Adverse Birth Outcomes Research—Key Results**

Reference	Location	Study Population (N)	Health Effects Assessed	Exposure Metric	Health Effects with Significant Associations	Distance to Traffic	Traffic Density	Adjusted Odds Ratio	95% CI
Wilhelm & Ritz, 2003	Los Angeles County, CA	1994–1996 live births (50,933)	Low birth weight, preterm births, low birth weight and preterm births	Distance-weighted traffic density (DWTd), and one or more freeways and distance to residence	Preterm births	750 ft	Highest-quintile DWTd	1.08*	1.01–1.15
					Preterm births during fall/winter	750 ft	Highest-quintile DWTd	1.15*	1.05–1.26
					Low birth weight and preterm during fall/winter	750 ft	Highest-quintile DWTd	1.24*	1.03–1.48
					Low birth weight	750 ft	40th to 59th percentile DWTd	1.16*	1.03–1.30
							60th to 79th percentile DWTd	1.15*	1.02–1.29
Low birth weight during fall/winter	750 ft	Highest-quintile DWTd	1.33*	1.11–1.58					
Yang et al., 2003	East Kaohsiung, Taiwan	1992–1997 live births (6,251)	Preterm delivery	Residential distance to a major freeway with average daily traffic count—vehicles/day	Preterm delivery	500 m	93,000 vehicles/day	1.30	1.03–1.65
Ponce et al., 2005	112 ZIP codes in Los Angeles County	1994–1996 live births (37,347)	Low birth Weight	Residential ZIP codes distance intersected by freeways and major arterials, and DWTd	Low birth weight during winter	3.2-km buffer	DWTd 80th percentile	1.30	1.07–1.58

Notes:
Relative risk (RR) by comparison with low- or no-exposure categories.
N/A = not available (i.e., not reported).

areas of industrialized countries have been estimated to come from traffic (Wröbel, Rokita, & Maenhaut, 2000; Briggs et al., 1997).

Discussion and Policy Implications

Of the 29 studies reviewed, 25 reported statistically significant associations between residential proximity to traffic and at least one of the following adverse health effects: increased prevalence and severity of symptoms of asthma and other respiratory diseases; diminished lung function; adverse birth outcomes; childhood cancer; and increased mortality risks. These associations were reported across a broad range of exposure metrics ranging from self-report to sophisticated mobile-source models, a wide variety of analytical designs controlling for diverse confounders, and diverse geographical locations. The results were particularly consistent for 9 of 10 non-respiratory studies reporting statistically significant associations between residential proximity to traffic and childhood cancer; adverse birth outcomes; and cardio-

pulmonary, cardiovascular, cerebrovascular, and stroke mortality.

Mixed findings from studies of respiratory outcomes and residential proximity to traffic may be partly explained by issues related to case identification and definitions used for asthma diagnosis. First, asthma prevalence and respiratory symptoms in many of the studies were self-reported through surveys and were subject to recall bias (Garshick et al., 2003; Gauderman et al., 2005; Heinrich et al., 2005; Janssen et al., 2003; Lewis et al., 2004; McConnell et al., 2006; Schikowski et al., 2005; Venn et al., 2001; Venn et al., 2005). Obtaining accurate reporting of symptoms by young children is especially challenging because they may not be aware of or capable of verbalizing symptoms, or may not be able to recall symptoms as well as older children or adults (Kuehni & Frey, 2002). Second, studies have demonstrated that parental conceptual understanding of wheeze varies across ethnic groups (Cane, Pao, & McKenzie, 2001) and differs from definitions used by epidemiologists (Cane, Ranganathan, & McKenzie, 2000). Third, even a physician's diagnosis

of asthma can be unreliable because of changes in diagnostic practices and definitions over time (Hill, Williams, Tattersfield, & Britton 1989).

Residential distance to and density of traffic were reported to be important factors in the assessment of the relationship between traffic exposure and adverse health outcomes. The majority of studies using varying distances to residences as exposure metrics reported associations with adverse health effects for distances up to 200 m but not for greater distances (Garshick et al., 2003; Gordian et al., 2006; Lin et al., 2002; McConnell et al., 2006; Nicolai et al., 2003; Schikowski et al., 2005; Sugiri et al., 2006; Venn et al., 2001; Wilkinson et al., 1999). Only three studies reported health effects associated with residential proximity greater than 300 m (Ponce et al., 2005; Yang et al., 2003; Maheswaran & Elliot, 2003).

Four of five studies evaluating residential proximity to major highways (freeways) reported statistically significant associations with adverse health effects (Finkelstein et al., 2004; Ponce et al., 2005; Hoek et al., 2002; Yang et al., 2003). Adverse effects were reported

for traffic counts as low as 5,500–9,000 vehicles/day (English et al., 1999), 10,000 vehicles/day (Garshick et al., 2003; Schikowski et al., 2005), and approximately 24,000 vehicles/day (Smargiassi, Berrada, Fortier, & Kosatsky 2006; Wilkinson et al., 1999), as well as for busy-highway (freeway) averages of up to 93,000 vehicles/day (Yang et al., 2003).

Vehicle mix was also suggested as an important factor. Lin and co-authors (2002) reported an association between asthma hospitalizations and residential proximity within 200 m of heavy truck traffic. Ryan and co-authors (2005) reported a statistically significant association between residential proximity within 100 m of stop-and-go bus and truck traffic and parent-reported wheeze in infants less than 12 months old. Janssen and co-authors (2003) assessed health impacts of residential proximity to car and truck traffic and reported elevated risks of conjunctivitis and an itchy rash only for truck traffic.

Several challenges existed to the assessment of relationships between exposure to traffic and health effects. First, personal monitoring that documents duration of exposure and air concentrations of traffic emissions that people actually breathe is typically cost-prohibitive and not practical for most epidemiological studies to date. Therefore, researchers used surrogate exposure metrics to estimate exposure. The precision of exposure metrics used by studies covered in this review ranged from self-report (Heinrich et al., 2005; Steffan et al., 2004; Sugiri et al., 2006) to outputs of sophisticated mobile-source models and geographic information system mapping of residential addresses (Gauderman et al., 2005; Smargiassi et al., 2006). Second, most studies did not account for the relative amount of time people spend in microenvironments other than residences such as work, school, or commuting, where levels of exposure to traffic emissions can vary. Third, estimates of exposure typically do not take into account indoor sources of air pollutants or the variability in residential penetration

of pollutants such as benzene and PM (Sioutas, Delfino, & Singh, 2005). Fourth, physical and chemical properties, composition, and toxicity of fuel mixtures for natural gas, gasoline, and diesel vary in different parts of the world and even within countries (Verma & Tombe, 2002). Finally, constituents, concentrations, and duration of traffic-related residential exposures are affected by many factors, including varied fleet characteristics such as average ages and types of vehicles; designs, grades, and distributions of roads; traffic congestion and driving habits; and different inspection and maintenance programs, as well as variations in national and local regulations (Gwilliam, 2003).

The consistency of reported results across the studies we reviewed provided a “weight-of-evidence” finding suggesting that residential proximity to traffic can be associated with adverse health effects and poses a public health threat. A number of steps can be taken to decrease exposure to traffic-related pollutants and to protect the public. Exposures, especially diesel, can be minimized for children, the elderly, and other vulnerable populations by improved land use and community planning that ensures that schools, daycare centers, and nursing homes are not located within 300 m of a busy road. Also, prohibiting prolonged idling of school buses outside schools and widespread conversion of diesel buses to cleaner alternatives, including buses that use low-sulfur fuels and particulate traps, can reduce exposures of school-aged children (Behrentz et al., 2005; Sabin et al., 2005). Furthermore, federal, state, and local governments can reduce emissions by adopting and enforcing regulations on tailpipe emissions and higher fuel economy standards; promoting use of alternative fuels and low-sulfur diesel; promoting carpooling through use of subsidies and high-occupancy vehicle lanes; implementing smart-growth strategies to reduce urban sprawl; and providing convenient, affordable mass transit options. Employers can reduce traffic-related emissions by providing car- and van-pooling incentives and allowing employ-

ees to telecommute. Individuals can purchase low-polluting vehicles, combine trips, and use alternative means of transportation such as bicycling or walking.

Conclusions

Studies we reviewed consistently reported statistically significant associations between residential proximity to traffic and at least one of the following adverse health effects: increased prevalence and severity of symptoms of asthma and other respiratory diseases; diminished lung function; adverse birth outcomes; childhood cancer; and increased mortality risks. At present, however, epidemiological studies cannot determine causality, and uncertainties exist because of a lack of individual exposure assessments that could rule out confounding by other, unmeasured factors. Also, the studies reviewed did not elucidate which traffic-related pollutant or mixtures of pollutants may have contributed most to the observed adverse health effects. Improved exposure assessments and mechanistic or toxicological studies are needed to identify contributing pollutants and mechanisms of action. Meanwhile, public health can be better protected through enhanced, precautionary land use; smart growth; and transportation policies, as well as through government and private-sector incentive programs and individual actions. 🐾

Disclaimer: This report does not constitute an endorsement by CDC of authors or organizations whose work is reviewed here. The views and opinions of these authors and organizations are not necessarily those of CDC or the U.S. Department of Health and Human Services (HHS).

Corresponding Author: Derek G. Shendell, Assistant Professor, UMDNJ-SPH and EOH-SI, 683 Hoes Lane West, Room 384, P.O. Box 9, Piscataway, NJ 08854. E-mail: shendedg@umdnj.edu.

REFERENCES

- Barker, D.J.P. (1995). The fetal and infant origins of disease. *European Journal of Clinical Investigation*, 25, 457–463.
- Behrentz, E., Sabin, L.D., Winer, A.M., Fitz, D.R., Pankratz, D.V., Colome, S.D., & Fruin, S.A. (2005). Relative importance of school bus-related microenvironments to children's pollutant exposure. *Journal of Air Waste Management Association*, 55(10), 1418–1430.
- Briggs, D.J., Collins, S., Elliott, P., Ficher, P., Kingham, S., Lebet, E., Pyl, K., Reeuwijk, H.V., Smallbone, K., & Van Der Veen, A. (1997). Mapping urban air pollution using GIS: A regression-based approach. *International Journal of Geographic Information Science*, 11, 699–718.

continued on page 40

REFERENCES continued from page 39

- Brunekreef, B., & Holgate, S.T. (2002). Air pollution and health. *Lancet*, 360, 1233-1242.
- Cane, R., Pao, C., & McKenzie, S. (2001). Understanding childhood asthma in focus groups: Perspectives from mothers of different ethnic backgrounds. *Biomedical Central Family Practice*, 2, 4.
- Cane, R.S., Ranganathan, S.C., & McKenzie, S.A. (2000). What do parents of wheezy children understand by "wheeze"? *Archives of Disease in Childhood*, 82, 327-332.
- Crosignani, P., Tittarelli, A., Borgini, A., Codazzi, T., Rovelli, A., Porro, E., Contiero, P., Bianchi, N., Tagliabue, G., Fissi, R., Rossitto, F., & Berrino, F. (2004). Childhood leukemia and road traffic: A population-based case-control study. *International Journal of Cancer*, 108(4), 596-599.
- Dockery, D.W. (2001). Epidemiologic evidence of cardiovascular effects of particulate air pollution. *Environmental Health Perspectives*, 109(Suppl. 4), 483-486.
- English, P., Neutra, R., Scalf, R., Sullivan, M., Waller, L., & Zhu, L., (1999). Examining associations between childhood asthma and traffic flow using a geographic information system. *Environmental Health Perspectives*, 107, 761-767.
- Finkelstein, M. (2000). Leukemia after exposure to benzene: Temporal trends and implications for standards. *American Journal Industrial Medicine*, 38, 1-7.
- Garshick, E., Laden, F., Hart, J.E., & Caron, A. (2003). Residence near a major road and respiratory symptoms in U.S. Veterans. *Epidemiology*, 14(6), 728-736.
- Gauderman, W.J., Avol, E., Lurmann, F., Kuenzli, N., Gilliland, F., Peters, J., & McConnell, R. (2005). Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology*, 16(6), 737-743.
- Gilbert, N.L., Goldberg, M.S., Beckerman, B., Brook, J.R., & Jerrett, M. (2005). Assessing spatial variability of ambient nitrogen dioxide in Montreal, Canada, with a land-use regression model. *Journal of Air and Waste Management Association*, 55(8), 1059-1063.
- Gordian, M.E., Haneuse, S., & Wakefield, J. (2006). An investigation of the association between traffic exposure and the diagnosis of asthma in children. *Journal of Exposure Analysis and Environmental Epidemiology*, 16(1), 49-55.
- Gwilliam, K. (2003). Urban transport in developing countries. *Transport Reviews*, 23(2), 197-216.
- Heinrich, J., Topp, R., Gehring, U., & Thefeld, W. (2005). Traffic at residential address, respiratory health, and atopy in adults: The National German Health Survey 1998. *Environmental Research*, 98(2), 240-249.
- Hill, R., Williams, J., Tattersfield, A., & Britton, J. (1989). Change in use of asthma as a diagnostic label for wheezing illness in schoolchildren. *British Medical Journal*, 299, 898.
- Hoek, G., Brunekreef, B., Goldbohm, S., Fischer, P., & van den Brandt, P.A. (2002). Association between mortality and indicators of traffic-related air pollution in the Netherlands: A cohort study. *Lancet*, 19, 1203-9.
- Janssen, N.A.H., Brunekreef, B., van Vliet, P., Aarts, F., Meliefste, K., Harssema, H., & Fischer, P. (2003). The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. *Environmental Health Perspectives*, 111, 1512-1518.
- Kuehni, C.E., & Frey, U. (2002). Age-related differences in perceived asthma control in childhood: Guidelines and reality. *European Respiratory Journal*, 20, 880-889.
- Lewis, S., Butland, B.K., Strachan, D.P., Bynner, J., Richards, D., Butler, N., & Britton, J. (1996). Study of the aetiology of wheezing illness at age 16 in two national British birth cohorts. *Thorax*, 51, 670-676.
- Lin, S., Munsie, J.P., Hwang, S.A., Fitzgerald, E., & Cayo, M.R. (2002). Childhood asthma hospitalization and residential exposure to state route traffic. *Environmental Research*, 88, 73-81.
- Lloyd, A.C., & Cackette, T.A. (2001). Diesel engines: Environmental impact and control. *Journal Air and Waste Management Association*, 51, 809-847.
- Lwebuga-Mukasa, J.S., Oyana, T., Thenappan, A., & Ayirookuzhi, S.J. (2004). Association between traffic volume and health care use for asthma among residents at a U.S.-Canadian border crossing point. *Journal of Asthma*, 41(3), 289-304.
- Maheswaran, R., & Elliott, P. (2003). Stroke mortality associated with living near main roads in England and Wales: A geographical study. *Stroke*, 34(12), 2776-2780.
- Mauderly, J.L. (1999). Diesel exhaust. In M. Lippmann (Ed.), *Environmental toxicants: Human exposures and their health effects* (2nd ed., pp. 193-241). New York: John Wiley & Sons.
- McConnell, R., Berhane, K., Yao, L., Jerrett, M., Lurmann, F., Gilliland, F., Kunzli, N., Gauderman, J., Avol, E., Thomas, D., & Peters, J. (2006). Traffic, susceptibility, and childhood asthma. *Environmental Health Perspectives*, 114(5), 766-772.
- Nel, A.E., Diaz-Sanchez, D., & Li, N. (2001). The role of particulate pollutants in pulmonary inflammation and asthma: evidence for the involvement of organic chemicals and oxidative stress. *Current Opinion in Pulmonary Medicine*, 7(1), 20-26.
- Nicolai, T., Carr, D., Weiland, S.K., Duhme, H., von Ehrenstein, O., Wagner, C., & von Mutius, E. (2003). Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *European Respiratory Journal*, 21, 956-63.
- Oberdörster, G. (2001). Pulmonary effects of inhaled ultrafine particles. *International Archives of Occupational and Environmental Health*, 74, 1-8.
- Osmond, C., & Baker, D.J.P. (2000). Fetal, infant and childhood growth are predictors of coronary heart disease, diabetes, and hypertension in adult men and women. *Environmental Health Perspectives*, 18, 545-553.
- Paxton, M.B. (1996). Leukemia risk associated with benzene exposure in the Pliofilm cohort. *Environmental Health Perspectives*, 104(Suppl. 6), 1431-1436.
- Pearson, R.L., Wachtel, H., & Ebi, K.L. (2000). Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *Journal of Air and Waste Management Association*, 50(2), 175-180.
- Ponce, N.A., Hoggatt, K.J., Wilhelm, M., & Ritz, B. (2005). Preterm birth: The interaction of traffic-related air pollution with economic hardship in Los Angeles neighborhoods. *American Journal of Epidemiology*, 162, 140-148.
- Pope, C.A., III, Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., & Ito, K., & Thurston, J.D. (2002). Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*, 287, 1132-1141.

REFERENCES

- Reynolds, P., Von Behren, J., Gunier, R.B., Goldberg, D.E., Hertz, A., & Smith, D. (2002). Traffic patterns and childhood cancer incidence rates in California, United States. *Cancer Causes Control*, 13(7), 665-673.
- Rinsky, R.A., Young, R.J., & Smith, A.B. (1981). Leukemia in benzene workers. *American Journal of Industrial Medicine*, 2, 217-245.
- Rinsky, R.A., Smith, A.B., Hornung, R., Filloon, T.G., Young, R.J., Okun, A.H., & Landrigan, P.J. (1987). Benzene and leukemia: An epidemiologic risk assessment. *New England Journal of Medicine*, 315(17), 1044-1050.
- Ritz, B., & Yu, F. (1999). The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environmental Health Perspectives*, 107, 17-25.
- Ritz, B., Yu, F., Chapa, G., & Fruin, S. (2000). Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology*, 11, 502-511.
- Ritz, B., Yu, F., Fruin, S., Chapa, G., Shaw, G.M., & Harris, J.A. (2002). Ambient air pollution and risk of birth defects in Southern California. *American Journal of Epidemiology*, 155, 17-25.
- Ryan, P.H., LeMasters, G., Biagini, J., Bernstein, D., Grinshpun, S.A., Shukla, R., Wilson, K., Villareal, M., Burkle, J., & Lockey, J. (2005). Is it traffic type, volume, or distance? Wheezing in infants living near truck and bus traffic. *Journal of Allergy Clinical Immunology*, 116(2), 279-284.
- Rosenbaum, A.S., Axelrad, D.A., Woodruff, T. J., Wei, Y.H., Ligocki, M.P., & Cohen, J.P. (1999). National estimates of outdoor air toxics concentrations. *Journal of Air and Waste Management Association*, 49, 1138-1152.
- Sabin, L.D., Behrentz, E., Winer, A.M., Jeong, S., Fitz, D.R., Pankratz, D.V., Colome, S.D., & Fruin, S.A. (2005). Characterizing the range of children's air pollutant exposure during school bus commutes. *Journal of Exposure Analysis and Environmental Epidemiology*, 15(5), 377-387.
- Schikowski, T., Sugiri, D., Ranft, U., Gehring, U., Heinrich, J., Wichmann, H.E., & Kramer, U. (2005). Long-term air pollution exposure and living close to busy roads are associated with COPD in women. *Respiratory Research*, 22(6), 152.
- Sioutas, C., Delfino, R.J., & Singh, M. (2005). Exposure assessment for atmospheric ultrafine particles (UFPs) and implications in epidemiological research. *Environmental Health Perspectives*, 113, 947-955.
- Smargiassi, A., Berrada, K., Fortier, I., & Kosatsky, T. (2006). Traffic intensity, dwelling value, and hospital admissions for respiratory disease among the elderly in Montreal (Canada): A case-control analysis. *Journal of Epidemiology and Community Health*, 60(6), 507-512.
- Steffen, C., Auclerc, M.F., Auvrignon, A., Baruchel, A., Kebaili, K., Lambilliotte, A., Leverger, G., Sommelet, D., Vilmer, E., Hemon, D., & Clavel, J. (2004). Acute childhood leukaemia and environmental exposure to potential sources of benzene and other hydrocarbons: A case-control study. *Occupational and Environmental Medicine*, 61(9), 773-778.
- Sugiri, D., Ranft, U., Schikowski, T., & Kramer, U. (2006). The influence of large-scale airborne particle decline and traffic-related exposure on children's lung function. *Environmental Health Perspectives*, 114, 282-288.
- Sydbom, A., Blomberg, A., Parnia, S., Stenfors, N., Sandstrom, T., & Dahlen, S.E. (2001). Health effects of diesel exhaust emissions. *European Respiratory Journal*, 17, 733-746.
- U.S. Environmental Protection Agency, Office of Mobile Sources. (1994). Automobile emissions: An overview (EPA 400-F-92-007). Retrieved December 11, 2007, from <http://www.epa.gov/otaq/consumer/05-autos.pdf>.
- Venn, A.J., Lewis, S.A., Cooper, M., Hubbard, R., & Britton, J. (2001). Living near a main road and the risk of wheezing illness in children. *American Journal of Respiratory and Critical Care Medicine*, 164, 2177-2180.
- Venn, A., Yemaneberhan, H., Lewis, S., Parry, E., & Britton, J. (2005). Proximity of the home to roads and the risk of wheeze in an Ethiopian population. *Occupational and Environmental Medicine*, 62(6), 376-380.
- Verma, D.K., & Tombe, K.D. (2002). Benzene in gasoline and crude oil: Occupational and environmental implications. *American Industrial Hygiene Association Journal*, 63, 225-230.
- Wilhelm, M., & Ritz, B. (2003). Residential proximity to traffic and adverse birth outcomes in Los Angeles County, California, 1994-1996. *Environmental Health Perspectives*, 111, 207-216.
- Wilhelm, M., & Ritz, B. (2005). Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, California, USA. *Environmental Health Perspectives*, 113, 1212-1221.
- Wilkinson, P., Elliott, P., Grundy, C., Shaddick, G., Thakrar, B., Walls, P., & Falconer, S. 1999. Case-control study of hospital admission with asthma in children aged 5-14 years: Relation with road traffic in north west London. *Thorax*, 54, 1070-1074.
- Wróbel, A., Rokita, E., & Maenhaut, W. (2000). Transport of traffic-related aerosols in urban areas. *Science of the Total Environment*, 257, 199-211.
- Yang, C.Y., Chang, C.C., Chuang, H.Y., Ho, C.K., Wu, T.N., & Tsai, S.S. (2003). Evidence for increased risks of preterm delivery in a population residing near a freeway in Taiwan. *Archives of Environmental Health*, 58(10), 649-654.
- Zhu, Y., Hinds, W.C., Kim, S., & Sioutas, C. (2002). Concentration and size distribution of ultrafine particles near a major highway. *Journal of Air and Waste Management Association* 52, 1032-1042.
- Zhu, Y., Hinds, W.C., Kim, S., Shen, S., & Sioutas, C. (2002). Study on ultrafine particles and other vehicular pollutants near a major highway with heavy duty diesel traffic. *Atmospheric Environment*, 36, 4323-4335.
- Zhu, Y., Kuhn, T., Mayo, P., & Hinds, W.C. (2006). Comparison of daytime and nighttime concentration profiles and size distributions of ultrafine particles near a major highway. *Environmental Science Technology*, 40(8), 2531-2536.
- Zmirou, D., Gauvin, S., Pin, I., Momas, I., Sahrroui, F., Just, J., Le Moullec, Y., Bremont, F., Cassadou, S., Reungoat, P., Albertini, M., Lauvergne, N., Chiron, M., & Labbe, A. (2004). Traffic related air pollution and incidence of childhood asthma: Results of the Vesta case-control study. *Journal of Epidemiology and Community Health* 58, 18-23.

Traffic, Air Pollution, and Health

Jonathan M. Samet

Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland, USA

This paper discusses emerging information on exposure to air pollution from traffic and health and raises two key questions with regard to the impact of traffic on public health: 1) is there sufficient evidence to infer that traffic is causing adverse health effects, and; 2) what is the magnitude of the adverse impact of traffic on health? Topics addressed include characterization of exposure to traffic-related pollutants; the findings of epidemiological studies to date; and investigative approaches and the related challenges—including bias, model-based conclusions, and sample size issues—in characterizing the health effects of air pollution from traffic. Also considered are the known health effects of two of the major pollutants produced by vehicle exhaust—particles and ozone. The evidence points to traffic as a threat to public health that will be managed with great difficulty; however, more research is needed to refine our understanding of the health consequences of traffic exposures and as a basis for formulating mitigation policies.

Traffic is an increasingly dominant contributor to air pollution, both in the United States and elsewhere. Its rise in relative importance reflects not only the rising volume of vehicles and miles traveled, but the decline in pollution from manufacturing, as much of the activity in this sector has left the United States and other developed countries for countries in the developing world. The findings of recent studies focused on pollution from traffic suggest that traffic may be a significant contributor to the adverse effects of air pollution on public health.

While the health effects of the major outdoor air pollutants have been well characterized, particularly of the “criteria pollu-

ants” regulated by the U.S. Environmental Protection Agency (EPA), there is only limited evidence and understanding of the extent to which particular sources, including traffic, contribute to the burden of morbidity and mortality from ambient air pollution. In fact, the emphasis on obtaining evidence for individual pollutants in support of setting the National Ambient Air Quality Standards (NAAQS) has limited more holistic assessments of risks of the ambient air pollution mixture. Recently, however, in the case of airborne particles, there is new emphasis on identifying the sources of those particles that may be injurious to human health so as to more effectively target control measures (National Research Council [NRC], 2004). Additionally, the National Research Council report on air quality management called for broader, multipollutant research (NRC, 2004).

This article provides an overview of the emerging information on exposure to air pollution from traffic and health. The intent is to provide a perspective and not to systematically review all of the available information. The article addresses characterization of exposure to traffic-related pollutants; the findings of epidemiological studies to date; and investigative approaches and the related challenges in characterizing the health effects of air pollution from traffic. It also considers, but does not systematically

Received 5 May 2007; accepted 15 June 2007.

Address correspondence to Jonathan M. Samet, MD, MS, Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, 615 N. Wolfe St., Suite W6041, Baltimore, MD 21205, USA. E-mail: jsamet@jhsph.edu

Although the research described in this article has been funded wholly or in part by the United States Environmental Protection Agency through grant/cooperative agreement # RD-83241701 to Dr. Jonathan Samet, it has not been subjected to the Agency’s required peer and policy review and therefore does not necessarily reflect the views of the Agency and no official endorsement should be inferred.

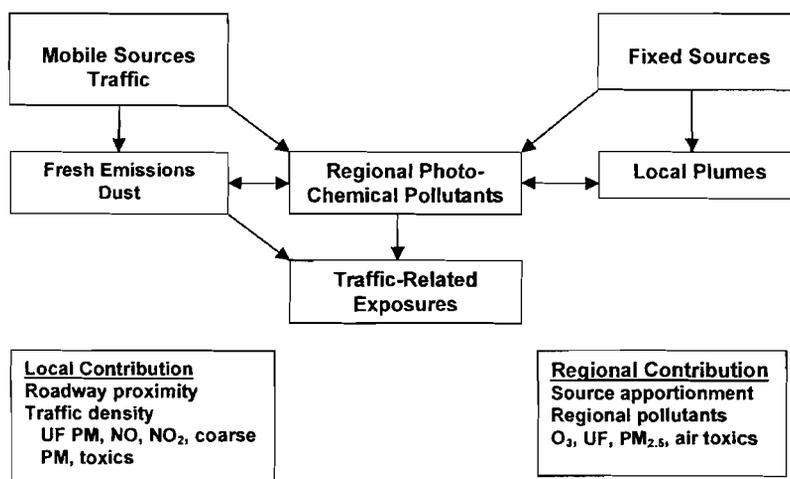


FIG. 1. Local and regional contributions to traffic-related exposures.

cover, the health effects of two of the major pollutants produced by vehicle exhaust—particles and ozone. Comprehensive summaries are available for these pollutants (U.S. EPA, 2004a, 2004b, 2004c, 2006; Pope & Dockery, 2006; World Health Organization [WHO], 2006, 2007).

At present, two main questions need to be answered with regard to traffic and public health: First, is there sufficient evidence to infer that traffic is *causing* adverse health effects? And second, what is the magnitude of the adverse impact of traffic on health? The answer to the first question is relevant to determining whether regulatory or other strategies are needed to protect public health; answer to the second describes the scope of the problem and indicates the potential benefit of interventions directed at exposures to traffic-related air pollutants.

Broadly, the complex mixture of air pollution in urban environments includes most prominently particles, ozone, carbon monoxide, and nitrogen oxides, and the array of pollutants classified as “air toxics.” Traffic is only one of the many sources of these and other air pollutants in urban environments (Figure 1). Airborne particles may originate from local and distant sources; during long-range transport, the particles undergo chemical and physical transformations that may affect the associated risk to health (U.S. EPA, 2003). On smaller geographic scales within cities, particle concentrations may fluctuate widely depending on traffic and weather, as well as the physical configuration of the environment. With regard to particle emissions from traffic, diesel vehicles are a particular concern; in specific areas, diesel vehicles may be a particularly important source of particles because of freeways, shipping terminals or bus depots (Kinney et al., 2000; U.S. EPA, 2004b).

Ozone, another traffic-related pollutant, has a well-characterized pattern of spatial and temporal variation that is relevant to population exposure. Ozone is generated through photochemical reactions involving the action of sunlight on hydrocarbons and nitrogen oxides. Originally identified at the mid-

20th century in southern California, the photochemical pollution for which ozone is the indicator is now widespread throughout the United States (U.S. EPA, 2006). High levels of ozone occur routinely in the summer across the southeastern United States and in major cities of the West. The traffic congestion and weather conditions that generate ozone lead to elevated concentrations on broad geographic scales. Levels rise with the morning traffic rush, peak around mid-day, and typically fall across the afternoon (U.S. EPA, 2006). They are lower in central metropolitan areas, where ozone is chemically quenched by nitrogen oxides in vehicle exhausts.

Many pollutants, other than the two criteria pollutants, ozone and particulate matter, are relevant to the potential for traffic exposure to cause adverse health effects. Many “air toxics” are emitted, such as acrolein, benzene, and butadiene, and their concentrations may be particularly high in “hot spots” in urban locations with heavy traffic congestion.

EXPOSURE TO TRAFFIC-RELATED AIR POLLUTION

Background Considerations

Risks to health from ambient air pollution are determined by personal exposures to these pollutants and ultimately to the doses of pollutants reaching target sites in the respiratory tract and elsewhere. The microenvironmental model is a useful construct for considering how and when exposures to traffic-related pollutants take place for the general population. In this model, personal exposure to air pollution reflects the pollutant concentrations in the microenvironments where time is spent and the time spent in those environments. More specifically, total personal exposure to a pollutant (E) is given by:

$$E = \sum_i c_i t_i$$

where c_i is the concentration of the pollutant of interest in microenvironment i and t_i is the time spent there. Extended to exposure to traffic, total personal exposures for the various traffic-related pollutants are seen to depend on the concentrations of the pollutants not only in outdoor environments on or near roadways, but also on the penetration of the pollutants indoors and the resulting concentrations in indoor environments. Indoor environments are highly relevant to total personal exposure because most time is spent indoors, with the majority at home.

The risk to health depends on the pollutant doses received at the target sites. For the respiratory track, dose depends on the level and pattern of ventilation. Exercise or other forms of exertion increase ventilation and the resulting lung doses of inhaled pollutants. The timing and locations of exertion may also be critical determinants of dose. Additionally, people may alter their activity patterns to avoid situations that are likely to increase the doses of inhaled pollutants.

Studies of air pollution near roadways have provided insights into the spatially and temporally dynamic nature of traffic-related pollution and the difficulty of estimating exposures using the microenvironmental approach. The epidemiological studies on traffic and health have largely focused on the consequences of exposures incurred by living near large roadways, such as freeways in southern California. Studies by researchers in Los Angeles have characterized particles alongside roadways, nicely documenting how particles change in size in relation to distance from the roadway and increasing time since their formation (Kim et al., 2002; Zhu et al., 2006). Immediately adjacent to roadways, the particles in fresh exhaust are clustered in a very small size range with a mode around 10 nm in aerodynamic diameter; with

increasing distance from the roadway, as the particles in fresh exhaust agglomerate, the size increases, moving towards 50 to 100 nm ($0.1 \mu\text{m}$) in aerodynamic diameter at a distance of 50 to 100 m from the freeway. One implication of these findings is that distance from a major roadway may be a surrogate for the characteristics of the particles to which occupants of buildings are exposed.

An additional consideration in extending the microenvironmental model to traffic-related exposures is the penetration of traffic-related pollutants into indoor environments. There is a general literature on penetration of particles and ozone into indoor environments that provides insights that can be extended to traffic-related pollution. In general, ozone levels in indoor environments track with outdoor levels but are substantially lower because of the reactivity of ozone with surfaces. Concentrations indoors depend on the rate of exchange of indoor with outdoor air. Particles in the respirable range, less than 2 to 3 μm in aerodynamic diameter, in outdoor air do penetrate indoors through mechanical systems and natural infiltration. While indoor sources, such as cigarette smoking and cooking, are also significant contributors, penetration of particles in outdoor air into indoor environments is an important contributor to particle concentrations indoors.

My colleagues, Drs. Pat Breyse, Tim Buckley, and Alison Geyh, investigated the temporal pattern of particle concentration indoors in relation to outdoor concentrations and traffic in a row house situated alongside a busy Baltimore City street (Geyh, personal communication, 2007). Figure 2 shows the 1-h average concentrations of indoor and outdoor black carbon concentrations, as well as traffic counts on the street where

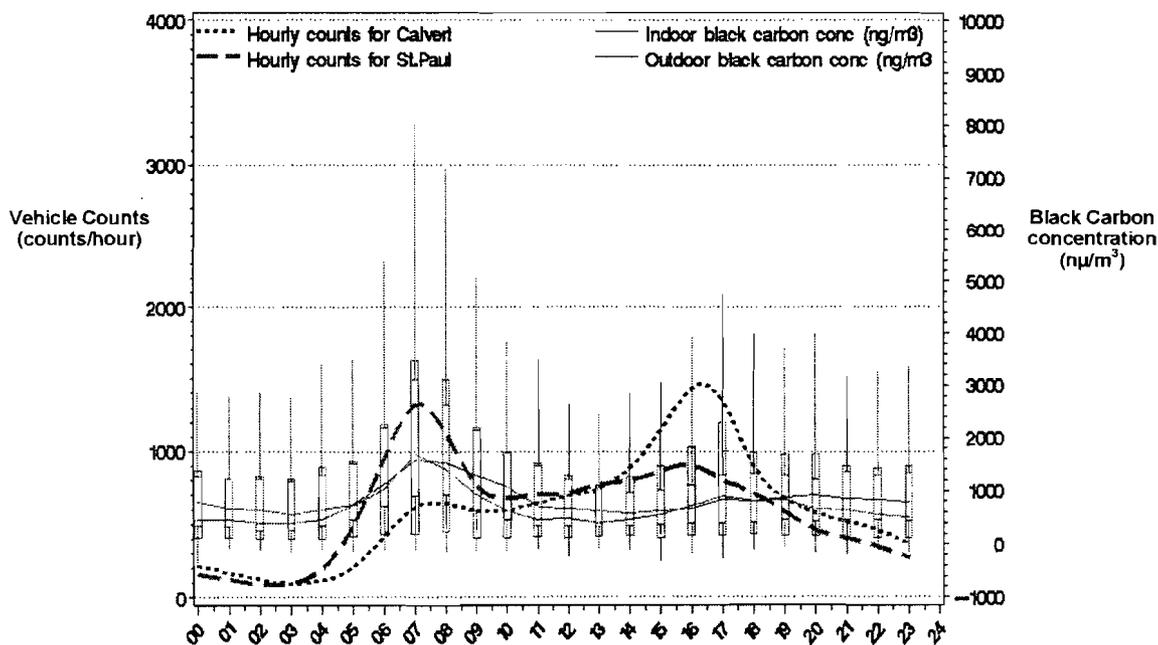


FIG. 2. Weekday 1-h mean BC and traffic count by hour of day (October 1, 2001, to May 31, 2002; unpublished).

the building was located (St. Paul) and a parallel major street (Calvert). The black carbon concentrations outdoors and indoors track with the amount of traffic with morning and afternoon increases; the indoor and outdoor black carbon concentrations are close.

Data on air toxics further illustrate the impact of traffic on air pollution in urban environments. Alongside urban streets, concentrations of air pollution may be affected by local traffic conditions and microscale building geometries and wind conditions. Not surprisingly, monitoring studies show that concentrations of traffic-related pollutants reflect traffic patterns. Levy and colleagues (Levy et al., 2001) mapped concentrations of fine particles and particle-bound polycyclic aromatic hydrocarbons (PAHs) in Roxbury, MA, by providing portable samplers to community residents, who walked the streets with the monitors. Their mapping showed a clear tracking of these pollutants alongside the roadways and higher levels adjacent to a bus depot. Kinney and colleagues (Kinney et al., 2000) monitored 8-h concentrations of PM_{2.5} (particulate matter less than 2.5 μm in aerodynamic diameter) and of elemental carbon (EC), a surrogate for particles from diesel engines, in four sidewalk locations in Harlem. The PM_{2.5} concentrations were close across the sites, reflecting regional-scale contributors to the particles, while EC concentrations varied fourfold and were correlated with truck and bus counts.

These measurement data provide a useful context for application of the microenvironmental model to traffic exposure, particularly for particulate matter. Not surprisingly, they show the influence of regional and local sources on particle concentrations alongside streets. The results of the Baltimore study, as well findings of other studies, confirm that particles outdoors do penetrate indoors (U.S. EPA, 2004c). The work of researchers in Los Angeles documents a strong gradient of increasing particle size with increasing distance from major roadways. The spatial patterns of ozone concentrations, with their relative regional homogeneity and reduction in high traffic areas, would not track with particle concentrations. Spatial patterns of air toxics in urban environments may be dominated by "hot spots" reflecting traffic and point sources.

Classification of Exposure to Traffic

Epidemiologists have used a variety of measures to classify exposure to traffic (Table 1). The indicators range from self-report of proximity to traffic to model-based estimates that may reflect information on proximity to roadways, traffic density, and land use. Exposure has been classified on a dichotomous basis, that is, exposed or not exposed, or on an ordinal basis, providing a relative indication of exposure. To date, little validation of these indices against specific pollutant indicators has been carried out. The first wave of epidemiological studies on traffic largely used measures of proximity to major roadways; subsequently, the complexity of traffic exposure has been better reflected by turning to approaches based on residence location, models, and measurements (Van Atten et al., 2005; Jerrett et al., 2005).

TABLE 1
Methods of classifying exposure to traffic

Source of information	Type of information
Source strength	Emission rate (mass per time), traffic density
Geographical information	Distance of the place of residence from the source or from major roadways
Dispersion models	Spatiotemporal concentration distributions from modeling of emission rates, meteorology, air chemistry, geography
Stationary monitoring and interpolation	Interpolation of monitor data to place of residence
Questionnaires and interviews	Source strength, distance from the source, time-activity
Personal monitoring	Continuous or cumulated concentrations over time
Human samples	Concentration of biomarkers of exposure in biological specimens

The measures used by epidemiologists cannot reflect the full extent of spatial and temporal variation of traffic-related pollution, and most anchor the exposure estimates in residence location. Consequently, exposures in other environments are implicitly assumed to be equivalent when exposures of individuals are classified solely on the basis of residence location. The resulting misclassification may not be random or nondifferential, as persons living in residences associated with higher levels of exposure to traffic-related pollution may also be more likely to have higher transportation and work exposures. The epidemiological measures are certain to be affected by random misclassification as well, which inherently reduces the magnitude of exposure estimates.

The difficulties of classifying exposure to traffic-related pollution have been widely acknowledged and were summarized in a report on a 2003 workshop (Van Atten et al., 2005). A conceptual framework proposed by workshop participants included factors related to emissions, to dispersion and transformation, and to time-activity. The framework's elements have further detail; dispersion and transformation, for example, are affected by the physical configuration of roadways and adjacent buildings, atmospheric conditions, and the chemical composition of the emissions mixture. Epidemiological approaches cannot capture this level of detail.

Jerrett and colleagues (Jerrett et al., 2005) have proposed modeling approaches as useful for estimating air pollution exposures within cities. They offer six modeling approaches: (1) proximity models, which are based in distance from a source; (2) interpolation models, which extend modeling results to residence locations; (3) land use regression models, which use source descriptors and other information to estimate pollutant concentrations at receptor sites; (4) dispersion models,

which combine emissions information with meteorological and topographical data to estimate concentrations; (5) integrated meteorological-emission models, which incorporate representations of meteorological and chemical processes; and (6) hybrid models, which combine multiple approaches, such as personal and regional monitoring. Jerrett and colleagues (2005) provide a comprehensive review of these approaches and consider their application to epidemiological studies. Although modeling approaches are being used increasingly in epidemiological studies, validation of the estimates is lacking, even though needed. Additionally, the full extent of the uncertainty associated with model-based estimates has not been incorporated into analyses of associations with health outcomes.

Validation of models is particularly challenging. Possibilities include comparison to values measured by urban monitors or by monitors sited at residences. Comparison could be made with personal monitoring results. Biomarkers might also be considered. In a study in the United Kingdom, the carbon content of alveolar macrophages of children was positively associated with estimated PM_{10} from traffic (Kulkarni et al., 2006).

EPIDEMIOLOGICAL STUDIES OF TRAFFIC AND HEALTH

Background

There is already extensive epidemiological literature on the principal criteria air pollutants associated with traffic: particulate matter, ozone, nitrogen oxides, and carbon monoxide. The most recent studies show that current levels of particulate matter and ozone remain associated with morbidity and premature mortality (World Health Organization, 2006). The studies do not, however, address whether particulate matter associated with traffic has greater toxicity than particulate matter from other sources. Characterizing sources of particles that may have greater toxicity was identified as a high priority by the National Research

Council's Committee on Research Priorities for Airborne Particulate Matter (NRC, 2004).

An increasing number of epidemiological studies have addressed exposure to traffic and health. In a PubMed search using the terms "traffic" and "health effects," we identified few studies before 1990, and then a steadily increasing number of publications up to the 2006 figure of 103. Of these 103, 37 are concerned with the findings of epidemiological studies. In this article, I do not provide a systematic review of these studies, but rather select examples to describe their methods and the challenges of epidemiological studies on traffic and health. There are also toxicological studies on traffic-related particulate matter generally, as well as on specific types of exhaust. de Kok and colleagues (de Kok et al., 2006) recently reviewed the literature.

Challenges in Interpreting Findings of Studies on Traffic and Health

Interpretation of associations of adverse health outcomes with traffic exposure, as for any target of epidemiologic research, needs to consider bias as an alternative to causation. For indicators of traffic exposure, correlates in a complex set of correlates figures as determinants of exposure and as potential confounders (Figure 3). Some of the same factors that drive residence location, such as income and education, or socioeconomic status (SES), are also determinants of health status. They may also determine propensity for other exposures that are relevant to the health outcomes linked to traffic exposures. As a surrogate for SES, lower income, for example, may be linked to greater indoor air pollution exposure from smoking by the parents of a child and also to the likelihood of living near a busy roadway or a bus depot in an urban area. Housing characteristics, which may be relevant to respiratory health, may also be associated with exposure to traffic-related pollution.

Epidemiological and biostatistical methods may be challenged in attempting to estimate the effect of traffic-related pollutants

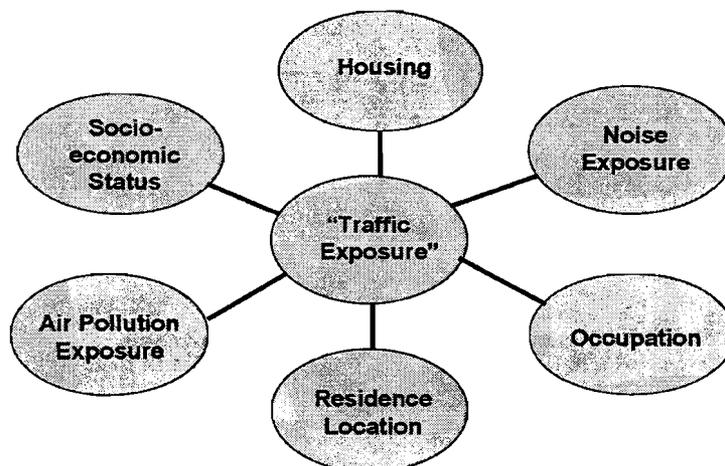


FIG. 3. What does traffic exposure mean?

that are elements of a complex causal web that includes determinants of exposure and potential confounding and modifying factors of the relationship of these pollutants with health outcomes. Model-based conclusions are dependent on the assumptions inherent to the model, for example, linearity of dose response. Additionally, most of the variables considered as potential confounders or modifiers are measured with error; consequently, confounding may not be fully controlled by a model and assessments of effect modification may be biased.

Sample size may further limit interpretation of model results. Generally, traffic-related pollution is associated with "small effects," and limited precision of estimates may cloud interpretation of findings. Generally, as noted, the estimates of the effect of pollution do not acknowledge the uncertainty associated with reliance on models for estimates of exposure; additionally, the precision of effect estimates may be overestimated, if the spatial correlation among estimates for individual participants is not analytically acknowledged.

Findings of Epidemiological Studies on Traffic

The epidemiological literature on the health effects of traffic-related air pollution is growing rapidly. To date, the findings indicate associations of traffic indicators with increased risk for multiple adverse health effects including asthma and allergic diseases, cardiac effects, respiratory symptoms, reduced lung function growth, adverse reproductive outcomes, premature mortality, and lung cancer (White et al., 2005). Additionally, far more extensive evidence links current levels of particulate matter and ozone to increased risk for premature death and a variety of morbidity indicators. Traffic is a major contributor to particulate matter in urban locations and a key source of the precursors of photochemical pollution.

In introducing the topic of air pollution and health, I commented on the two questions to be answered: Does traffic-related air pollution *cause* adverse health effects, and what is the magnitude of the associated risk? If the full sweep of the evidence on particulate matter and ozone is considered, along with the evidence from studies focused on traffic, then the answer to the first question is affirmative (White et al., 2005). The ease with which a causal conclusion can be reached indicates a need to refine the question for public health protection. From the perspective of air quality management, the first question might be sharpened to ask whether control of particulate matter and ozone will reduce the risks from traffic-related pollution; whether the mixture of traffic-related pollutants has toxicity beyond that expected from associated exposures to particulate matter and ozone; and whether effects attributed to traffic reflect the toxicity of pollutants other than particulate matter and ozone.

The second question, the magnitude of the traffic-related burden of disease, needs to be answered to guide strategies for reducing risk of traffic-related pollution. Such estimates are needed to gauge the urgency of taking measures to reduce population exposures to traffic-related pollutants. Methodologies are available to carry out the burden estimation, but we need more accurate

descriptions of exposure-response relationships from epidemiological studies to support such estimates.

CONCLUSIONS

An enlarging body of research evidence indicates that exposure to traffic-related air pollution adversely affects health. The relevant evidence includes monitoring data on the characteristics of near-roadway pollution, the penetration of traffic-generated particles indoors, and the existence of hot spots of pollution in heavily trafficked areas. Epidemiological studies have linked indicators of exposure to traffic to adverse health effects, although the particular pollutants mediating these effects are still not identified. Additionally, difficult methodological issues call for caution in interpreting the epidemiological findings; there is potential for uncontrolled confounding, exposure measures are subject to misclassification, and uncertainty is not fully accounted for.

Nonetheless, the evidence raises concern about a threat to public health that will be managed with great difficulty. Exposures to traffic reflect the amount of traffic and the coupling of emissions from traffic to pollutant concentrations in the environments where people spend time. Control will require both reduced emissions and increased separation of people from emissions. There is a need for further research to refine our understanding of the health consequences of traffic exposures and as a basis for formulating mitigation policies. While we continue to obtain further evidence, prudent, "no-regret" strategies to reduce exposures merit consideration.

REFERENCES

- de Kok, T. M., Drieste, H. A., Hogervorst, J. G., and Briede, J. J. 2006. Toxicological assessment of ambient and traffic-related particulate matter: A review of recent studies. *Mutat. Res.* 613:103–122.
- Jerrett, M., Arain, A., Kanaroglou, P., Beckerman, B., Potoglou, D., Sahuvaroglu, T., Morrison, J., and Giovis, C. 2005. A review and evaluation of intraurban air pollution exposure models. *J. Expos. Anal. Environ. Epidemiol.* 15:185–204.
- Kim, S., Shen, S., and Sioutas, C. 2002. Size distribution and diurnal and seasonal trends of ultrafine particles in source and receptor sites of the Los Angeles basin. *J. Air Waste Manage. Assoc.* 52:297–307.
- Kinney, P. L., Aggarwal, M., Northridge, M. E., Janssen, N. A., and Shepard, P. 2000. Airborne concentrations of PM(2.5) and diesel exhaust particles on Harlem sidewalks: A community-based pilot study. *Environ. Health Perspect.* 108:213–218.
- Kulkarni, N., Pierce, N., Rushton, L., and Grigg, J. 2006. Carbon in airway macrophages and lung function in children. *N. Engl. J. Med.* 355:21–30.
- Levy, J. I., Houseman, E. A., Spengler, J. D., Loh, P., and Ryan, L. 2001. Fine particulate matter and polycyclic aromatic hydrocarbon concentration patterns in Roxbury, Massachusetts: A community-based GIS analysis. *Environ. Health Perspect.* 109:341–347.
- National Research Council (NRC) and Committee on Research Priorities for Airborne Particulate Matter. 2004. *Research priorities*

- for airborne particulate matter: IV. Continuing research progress.* Washington, DC: National Academies Press.
- Pope, C. A. III, and Dockery, D. W. 2006. Health effects of fine particulate air pollution: lines that connect. *J. Air Waste Manage. Assoc.* 56:709–742.
- U.S. Environmental Protection Agency. 2004a. *The ozone report.* U.S. Environmental Protection Agency, Office of Air Quality and Planning Standards, Emissions, Monitoring, and Analysis Division, Research Triangle Park, NC.
- U.S. Environmental Protection Agency 2004b. *The particle pollution report: Current understanding of air quality and emissions through 2003.* U.S. Environmental Protection Agency Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- U.S. Environmental Protection Agency. 2003. *Latest findings on national air quality. 2002 Status and trends.* EPA 454/K-03-001. Research Triangle Park, NC: U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards.
- U.S. Environmental Protection Agency. 2004c. *Air quality criteria for particulate matter.* EPA/600/p-99/022aD and bD. 2004c. Research Triangle Park, NC: U.S. EPA, National Center for Environmental Assessment.
- U.S. Environmental Protection Agency. 2006. *Air quality criteria for ozone and related photochemical oxidants (final).* EPA/600/R-05/004aF-cF. 2-28-2006. Washington, DC: U.S. Environmental Protection Agency.
- Van Atten, C., Brauer, M., Funk, T., Gilbert, N. L., Graham, L., Kaden, D., Miller, P. J., Bracho, L. R., Wheeler, A., and White, R. H. 2005. Assessing population exposures to motor vehicle exhaust. *Rev. Environ. Health* 20:195–214.
- White, R. H., Spengler, J. D., Dilwali, K. M., Barry, B. E., and Samet, J. M. 2005. Report of workshop on traffic, health, and infrastructure planning. *Arch. Environ. Occup. Health* 60:70–76.
- World Health Organization. 2006. *Air quality guidelines for Europe,* 3rd ed. Copenhagen: WHO.
- Zhu, Y., Kuhn, T., Mayo, P., and Hinds, W. C. 2006. Comparison of daytime and nighttime concentration profiles and size distributions of ultrafine particles near a major highway. *Environ. Sci. Technol.* 40:2531–2536.

Report of Workshop on Traffic, Health, and Infrastructure Planning

Ronald H. White, MST; John D. Spengler, PhD; Kumkum M. Dilwali, MS;
Brenda E. Barry, PhD; Jonathan M. Samet, MD, MS

ABSTRACT. Recent air pollutant measurement data document unique aspects of the air pollution mixture near roadways, and an expanding body of epidemiological data suggests increased risks for exacerbation of asthma and other respiratory diseases, premature mortality, and certain cancers and birth outcomes from air pollution exposures in populations residing in relatively close proximity to roadways. The Workshop on Traffic, Health, and Infrastructure Planning, held in February 2004, was convened to provide a forum for interdisciplinary discussion of motor vehicle emissions, exposures and potential health effects related to proximity to motor vehicle traffic. This report summarizes the workshop discussions and findings regarding the current science on this issue, identifies planning and policy issues related to localized motor vehicle emissions and health concerns, and provides recommendations for future research and policy directions.

KEY WORDS: air pollution, exposure assessment, health effects, motor vehicle emissions, traffic proximity

The identification of adverse health effects associated with elevated exposures to motor vehicle emissions (MVE) near busy roadways has emerged as a significant public health concern. This concern is based on recent air-pollutant measurement data documenting the unique aspects of the air-pollution mixture near roadways and an expanding body of epidemiological data suggesting increased risks for exacerbation of asthma and other respiratory diseases, premature mortality, and certain cancers and birth outcomes from air-pollution exposures in populations residing in relatively close proximity to roadways. Measurements indicate that pollutant concentrations can be substantially elevated near major highways and other roadways with large traffic volumes and that this pollutant mixture has specific characteristics reflecting its recent formation and dispersion behavior. Current transportation-planning policies primarily consider the potential impact of increased MVE on population health and on potential exceedances of National Ambient Air Quality Standards (NAAQS) at the metropolitan and regional levels. However, if future studies of health risks

from traffic proximity continue to demonstrate increased health risks for populations located near major roadways and transportation infrastructures, particularly in densely populated urban areas, the implications would be significant, not only for transportation and air-quality planning, but also for urban development.

The Workshop on Traffic, Health, and Infrastructure Planning, held in February 2004, was convened to provide a forum for interdisciplinary discussion of proximity to MVE and related health effects. The 43 workshop participants (a list of workshop participants is available at <http://www.jhsph.edu/RiskSciences/Research>) comprised an interdisciplinary group from the scientific and public-health communities in 4 specific fields that covered mobile-source air-pollution emissions and exposures, their associated health effects, and transportation-infrastructure planning and policy. The objectives of the workshop were to (1) promote dialogue among environmental-health scientists, transportation and urban planners, environmental and transportation advocates and policy-makers, and the motor-vehicle industry regarding the

Ronald H. White and Jonathan M. Samet are with the Department of Epidemiology at Johns Hopkins Bloomberg School of Public Health in Baltimore, MD. John D. Spengler is with the Department of Environmental Health at the School of Public Health at Harvard University in Boston, MA. Kumkum M. Dilwali is with Environmental Health and Engineering, Inc, in Newton, MA. Brenda E. Barry was with Environmental Health and Engineering, Inc, at the time of the workshop and is currently with the Cadmus Group, Inc, in Watertown, MA.

implications of traffic-related health effects for motor-vehicle technology, transportation planning, and urban design; (2) evaluate the scientific evidence regarding the relationship of roadway proximity to MVE exposures and adverse health outcomes and identify research needs; (3) present approaches to assessing pollutant-based health risks from roadways; and (4) consider approaches for integrating planning and design strategies that mitigate potential health risks associated with traffic into transportation-infrastructure design and policy, reflecting the emerging knowledge of exposure and health consequences of MVE.

Although the discussion of policy and planning issues at the workshop focused on the US experience, the scientific information that provided the technical basis for discussion was international in scope, and the findings and recommendations from the workshop have broad implications. The workshop agenda focused on the following 6 key questions developed by the planning committee:

1. What is the current state-of-the-science for on-road mobile-source emissions, localized exposures and health effects?
2. Is this information sufficient to warrant further action to protect public health?
3. What are the current strategies in place to address traffic-exposure and health issues? Have these current strategies been effective?
4. If there is a public-health basis for decreasing exposure to motor-vehicle emissions, then what information is needed to guide strategies to reduce exposures?
5. If further action is needed, is there sufficient information on the impact of various mobile-source types and the scale of exposure to influence decisions on mitigation strategies and policies?
6. Prospectively examine current trends in urbanization and changing motor vehicle and fuel technologies. With regard to these trends (eg, urban sprawl, metropolitan-area in-fill, tighter emission standards, hybrid engines, cleaner fuels), what is the net impact of these trends on exposure?

WORKSHOP FINDINGS AND RECOMMENDATIONS

This document summarizes findings regarding the current science, identifies the planning and policy issues related to localized MVE and health concerns, and provides recommendations for future directions. It also introduces new information and initiatives that have emerged since February 2004. It is not intended to serve as a comprehensive review of the scientific literature on proximity to traffic and health, and the citations listed are for illustrative purposes only. Documents summarizing information on motor-vehicle pollution emissions, near-roadway air-pollution exposures and health effects, and transportation-planning and policy issues are available at <http://www.jhsph.edu/RiskSciences/Research>.

Motor-Vehicle Emissions and Resulting Air Pollution

Motor-vehicle traffic is a mixture of vehicles that vary in age, type, and fuels and that travel under different operating conditions at variable speeds and densities across different roadway configurations and geographical settings. This dynamic source produces a highly variable, complex mixture of air pollutants, and the set of factors determining its characteristics is likely to be important in understanding air pollution from traffic and potential human-health effects.

The mixture of traffic-related air pollution comprises components derived from diverse species, the primary and secondary formations of which are driven by physical and chemical processes. Primary emissions include particulate matter (PM), nitrogen oxides (NO_x), carbon monoxide (CO), and hydrocarbons (HC). Exhaust particles are emitted primarily as submicrometer aerosols (< 1 μm) from operating engines.^{1,2} The concentrations of emitted pollutants decrease as they are transported and dispersed by wind and the turbulence induced by vehicle movement. Particle concentrations undergo shifts in their size distributions through agglomeration, condensation, and evaporation, with increasing domination of larger particles at greater distances from roadways. This dynamic process occurs rapidly within tens of meters of the roadway.³⁻⁵ Ambient temperature and local meteorology can influence this process. In addition, the emitting mix of vehicles (passenger cars and trucks), driving behavior (acceleration, cruise, idle, braking), and speed all determine the local characteristics of near-field pollution.⁶

Traffic produces other potentially hazardous combustion products, including volatile organic compounds, carbonyls, and semivolatile organic compounds (polycyclic aromatic hydrocarbons [PAHs] and nitro-PAHs).⁷ Other traffic-related factors (eg, noise, stress) may also contribute to adverse health responses.

Abrasion of surfaces and friction-induced mechanical disruption resuspends "road dust," itself a complex mixture comprised predominantly of coarse particles (> 2.5 μm in aerodynamic diameter).⁸ Sandy, salty, dirty, and unpaved roadways can result in elevated concentrations of coarse particulate matter.⁹ The coarse fraction might contain earth-crustal material, along with asphalt, metals, latex tire fragments, pollens, and oil-coated particles. The wearing of vehicle parts, including tires and brake pads and discs, generates latex, metal, and ceramic debris.¹⁰ Motor oils evaporate off engine parts or drip onto road surfaces to become part of the complex mixture of contaminants.

Although they typically represent a relatively small percentage of the vehicle fleet, high-emitting vehicles (which often emit visible tailpipe smoke) have been found to contribute a substantial portion of in-use emissions.¹¹ These vehicles are typically poorly maintained and/or have emissions control systems that have been tampered with, and emissions levels in excess of 200 g/mi CO and 20 g/mi HC are possible without properly functioning emission-control systems. Older vehicles also generally contribute disproportionately more pollution

than newer vehicles because of previously less stringent emission standards and equipment deterioration over time.

Air-Pollution Exposures

Motor-vehicle traffic leads to pollutant exposures for vehicle occupants, pedestrians, and occupants of nearby homes, schools, and other buildings. Collective emissions from mobile sources typically give rise to higher concentrations of pollutants, such as CO and NO_x, within urban centers than in surrounding suburban and rural areas.

Several key components of the complex mixture of traffic-derived pollutants, such as CO, NO_x, ultrafine particles, and black carbon, have been found to follow a declining concentration gradient with distance from roadways.^{4,12} In addition, studies have found concentrations of benzene and other air toxics to be significantly higher near busy roads.¹³

Exposures to traffic-related pollutants reflect long-term trends in MVE emissions from changes in the fleet mix and engine design, control technology, and fuel formulation, as well as seasonal, daily, and hourly variations in emissions related to road-surface conditions, congestion, vehicle speed, and weather conditions.^{5,14,15} Current tools for understanding the composition of air pollution near roadways include traffic-demand models, traffic-flow models, source-receptor dispersion models, and transportation-tracking systems. Population exposures to air pollution are further affected by variation in the indoor contribution as it is modified by natural and mechanical ventilation of roadway-adjacent structures and their occupant-activity patterns.¹⁶

The workshop identified substantial limitations in the current understanding of characterization of near-roadway-population exposures to motor-vehicle pollution. In particular, the roles of population characteristics such as other pollutant exposures and social factors, including those related to environmental-justice concerns, are not well studied.

Exposure to elevated levels of pollution along and near roadways presents a special concern and poses challenges both for research and for contaminant control. Exposure-related issues discussed at the workshop included the definition of these areas in the context of near-roadway exposures; the detection of these areas, given the current and likely future limitations of available monitoring networks; and the identification of the extent and causes of these elevated pollutant levels and potential mechanisms to mitigate them.

Near-Roadway Traffic and Health

A rapidly expanding body of epidemiologic studies has documented associations of proximity to roadways or traffic exposure with a wide array of health effects, ranging from diminished quality of life to increased risk of cardiopulmonary mortality. Associations of adverse health outcomes with measures of traffic volume or distance of residential location from roadways have been found in Japan, Europe, Scandinavia, the United States, Canada, and elsewhere. Positive associations have been found for a broad spectrum of adverse health effects

with various surrogate indicators of traffic-related pollution exposure, and these findings are also generally consistent with the broader literature on the health effects of air pollution.

Most frequently reported health effects are associations of increased risk of respiratory symptoms, including exacerbation of asthma, with proximity to or measures of traffic pollution; studies from throughout the world have reported similar, generally positive, findings.¹⁷⁻²⁸ Other studies have identified an increased risk of cardiopulmonary and stroke mortality related to close proximity to traffic.^{29,30} However, studies of other health outcomes, such as asthma hospitalizations and medication use and risk for various cancers, have provided less consistent evidence.³¹⁻⁴⁸ Evidence also suggests that traffic density⁴⁹ and traffic proximity⁵⁰ may be linked to adverse preterm birth outcomes.

The specific components of the mixture contributing to the health effects are uncertain. Although no health researchers to date have quantified subject exposure to the full range of components of the complex traffic-related mixture of pollutants, surrogates, such as NO₂, CO, benzene, and carbon soot (reflectance), have been used for this purpose. Researchers have measured or modeled a few of these pollutants in the community and associated them with adverse health outcomes for children and adults.^{42,43,51} Furthermore, variations in these air-pollutant measures are reasonably predicted by geographic variations in traffic density or distance from high-volume roadways.⁵² A majority of the epidemiologic studies using distance from roadway as a measure of exposure have defined near-roadway proximity as radial distances up to 200 to 300 meters from the road, whereas studies using traffic volume as the exposure measure have found adverse effects associated with traffic volumes as low as 10,000 vehicles per day.²⁰

Health studies suggest near-roadway air pollution health effects are of concern for populations considered generally susceptible to the effects of air pollution (eg, persons with respiratory and cardiovascular disease, children). However, data on the scope of susceptible populations and the magnitude and distribution of the health risks are still lacking.

Policy- and Infrastructure-Planning Issues

Current federally mandated mobile source-control programs are generally insufficient to address and mitigate spatial disparities in traffic-related exposures. The Clean Air Act (CAA) "conformity" provision (Section 176(c)), which requires that planned development of additions to metropolitan transportation systems contribute to the emissions reductions needed for attainment of the NAAQS, requires assessment of the air-quality impact of these projects, primarily at the metropolitan level. However, localized, project-level analyses are required for CO and PM₁₀ nonattainment areas. Localized PM_{2.5} requirements have been proposed as an option for implementation of the PM_{2.5} NAAQS⁵³. The National Environmental Policy Act (NEPA) has played a limited role in reducing emissions by requiring consideration of less-polluting

alternatives in the development of major highways. However, this obligation has been limited to a consideration of alternatives that can be implemented at the scale of the corridor to be served by a particular highway.

Federal and state air-monitoring networks for NAAQS compliance and for hazardous air pollutants range from neighborhood-scale to a mesoscale of tens of kilometers. Near-roadway traffic-related impacts are detected at the microscale of meters, highlighting the challenges associated with identifying potential areas and exposures of concern and implementing mechanisms to monitor air-quality improvements. Given the potential number of locations of concern and the costs of air-quality monitoring, microscale air quality and human-exposure models will likely be used extensively. Additional data will be needed to further develop and evaluate these models. This modeling is also typically reliant upon integrated measures, although continuous data collections may be needed to fully understand the impact of a given source.

High levels of traffic-related air pollution can result from localized traffic congestion as well as from large vehicle volumes. Regulatory policy that crosses the domains of environmental protection and city planning has not been adequately formulated to address this broader problem. Although some "hot spots" of traffic are predictable (eg, toll plazas, truck stops), other situations are sporadic (eg, accidents, construction, poor weather, special events). Even where road conditions on freeways and commercial streets are static, traffic volume varies over time, and this temporal variability provides an additional challenge to transportation-management systems.

As another consideration in policy formulation, patterns of land use change with respect to transportation infrastructure. New transportation infrastructures attract users and often stimulate related development, which can lead to congestion and increased exposure to traffic emissions. This phenomenon is often referred to as "induced demand." The divergent trends of expanding urban sprawl, which increases traffic volumes and results in roadway expansions, and urban revitalization efforts, which encourage urban in-fill to populate urban centers, both have the potential to worsen population exposures to near-roadway motor-vehicle pollution.

Considering a health-assessment paradigm that extends from emissions to exposures to health effects, there are several potential strategies to mitigate health impacts of traffic. These include technical and regulatory control-program approaches, as well as traffic-oriented capital projects. In addition, local zoning and ordinances, procedures, and codes affecting development could mitigate traffic through land-use decisions.

Pollution-Exposure Reduction Options

Reducing population exposures to high levels of traffic-related air pollutants can be accomplished using the following 2 major approaches: (1) reduction of direct vehicle emissions and (2) increasing the separation of populations from emissions.

Reduction of MVE can be accomplished through a variety of approaches, including vehicle-based emission controls, fuel-based emission controls, travel-demand reduction (eg, increased walkability), mode shifting (eg, car to transit or bike), congestion mitigation (eg, traffic-signal coordination), and inspection and maintenance of emission controls on in-use vehicles. Mass emissions can be reduced through any of these measures, although consequences for population exposures may be different.

Reducing emissions at the source is accomplished by limiting emissions from vehicles based on emission-control technologies, modifications to engine design and the combustion characteristics of traditional fuels to reduce pollutant formation, and/or the conversion from gasoline- and diesel-fueled vehicles to vehicles that use less-polluting or nonpolluting energy sources.

Although emissions standards for passenger vehicles will tighten somewhat over the next several years, only very modest gains in emission reductions are expected from the increased Corporate Average Fuel Economy (CAFE) standards and closing of some "light-duty truck" emissions standards loopholes for sport utility vehicles (SUVs). In contrast, widespread use of low-sulfur diesel fuels and particle traps, along with selective catalytic reduction technology, are expected to result in a substantial reduction of new heavy-duty truck emissions. Because existing truck engines are designed to power vehicles for hundreds of thousands of miles, thus extending the time required for fleet turnover, near-term gains for local air quality from these sources will necessarily depend on improved vehicle maintenance and retrofitted controls.

As part of State Implementation Plans (SIPs) containing the air-pollution control measures required to demonstrate attainment of the NAAQS under the CAA, states have adopted strategies to reduce vehicle emissions by means other than reducing direct emissions from vehicles. These strategies include reductions in vehicle miles traveled through the development of public transportation alternatives to single-occupant vehicle travel and reductions of aggregate emissions by decreasing total-vehicle travel. Additional strategies include the implementation of transportation-control and/or land-use strategies that encourage personal travel by multiple-occupant vehicle modes and walking or bicycling rather than single-occupant vehicles; reducing trip lengths by land-use planning that facilitates closer proximity of trip origins and destinations; consolidating freight shipments onto larger platforms (eg, truck to rail or barge), encouraging freight transport by less-emitting modes of shipment; and reducing travel demand.

The public-health consequences of close-proximity exposures to MVE can also be reduced by separating populations from areas with large traffic volumes and/or high levels of traffic congestion where vehicle emissions are highly concentrated. This could be achieved by the isolation of truck and bus depots from residential neighborhoods, the relocation of rail switch yards to unpopulated areas, and the creation of open-space buffer zones along major freeway rights-of-way and near large airports. Exposures could also be reduced by

setbacks, berms, plantings, placement of building air intakes, or installing air-cleaning equipment in mechanical ventilation systems. The comparative quantitative consequences of these strategies to reduce exposures have not been well studied.

Research Needs

A broad range of research is needed to improve our understanding of the health consequences of exposures to MVE near roadways. Refinement of our knowledge and improvements to the analysis of the health, ecological, and economic consequences of congestion and air pollution from traffic will lead to more effective mitigation policies. Key research needs, identified based on the workshop discussions, are as follows:

- More information on the composition and spatial variation of MVE is needed, especially on those components that have received less attention, such as metals, PAHs, and air toxins. Standardized methods to collect and chemically speciate air samples are needed to improve the understanding of the range and variability of potential population exposures.
- Exposure studies to date are limited to 2 types: case studies of specific road configurations and studies to establish relationships among easily measured parameters and traffic- and land-use variables. Case studies of different configurations, effects of buffers, pollution penetration into structures, and personal exposure to traffic-related air pollution are needed.
- Quantification of the variation in traffic-related exposures across age, gender, ethnic, and economic variables that differentiate residential location, commuting patterns, and type of employment in the United States is needed. The application of existing air-quality models to near-roadway issues has been limited because of model uncertainty. Additional data on the above variables, as well as identification of traffic operating characteristics leading to elevated exposures and adverse effects, are needed to further develop and evaluate the utility of these models in understanding exposures to traffic-related air pollution.
- Health evidence relating exposure effects to key cofactors such as noise, socioeconomic status, and related social stressors is needed. Researchers must conduct studies to determine whether the impact of outdoor and indoor allergens is enhanced by coexposure to diesel particles. Additional studies on health outcomes such as cancer, cardiovascular disease, and developmental and immunologic effects are also needed. These should include both real-world as well as scripted studies so the full range of potential scenarios can be examined. Studies of other potentially susceptible populations are needed to address the lack of information on the role of health conditions other than asthma on potential adverse health effects from exposure to fresh automotive emissions.
- Controlled exposure and toxicological studies are needed to begin to evaluate the health impacts of the near-roadway air-pollution mixture (including MVE, road dust,

and emissions related to brakes and tire wear), to identify the pollution components most responsible for health effects, to investigate questions of causality and biological susceptibility, and to identify potential biological mechanisms of adverse health effects from the near-roadway air-pollution mixture.

- Demonstration projects linking traffic-relief strategies to corresponding improvement in health effects or their associated biological markers are needed. Performed as a community collaborative effort, such demonstration projects offer opportunities to educate politicians, planners, developers, and citizens.
- Social experiments are needed to understand factors that would lead to behavior modification. Stemming the ever-increasing vehicle miles traveled will be critical to reducing congestion in the long run. The market forces that are the precursors to traffic are poorly understood. Understanding the financial and other incentives needed to trade passenger miles among modes of transit is critical.

FUTURE DIRECTIONS

Specific and certain quantification of the health consequences of traffic is not possible at the present time, but the emerging evidence from exposure and health studies provides a warning. Living close to heavy traffic has been associated with increases in risk for several adverse health effects. Further research is needed, but the evidence is sufficient to warrant its consideration in many planning decisions and policies. In addition, the relationship between exposure to near-roadway traffic emissions and health impacts, which have primarily been expressed based on residential and, to a more limited extent, school locations, may extend to other locations, such as offices and elder care facilities in close proximity to high-traffic roadways with expected extended-duration exposures. The emerging literature on the relatively high levels of personal exposures to motor-vehicle-related air pollution encountered in-vehicle while traveling, including children's exposure to diesel emissions in school buses, also raises concerns regarding the impact of coexposures on public health.

Despite remaining uncertainties in the specific nature and magnitude of the health problems associated with near-roadway traffic-pollution exposures, workshop participants discussed a range of "no regret" strategies that are directionally correct both in terms of reducing local exposures and lowering the overall impact of mobile source emissions, including greenhouse gases. These strategies, which have national, regional, and local air-quality benefits accruing from reduced emissions and fuel reformulation, included increasing the CAFE standards; retrofitting trucks and buses with additional emission-control technology; promoting cleaner vehicles (including hybrids), electrified truck stops, and other idling-reduction measures. In addition, promotion of telecommuting, densification of housing stock, and expanding public transit, car sharing, and bicycling may lower

emissions and congestion levels. Strategic implementation of these improvements can potentially contribute to relieving or averting local areas of high traffic-related pollution.

In addition to the approaches discussed above that reduce exposures through reductions in vehicle emissions, other strategies that reduce pollution exposures are also available. These include revising building codes, zoning, and traffic-avoiding ordinances to minimize the potential for exposure to traffic-related pollution.

Municipal governments might consider the relationships between traffic and health when planning public investments. Examples of such strategies include siting schools with a buffer setback from heavily traveled roads; equipping diesel school buses with enhanced pollution controls, such as particle traps and positive crankcase ventilation; or converting current fuels to lower-polluting fuels (eg, biodiesel or liquid natural gas). Buildings with mechanical ventilation systems might orient air intakes to minimize vehicle-exhaust intrusion and use higher-efficiency filters and air cleaners.

The multi-disciplinary nature of the issue of air pollution from motor vehicles requires an integrated approach to problem recognition and response from the scientific, engineering, planning, and regulatory communities, which are typically in the domains of separate professional groups. Professionals and citizens alike find it difficult to be broadly informed in sufficient depth across multiple disciplines to comprehensively address traffic-related health concerns. Emerging scientific and engineering knowledge regarding near-roadway traffic air pollution and its impact on public health is typically in the domain of specialized scientific and technical organizations with seemingly little communication between those groups and city planners, architects, developers, or transportation policy makers or regulators. Enhanced communication between all these disciplines would be mutually beneficial to understanding and addressing this issue. It was with this perspective in mind that the workshop included representatives from many of the various disciplines and stakeholders described above.

Enhanced public awareness of the range of health and other societal impacts associated with high levels of motor-vehicle traffic is also needed. With more transparent and comprehensive accounting of these impacts, evaluation of alternative strategies to mitigate unwanted impacts and public support for control measures will improve.

Finally, it is important to note that, since completion of the Traffic, Health and Infrastructure Planning Workshop in February 2004, interest and concern regarding near-roadway-pollution exposures and health effects has continued to grow and is receiving increasing attention from the scientific, regulatory, and public-health communities. The US Environmental Protection Agency (EPA) and the Mickey Leland National Urban Air Toxics Research Center are supporting research on this issue, and the Health Effects Institute has identified this issue as a priority area for research in its 2005–2010 strategic plan.

CONCLUDING COMMENTS

In planning this workshop, there was strong recognition of the topic's immediacy and of the need for convening a multidisciplinary group. The multidisciplinary, multisector approach of this workshop offers a model for future efforts on this subject. In reviewing findings since the workshop, we are impressed by the continued surge of evidence on near-roadway-traffic-related impacts on health. Although the foundation of evidence for decision making still has many gaps, an array of policy options for control needs to be developed now. For some "no regrets" options, immediate implementation may be warranted; for others, specifying alternatives may sharpen planning research.

* * * * *

Funding for this workshop was provided by California Air Resources Board, Engine Manufacturers Association, Harvard University Particulate Matter Research Center, Mickey Leland National Urban Air Toxics Research Center, National Institute of Environmental Health Sciences, Southern California Environmental Health Sciences Center & Children's Environmental Health Center, US Environmental Protection Agency, and US Federal Highway Administration.

The authors of this report wish to acknowledge the work and contributions of all the workshop participants. They particularly acknowledge the efforts of N. Kunzli, F. Gilliland, C. Sioutas, R. Yuhnke, F. Salvucci, H. Nitta, S. Cadle, and M. Brauer for their work in preparing and presenting supporting materials for the workshop. They also thank Rebecca Nachman for her invaluable assistance in planning and executing the workshop logistics, as well as for her editorial assistance with this manuscript.

This report provides the proceedings of the workshop. There was no attempt to achieve group consensus on all issues; consequently, the report should not be necessarily construed as reflecting the views of all participants or their organizational affiliations, nor those of the sponsoring organizations. Although this work was reviewed by EPA and approved for publication, it may not necessarily reflect official agency policy.

Requests for reprints should be sent to Ronald H. White, MST, Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, 615 N Wolfe Street, Room W6035, Baltimore, MD 21205.

E-mail: rwhite@jhsph.edu

* * * * *

References

1. Morawska L, Bofinger ND, Kocis L, Nwankwoala A. Submicrometer and super micrometer particles from diesel vehicle emissions. *Environ Sci Technol.* 1998;32:2033–2042.
2. Ristovski ZD, Morawska L, Bofinger ND, Hitchins J. Submicrometer and supermicrometer particles from spark ignition vehicles. *Environ Sci Technol.* 1998;32:3845–3852.
3. Hitchins J, Morawska L, Wolff R, Gilbert D. Concentrations of submicrometre particles from vehicle emissions near a major road. *Atmos Environ.* 2000;34:51–59.
4. Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc.* 2002;52:1032–1042.
5. Zhang KM, Wexler AS, Zhu YF, Hinds WC, Sioutas C. Evolution of particle number distribution near roadways. Part II: the "road-to-ambient" process. *Atmos Environ.* 2004;38:6655–6665.
6. National Research Council. *Modeling Mobile Source Emissions.* Washington, DC: National Academy Press; 2000.
7. US Environmental Protection Agency. Health assessment document for diesel engine exhaust. Washington, DC: US Environmental Protection Agency; 2002. EPA/600/8-90/057F.
8. Rogge WF, Hildemann LM, Mazurek MA, Cass GR, Simoneit BR. Sources of fine organic aerosol. 3. Road dust, tire debris, and

- organometallic brake lining dust: roads as sources and sinks. *Environ Sci Technol.* 1993;27:1892-1904.
9. Abu-Allaban M, Gillies JA, Gertler AW, Clayton R, Proffitt D. Tailpipe, resuspended road dust, and brake-wear emission factors from on-road vehicles. *Atmos Environ.* 2003;37:5283-5293.
 10. Riediker M, Devlin RB, Griggs TR, et al. Cardiovascular effects in patrol officers are associated with fine particulate matter from brake wear and engine emissions. *Part Fibre Toxicol.* 2004;1:2.
 11. Pokharel SS, Bishop GA, Stedman DH, Slott R. Emissions reductions as a result of automobile improvement. *Environ Sci Technol.* 2003;37:5097-5101.
 12. Zhu Y, Hinds WC, Kim S, Shen S, Sioutas C. Study on ultrafine particles and other vehicular pollutants near a busy highway. *Atmos Environ.* 2002;36:4375-4383.
 13. Sapkota A, Buckley TJ. The mobile source effect on curbside 1,3-butadiene, benzene, and particle-bound polycyclic aromatic hydrocarbons assessed at a tollbooth. *J Air Waste Manag Assoc.* 2003;53:740-748.
 14. Charron A, Harrison RM. Primary particle formation from vehicle emissions during exhaust dilution in the roadside atmosphere. *Atmos Environ.* 2003;29:4109-4119.
 15. Zhu Y, Hinds WC, Ki S, Shen S, Sioutas C. Seasonal trends of concentration and size distributions of ultrafine particles near major freeways in Los Angeles. *Aerosol Sci Technol.* 2004;38:5-13.
 16. Ozkaynak H, Xue J, Weker R, et al. The particle TEAM (PTEAM) study: analysis of the data. Final report. Vol 3. Washington, DC: US Environmental Protection Agency; 1996. EPA/600/R-95/098.
 17. Brauer M, Hoek G, Van Vliet P, et al. Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Respir Crit Care Med.* 2002;166:1092-1098.
 18. Braun-Fahrlander C, Ackermann-Lieblich U, Schwartz J, Gnehm HP, Rutishauser M, Wanner HU. Air pollution and respiratory symptoms in preschool children. *Am Rev Respir Dis.* 1992;145:42-47.
 19. Ciccone G, Forastiere F, Agabiti N, et al. Road traffic and adverse respiratory effects in children. SIDRIA collaborative group. *Occup Environ Med.* 1998;55:771-778.
 20. Garshick E, Laden F, Hart JE, Caron A. Residence near a major road and respiratory symptoms in U.S. veterans. *Epidemiology.* 2003;14:728-736.
 21. Gehring U, Cyrys J, Sedlmeier G, et al. Traffic-related air pollution and respiratory health during the first 2 yrs of life. *Eur Respir J.* 2002;19:690-698.
 22. Kramer U, Koch T, Ranft U, Ring J, Behrendt H. Traffic-related air pollution is associated with atopy in children living in urban areas. *Epidemiology.* 2000;11:64-70.
 23. Murakami M, Ono M, Tamura K. Health problems of residents along heavy-traffic roads. *J Hum Ergol (Tokyo).* 1990;19:101-106.
 24. Nicolai T, Carr D, Weiland SK, et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *Eur Respir J.* 2003;21:956-963.
 25. Nitta H, Sato T, Nakai S, et al. Respiratory health associated with exposure to automobile exhaust. Results of cross-sectional studies in 1979, 1982, and 1983. *Arch Environ Health.* 1993;48:53-58.
 26. Oosterlee A, Drijver M, Lebret E, Brunekreef B. Chronic respiratory symptoms in children and adults living along streets with high traffic density. *Occup Environ Med.* 1996;53:241-247.
 27. van Vliet P, Knape M, de Hartog J, et al. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res.* 1997;74:122-132.
 28. Venn A, Lewis S, Cooper M, et al. Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study. *Occup Environ Med.* 2000;57:152-158.
 29. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *Lancet.* 2002;360:1203-1209.
 30. Maheswaran R, Elliott P. Stroke mortality associated with living near main roads in England and Wales: a geographical study. *Stroke.* 2003;34:2776-2780.
 31. Buckeridge DL, Glazier R, Harvey BJ, et al. Effect of motor vehicle emissions on respiratory health in an urban area. *Environ Health Perspect.* 2002;110:293-300.
 32. Duhme H, Weiland SK, Keil U, et al. The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. *Epidemiology.* 1996;7:578-582.
 33. Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. *Arch Environ Health.* 1994;49:223-227.
 34. English P, Neutra R, Scalf R, et al. Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ Health Perspect.* 1999;107:761-767.
 35. Feychting M, Svensson D, Ahlbom A. Exposure to motor vehicle exhaust and childhood cancer. *Scand J Work Environ Health.* 1998;24:8-11.
 36. Gordian ME, Haneuse S, Wakefield J. An investigation of the association between traffic exposure and the diagnosis of asthma in children. *J Expo Sci Environ Epidemiol.* 2006;16:49-55.
 37. Harrison RM, Leung PL, Somerville L, Smith R, Gilman E. Analysis of incidence of childhood cancer in the West Midlands of the United Kingdom in relation to proximity to main roads and petrol stations. *Occup Environ Med.* 1999;56:774-780.
 38. Lin S, Munsie JP, Hwang SA, Fitzgerald E, Cayo MR. Childhood asthma hospitalization and residential exposure to state route traffic. *Environ Res.* 2002;88:73-81.
 39. Livingstone AE, Shaddick G, Grundy C, Elliott P. Do people living near inner city main roads have more asthma needing treatment? Case control study. *BMJ.* 1996;312:676-677.
 40. McConnell R, Berhane K, Lurmann F, et al. Traffic and asthma prevalence in children. *Am J Respir Crit Care Med.* 2002;165:A492.
 41. Morris SE, Sale RC, Wakefield JC, et al. Hospital admissions for asthma and chronic obstructive airways disease in East London hospitals and proximity of residence to main roads. *J Epidemiol Community Health.* 2000;54:75-76.
 42. Nafstad P, Haheim LL, Oftedal B, et al. Lung cancer and air pollution: A 27-year follow up of 16,209 Norwegian men. *Thorax.* 2003;58:1071-1076.
 43. Nyberg F, Gustavsson P, Jarup L, et al. Urban air pollution and lung cancer in Stockholm. *Epidemiology.* 2000;11:487-495.
 44. Pearson RL, Wachtel H, Ebi KL. Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *J Air Waste Manag Assoc.* 2000;50:175-180.
 45. Reynolds P, Von Behren J, Gunier RB, Goldberg DE, Hertz A. Residential exposure to traffic in California and childhood cancer. *Epidemiology.* 2004;15:6-12.
 46. Shima M, Nitta Y, Adachi M. Traffic-related air pollution and respiratory symptoms in children living along trunk roads in Chiba prefecture, Japan. *J Epidemiol.* 2003;13:108-119.
 47. Wilkinson P, Elliott P, Grundy C, et al. Case-control study of hospital admission with asthma in children aged 5-14 years: relation with road traffic in North West London. *Thorax.* 1999;54:1070-1074.
 48. Zmirou D, Gauvin S, Pin I, et al. Traffic related air pollution and incidence of childhood asthma: results of the Vesta case-control study. *J Epidemiol Community Health.* 2004;58:18-23.
 49. Wilhelm M, Ritz B. Residential proximity to traffic and adverse birth outcomes in Los Angeles County, California, 1994-1996. *Environ Health Perspect.* 2003;111:207-216.
 50. Yang CY, Chang CC, Chuang HY, et al. Evidence for increased risks of preterm delivery in a population residing near a freeway in Taiwan. *Arch Environ Health.* 2003;58:649-654.
 51. Raaschou-Nielsen O, Hertel O, Thomsen BL, Olsen JH. Air pollution from traffic at the residence of children with cancer. *Am J Epidemiol.* 2001;153:433-443.
 52. Brauer M, Hoek G, van Vliet P, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. *Epidemiology.* 2003;14:228-239.
 53. U.S. Environmental Protection Agency. Options for PM2.5 and PM10 hot-spot analyses in the transportation conformity rule amendments for the new PM2.5 and existing PM10 national ambient air quality standards. Supplemental notice of proposed rule. 69 Fed. Reg. 72140-72156 (December 13, 2004).

Review

Open Access

Near-highway pollutants in motor vehicle exhaust: A review of epidemiologic evidence of cardiac and pulmonary health risks

Doug Brugge*¹, John L Durant² and Christine Rioux³

Address: ¹Tufts Community Research Center, Tufts University School of Medicine, Boston, MA, USA, ²Department of Civil and Environmental Engineering, Tufts University, Medford, MA 02155, USA and ³Interdisciplinary PhD Program, Tufts University, Medford, MA 02155, USA

Email: Doug Brugge* - dbrugge@aol.com; John L Durant - john.durant@tufts.edu; Christine Rioux - Christine.rioux@tufts.edu

* Corresponding author

Published: 9 August 2007

Received: 2 January 2007

Environmental Health 2007, **6**:23 doi:10.1186/1476-069X-6-23

Accepted: 9 August 2007

This article is available from: <http://www.ehjournal.net/content/6/1/23>

© 2007 Brugge et al; licensee BioMed Central Ltd.

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/2.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

There is growing evidence of a distinct set of freshly-emitted air pollutants downwind from major highways, motorways, and freeways that include elevated levels of ultrafine particulates (UFP), black carbon (BC), oxides of nitrogen (NO_x), and carbon monoxide (CO). People living or otherwise spending substantial time within about 200 m of highways are exposed to these pollutants more so than persons living at a greater distance, even compared to living on busy urban streets. Evidence of the health hazards of these pollutants arises from studies that assess proximity to highways, actual exposure to the pollutants, or both. Taken as a whole, the health studies show elevated risk for development of asthma and reduced lung function in children who live near major highways. Studies of particulate matter (PM) that show associations with cardiac and pulmonary mortality also appear to indicate increasing risk as smaller geographic areas are studied, suggesting localized sources that likely include major highways. Although less work has tested the association between lung cancer and highways, the existing studies suggest an association as well. While the evidence is substantial for a link between near-highway exposures and adverse health outcomes, considerable work remains to understand the exact nature and magnitude of the risks.

Background

Approximately 11% of US households are located within 100 meters of 4-lane highways [estimated using: [1,2]]. While it is clear that automobiles are significant sources of air pollution, the exposure of near-highway residents to pollutants in automobile exhaust has only recently begun to be characterized. There are two main reasons for this: (A) federal and state air monitoring programs are typically set up to measure pollutants at the regional, not local scale; and (B) regional monitoring stations typically do not measure all of the types of pollutants that are elevated next to highways. It is, therefore, critical to ask what is known about near-highway exposures and their possible health consequences.

Here we review studies describing measurement of near-highway air pollutants, and epidemiologic studies of cardiac and pulmonary outcomes as they relate to exposure to these pollutants and/or proximity to highways. Although some studies suggest that other health impacts are also important (e.g., birth outcomes), we feel that the case for these health effects are less well developed scientifically and do not have the same potential to drive public policy at this time. We did not seek to fully integrate the relevant cellular biology and toxicological literature, except for a few key references, because they are so vast by themselves.

We started with studies that we knew well and also searched the engineering and health literature on Medline. We were able to find some earlier epidemiologic studies based on citations in more recent articles. We include some studies that assessed motor vehicle-related pollutants at central site monitors (i.e., that did not measure highway proximity or traffic) because we feel that they add to the plausibility of the associations seen in other studies. The relative emphasis given to studies was based on our appraisal of the rigor of their methodology and the significance of their findings. We conclude with a summary and with recommendations for policy and further research.

Motor vehicle pollution

It is well known that motor vehicle exhaust is a significant source of air pollution. The most widely reported pollutants in vehicular exhaust include carbon monoxide, nitrogen and sulfur oxides, unburned hydrocarbons (from fuel and crankcase oil), particulate matter, polycyclic aromatic hydrocarbons, and other organic compounds that derive from combustion [3-5]. While much attention has focused on the transport and transformation of these pollutants in ambient air – particularly in areas where both ambient pollutant concentrations and human exposures are elevated (e.g., congested city centers, tunnels, and urban canyons created by tall buildings), less attention has been given to measuring pollutants and exposures near heavily-trafficked highways. Several lines of evidence now suggest that steep gradients of certain pollutants exist next to heavily traveled highways and that living within these elevated pollution zones can have detrimental effects on human health.

It should be noted that many different types of highways have been studied, ranging from California "freeways" (defined as multi-lane, high-speed roadways with restricted access) to four-lane (two in each direction), variable-speed roadways with unrestricted access. There is considerable variation in the literature in defining highways and we choose to include studies in our review that used a broad range of definitions (see Table 1).

It should also be noted that there may be significant heterogeneity in the types and amounts of vehicles using highways. The typical vehicle fleet in the US is composed of passenger cars, sports utility vehicles, motorcycles, pickup trucks, vans, buses, and small, medium, and large trucks. The composition and size of a fleet on a given highway may vary depending on the time of day, day of the week, and use restrictions for certain classes of vehicles. Fleets may also vary in the average age and state of repair of vehicles, the fractions of vehicles that burn diesel and gasoline, and the fraction of vehicles that have catalytic converters. These factors will influence the kinds and

amounts of pollutants in tailpipe emissions. Similarly, driving conditions, fuel chemistry, and meteorology can also significantly impact emissions rates as well as the kinds and concentrations of pollutants present in the near-highway environment. These factors have rarely been taken into consideration in health outcome studies of near-highway exposure.

Based on our review of the literature, the pollutants that have most consistently been reported at elevated levels near highways include ultrafine particles (UFP), black carbon (BC), nitrogen oxides (NO_x), and carbon monoxide (CO). In addition, PM_{2.5} and PM₁₀ were measured in many of the epidemiologic studies we reviewed. UFP are defined as particles having an aerodynamic diameter in the range of 0.005 to 0.1 microns (μm). UFP form by condensation of hot vapors in tailpipe emissions, and can grow in size by coagulation. PM_{2.5} and PM₁₀ refer to particulate matter with aerodynamic diameters of 2.5 and 10 μm, respectively. BC (or "soot carbon") is an impure form of elemental carbon that has a graphite-like structure. It is the major light-absorbing component of combustion aerosols. These various constituents can be measured in real time or near-real time using particle counters (UFP) and analyzers that measure light absorption (BC and CO), chemiluminescence (NO_x), and weight (PM_{2.5} and PM₁₀). Because UFP, NO_x, BC, and CO derive from a common source – vehicular emissions – they are typically highly inter-correlated.

Air pollutant gradients near highways

Several recent studies have shown that sharp pollutant gradients exist near highways. Shi et al. [6] measured UFP number concentration and size distribution along a roadway-to-urban-background transect in Birmingham (UK), and found that particle number concentrations decreased nearly 5-fold within 30 m of a major roadway (>30,000 veh/d). Similar observations were made by Zhu et al. [7,8] in Los Angeles. Zhu et al. measured wind speed and direction, traffic volume, UFP number concentration and size distribution as well as BC and CO along transects downwind of a highway that is dominated by gasoline vehicles (Freeway 405; 13,900 vehicles per hour; veh/h) and a highway that carries a high percentage of diesel vehicles (Freeway 710; 12,180 veh/h). Relative concentrations of CO, BC, and total particle number concentration decreased exponentially between 17 and 150 m downwind from the highways, while at 300 m UFP number concentrations were the same as at upwind sites. An increase in the relative concentrations of larger particles and concomitant decrease in smaller particles was also observed along the transects (see Figure 1). Similar observations were made by Zhang et al. [9] who demonstrated "road-to-ambient" evolution of particle number distributions near highways 405 and 710 in both winter and sum-

Table 1: Summary of near-highway pollution gradients

Citation	Location	Highway traffic intensity ^a	Pollutants measured ^b	Observed Pollution Gradients
Shi et al. 1999 (6)	Birmingham, UK	30,000 veh/d	UFP + FP (10-10 ⁴ nm)	2-100 m ^c
Zhu et al. 2002 (8)	Los Angeles; Freeway 710	12,180 veh/h	UFP, CO, BC	17-300 m ^c
Zhu et al. 2002 (7)	Los Angeles; Freeway 405	13,900 veh/h	UFP, CO, BC	30-300 m ^c
Hitchins et al. 2002 (11)	Brisbane (Austr.)	2,130-3,400 veh/h	UFP + FP (15-2 × 10 ⁴ nm), PM _{2.5}	15-375 m ^c
Fischer et al. 2000 (13)	Amsterdam	<3,000-30,974 veh/d	PM _{2.5} , PM ₁₀ , PPAH, VOCs	NA
Roorda-Knape et al. 1998 (14)	Netherlands	80,000-152,000 veh/d	PM _{2.5} , PM ₁₀ , BC, VOCs, NO ₂	15-330 m ^c
Janssen et al. 2001 (15)	Netherlands	40,000-170,000 veh/d	PM _{2.5} , VOCs, NO ₂	< 400 m ^c
Morawska et al. 1999 (12)	Brisbane (Austr.)	NA	UFP	10-210 m ^c

^aAs defined in article cited (veh/d = vehicles per day; veh/h = vehicles per hour).

^bUFP = ultrafine particles; FP = fine particles; PM_{2.5} = particles with aerodynamic diameter ≤ 2.5 μm; PM₁₀ = particles with aerodynamic diameter ≤ 10 μm; BC = black carbon; PPAH = particle-bound polycyclic aromatic hydrocarbons; VOCs = volatile organic compounds

^cPollutant measurements were made along a transect away from the highway

NA = not applicable; measurements were not made.

mer. Zhang et al. observed that between 30-90 m downwind of the highways, particles grew larger than 0.01 μm due to condensation, while at distances >90 m, there was both continued particle growth (to >0.1 μm) as well as particle shrinkage to <0.01 μm due to evaporation. Because condensation, evaporation, and dilution alter size distribution and particle composition, freshly-emitted UFP near highways may differ in chemical composition from UFP that has undergone atmospheric transformation during transport to downwind locations [10].

Two studies in Brisbane (Australia) highlight the importance of wind speed and direction as well as contributions of pollutants from nearby roadways in tracking highway-generated pollutant gradients. Hitchins et al. [11] measured the mass concentrations of 0.1-10 μm particles as well as total particle number concentration and size distribution for 0.015-0.7 μm particles near highways (2,130-3,400 veh/h). Hitchins et al. observed that the distance from highways at which number and mass concentrations decreased by 50% varied from 100 to 375 m depending on the wind speed and direction. Morawska et al. [12] measured the changes in UFP number concentrations along horizontal and vertical transects near highways to distinguish highway and normal street traffic contributions. It was observed that UFP number concentrations were highest <15 m from highways, while 15-200 m from highways there was no significant difference in UFP number concentrations along either horizontal or vertical transects - presumably due to mixing of highway pollutants with emissions from traffic on nearby, local roadways.

In addition to UFP, other pollutants - such as PM_{2.5}, PM₁₀, NO₂ (nitrogen dioxide), VOCs (volatile organic

compounds), and particle-bound polycyclic aromatic hydrocarbons (PPAH) - have been studied in relation to heavily-trafficked roadways. Fischer et al. [13] measured PM_{2.5}, PM₁₀, PPAH, and VOC concentrations outside and inside homes on streets with high and low traffic volumes in Amsterdam (<3,000-30,974 veh/d). In this study, PPAH and VOCs were measured using methods based on gas chromatography. Fischer et al. found that while PM_{2.5} and PM₁₀ mass concentrations were not specific indicators of traffic-related air pollution, PPAH and VOC levels were ~2-fold higher both indoor and outdoor in high traffic areas compared to low traffic areas. Roorda-Knape et al [14] measured PM_{2.5}, PM₁₀, black smoke (which is similar to BC), NO₂, and benzene in residential areas <300 m from highways (80,000-152,000 veh/d) in the Netherlands. Black smoke was measured by a reflectance-based method using filtered particles; benzene was measured using a method based on gas chromatography. Roorda-Knape et al reported that outdoor concentrations of black smoke and NO₂ decreased with distance from highways, while PM_{2.5}, PM₁₀, and benzene concentrations did not change with distance. In addition, Roorda-Knape et al. found that indoor black smoke concentrations were correlated with truck traffic, and NO₂ was correlated with both traffic volume and distance from highways. Janssen et al. [15] studied PM_{2.5}, PM₁₀, benzene, and black smoke in 24 schools in the Netherlands and found that PM_{2.5} and black smoke increased with truck traffic and decreased with distance from highways (40,000-170,000 veh/d).

In summary, the literature shows that UFP, BC, CO and NO_x are elevated near highways (>30,000 veh/d), and that other pollutants including VOCs and PPAHs may also be elevated. Thus, people living within about 30 m of highways are likely to receive much higher exposure to

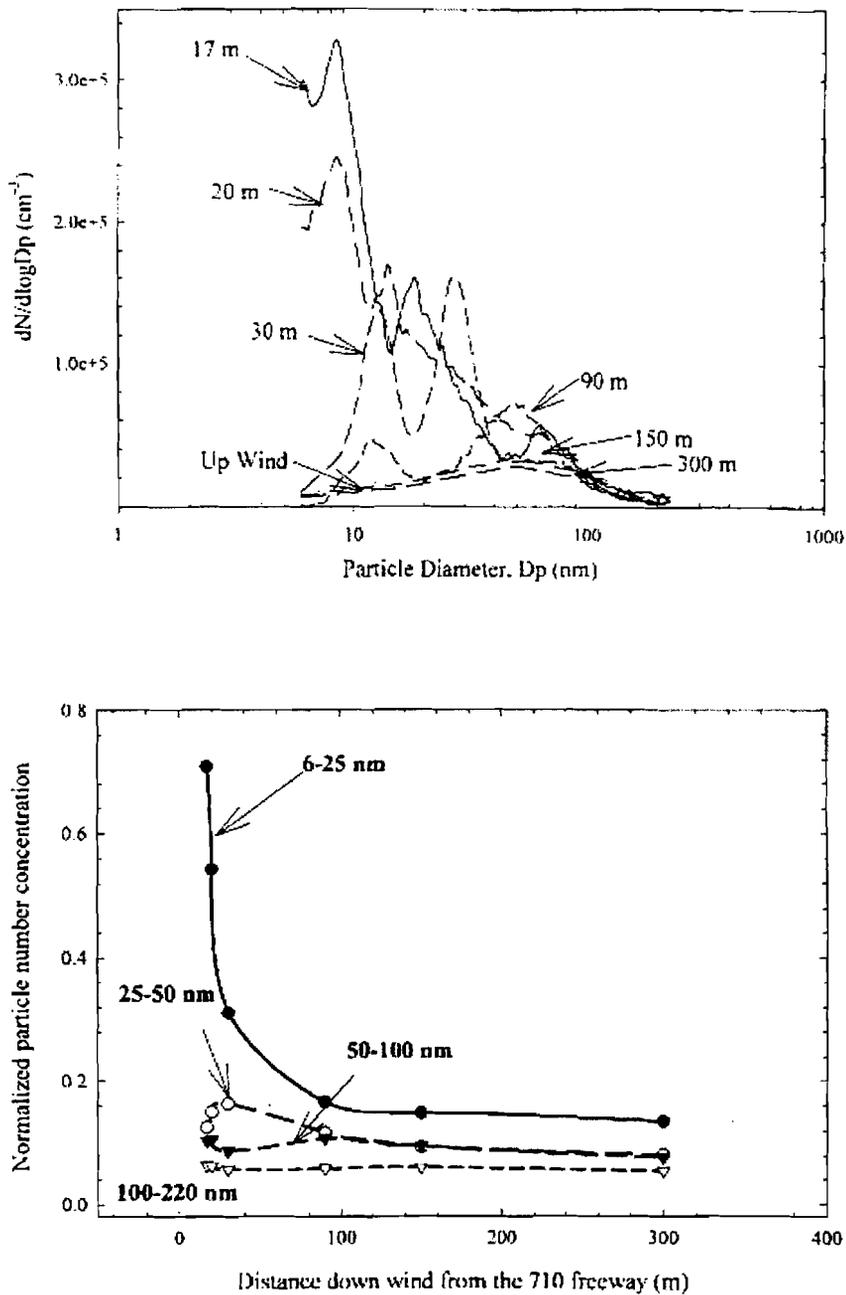


Figure 1 Ultrafine particle size distribution (top panel) and normalized particle number concentration for different size ranges (bottom panel) as a function of distance from a highway in Los Angeles. From Zhu et al. (8). Reprinted with permission from Elsevier.

traffic-related air pollutants compared to residents living >200 m (+/- 50 m) from highways.

Cardiovascular health and traffic-related pollution

Results from clinical, epidemiological, and animal studies are converging to indicate that short-term and long-term exposures to traffic-related pollution, especially particulates, have adverse cardiovascular effects [16-18]. Most of these studies have focused on, and/or demonstrated the strongest associations between cardiovascular health outcomes and particulates by weight or number concentrations [19-21] though CO, SO₂, NO₂, and BC have also been examined. BC has been shown to be associated with decreases in heart rate variability (HRV) [22,23] and black smoke and NO₂ shown to be associated with cardiopulmonary mortality [24].

Short-term exposure to fine particulate pollution exacerbates existing pulmonary and cardiovascular disease and long-term repeated exposures increases the risk of cardiovascular disease and death [25,26].

Though not focused on near-highway pollution, two large prospective cohort studies, the Six-Cities Study [27] and the American Cancer Society (ACS) Study [28] provided the groundwork for later research on fine particulates and cardiovascular disease. Both of these studies found associations between increased levels of exposure to ambient PM and sulfate air pollution recorded at central city monitors and annual average mortality from cardiopulmonary disease, which at the time combined cardiovascular and pulmonary disease other than lung cancer. The Six-Cities Study examined PM_{2.5} and PM_{10/15}. The ACS study examined PM_{2.5}. Relative risk ratios of mortality from cardiopulmonary disease comparing locations with the highest and lowest fine particle concentrations (which had differences of 24.5 and 18.6 ug/m³ respectively) were 1.37 (1.11, 1.68) and 1.31 (1.17, 1.46) in the Six Cities and ACS studies, respectively. These analyses controlled for many confounders, including smoking and gas stoves but not other housing conditions or time spent at home. The studies were subject to intensive replication, validation, and reanalysis that confirmed the original findings. PM_{2.5} generally declined following implementation of new US Environmental Protection Agency standards in 1997 [17,29], yet since that time studies have shown elevated health risks due to long-term exposures to the 1997 PM threshold concentrations [29,30].

Much of the epidemiological research has focused on assessing the early physiological responses to short-term fluctuations in air pollution in order to understand how these exposures may alter cardiovascular risk profiles and exacerbate cardiovascular disease [31]. Heart rate variability, a risk factor for future cardiovascular outcomes, is

altered by traffic-related pollutants particularly in older people and people with heart disease [22,23,32]. With decreased heart rate variability as the adverse outcome, negative associations between HRV and particulates were strongest for the smallest size fraction studied [33] (PM_{0.3-1.0}); [34] (PM_{0.02-1}). In two studies that included other pollutants, black carbon, an indicator of traffic particles, also elicited a strong association with both time and frequency domain HRV variables; associations were also strong for PM_{2.5} for both time and frequency HRV variables in the Adar et al study [23]; this and subsequent near highway studies are summarized in Table 2], however, PM_{2.5} was not associated with frequency domain variables in the Schwartz et al. study [22].

Several studies show that exposure to PM varies spatially within a city [35-37], and finer spatial analyses show higher risks to individuals living in close proximity to heavily trafficked roads [18,37]. A 2007 paper from the Woman's Health Initiative used data from 573 PM_{2.5} monitors to follow over 65,000 women prospectively. They reported very high hazard ratios for cardiovascular events (1.76; 95% CI, 1.25 to 2.47) possibly due to the fine grain of exposure monitoring [18]. In contrast, studies that relied on central monitors [27,28] or interpolations from central monitors to highways are prone to exposure misclassification because individuals living close to highways will have a higher exposure than the general area. A possible concern with this interpretation is that social gradients may also situate poorer neighborhoods with potentially more susceptible populations closer to highways [38-40].

At a finer grain, Hoek et al. [24] estimated home exposure to nitrogen dioxide (NO₂) and black smoke for about 5,000 participants in the Netherlands Cohort Study on Diet and Cancer. Modeled exposure took into consideration proximity to freeways and main roads (100 m and 50 m, respectively). Cardiopulmonary mortality was associated with both modeled levels of pollutants and living near a major road with associations less strong for background levels of both pollutants. A case-control study [41], found a 5% increase in acute myocardial infarction associated with living within 100 m of major roadways. A recent analysis of cohort data found that traffic density was a predictor of mortality more so than was ambient air pollution [42]. There is a need for studies that assess exposure at these scales, e.g., immediate vicinity of highways, to test whether cardiac risk increases still more at even smaller scales.

Although we cannot review it in full here, we note that evidence beyond the epidemiological literature support the contention that PM_{2.5} and UFP (a sub-fraction of PM_{2.5}) have adverse cardiovascular effects [16,17]. PM_{2.5} appears

Table 2: Summary of near-highway health effects studies

Citation	Location	Highway traffic intensity ^a	Pollutants measured ^b	Distance from highway	Health Outcomes	Statistical association ^c
Schwartz et al. 2005 (22)	Boston	NA	PM _{2.5} , BC, CO	NA	Heart rate variability	Decreases in measures of heart rate variability
Adar et al. 2007 (23)	St. Louis, Missouri	NA	PM _{2.5} , BC, UFP	On highway in busses	Heart rate variability	Decreases in measures of heart rate variability
Hoek et al. 2002 (24)	Netherlands	NA	BC, NO ₂	Continuous ^d	Cardio-pulmonary mortality, lung cancer	1.41 OR for living near road
Tonne et al. 2007 (41)	Worcester, Mass.	NA	PM _{2.5}	Continuous ^d	Acute myocardial infarction (AMI)	5% increase in odds of AMI
Venn et al. 2001 (49)	Nottingham, UK	NA	NA	Continuous ^d	Wheezing in children	1.08 OR for living w/ in 150 m of road
Nicolai et al. 2003 (58)	Munich, Germany	>30,000 veh/d	Soot, benzene, NO ₂	Traffic counts within 50 m of house	Asthma, respiratory symptoms	1.79 OR for asthma and high traffic volume
Gauderman et al. 2005 (65)	Southern California	NA	NO ₂	Continuous ^d	Asthma, respiratory symptoms	Increased asthma closer to freeways
McConnell et al. 2006 (57)	Southern California	NA	NA	Continuous ^d	Asthma	Large risk for children living w/in 75 m of road
Ryan, et al. 2007 (59)	Cincinnati, Ohio	> 1,000 trucks/d	PM _{2.5}	400 m	Wheezing in children	NA
Kim et al. 2004 (60)	San Francisco	90,000 – 210,000 veh/d	PM, BC, NO _x	School sites	Childhood asthma	1.07 OR for high levels of NO _x
Wjst et al. 1993 (68)	Munich, Germany	7,000–125,000 veh/d	NO _x , CO	School sites	Asthma, bronchitis	Several statistical associations found
Brunekreef et al. 1997 (69)	Netherlands	80,000 – 152,000 veh/d	PM ₁₀ , NO ₂	Continuous ^d	Lung function	Decreased FEV with proximity to high truck traffic
Janssen et al. 2003 (74)	Netherlands	30,000–155,000 veh/d	PM _{2.5} , NO ₂ , benzene	< 400 m ^c	Lung function, respiratory symptoms	No association with lung function
Peters et al. 1999 (82)	Southern California	NA	PM ₁₀ , NO ₂	NA	Asthma, bronchitis, cough, wheeze	1.54 OR of wheeze for boys with exposure to NO ₂
Brauer et al. 2007 (67)	Netherlands	Highways and streets	PM _{2.5} , NO ₂ , soot	Modeled exposure	Asthma, allergy, bronchitis, respiratory symptoms	Strongest association was with food allergies
Visser et al. 2004 (91)	Amsterdam	> 10,000 veh/d	NA	NA	Cancer	Multiple associations
Vineis et al. 2006 (87)	10 European countries	NA	PM ₁₀ , NO ₂ , SO ₂	NA	Cancer	1.46 OR near heavy traffic, 1.30 OR for high exposure to NO ₂
Gauderman et al. 2007 (73)	Southern California	NA	PM ₁₀ , NO ₂	Continuous ^d	Lung Function	Decreased FEV for those living near freeway

^aAs defined in article cited (veh/d = vehicles per day; veh/h = vehicles per hour).

^bUFP = ultrafine particles; FP = fine particles; PM_{2.5} = particles with aerodynamic diameter ≤ 2.5 μm; PM₁₀ = particles with aerodynamic diameter ≤ 10 μm; BC = black carbon; PPAH = particle-bound polycyclic aromatic hydrocarbons; VOCs = volatile organic compounds

^cPollutant measurements were made along a transect away from the highway

^dProximity of each participant to a major road was calculated using GIS software

^eStatistical association between proximity to highway or exposure to traffic-generated pollutants and measured health outcomes

NA = not applicable; measurements were not made.

to be a risk factor for cardiovascular disease via mechanisms that likely include pulmonary and systemic inflammation, accelerated atherosclerosis and altered cardiac autonomic function [17,22,43-46]. Uptake of particles or particle constituents in the blood can affect the autonomic control of the heart and circulatory system. Black smoke, a large proportion of which is derived from mobile source emissions [30], has a high pulmonary deposition efficiency, and due to their surface area-to-volume ratios can carry relatively more adsorbed and condensed toxic air pollutants (e.g., PPAH) compared to larger particles [17,47,48]. Based on high particle numbers, high lung deposition efficiency and surface chemistry, UFP may provide a greater potential than PM_{2.5} for inducing inflammation [10]. UFPs have high cytotoxic reactive oxygen species (ROS) activity, through which numerous

inflammatory responses are induced, compared to other particles [10]. Chronically elevated UFP levels such as those to which residents living near heavily trafficked roadways are likely exposed can lead to long-term or repeated increases in systemic inflammation that promote arteriosclerosis [18,29,34,37].

Asthma and highway exposures

Evidence that near highway exposures present elevated risk is relatively well developed with respect to child asthma studies. These studies have evolved over time with the use of different methodologies. Studies that used larger geographic frames and/or overall traffic in the vicinity of the home or school [49-52] or that used self-report of traffic intensity [53] found no association with asthma prevalence. Most recent child asthma studies have,

instead, used increasingly narrow definitions of proximity to traffic, including air monitoring or modeling) and have focused on major highways instead of street traffic [54-59]. All of these studies have found statistically significant associations between the prevalence of asthma or wheezing and living very close to high volume vehicle roadways. Confounders considered included housing conditions (pests, pets, gas stoves, water damage), exposure to tobacco smoke, various measures of socioeconomic status (SES), age, sex, and atopy, albeit self-reported and not all in a single study.

Multiple studies have found girls to be at greater risk than boys for asthma resulting from highway exposure [55,57,60]. A recent study also reports elevated risk only for children who moved next to the highway before they were 2 years of age, suggesting that early childhood exposure may be key [57]. The combined evidence suggests that living within 100 meters of major highways is a risk factor, although smaller distances may also result in graded increases in risk. The neglect of wind direction and the absence of air monitoring from some studies are notable missing factors. Additionally, recent concerns have been raised that geocoding (attaching a physical location to addresses) could introduce bias due to inaccuracy in locations [61].

Studies that rely on general area monitoring of ambient pollution and assess regional pollution on a scale orders of magnitude greater than the near-roadway gradients have also found associations between traffic generated pollution (CO and NO_x) and prevalence of asthma [62] or hospital admission for asthma [63]. Lweguga-Mukasa et al. [64] monitored air up and down wind of a major motor vehicle bridge complex in Buffalo, NY and found that UFP were higher downwind, dropping off with distance. Their statistical models did not, however, support an association of UFP with asthma. A study in the San Francisco Bay Area measured PM_{2.5}, BC and NO_x over several months next to schools and found both higher pollution levels downwind from highways and a linear association of BC with asthma in long-term residents [60].

Gauderman et al. [65] measured NO₂ next to homes of 208 children. They found an odds ratio (OR) of 1.83 (confidence interval (CI): 1.04-3.22) for outdoor NO₂ (probably a surrogate for total highway pollution) and lifetime diagnosis of asthma. They also found a similar association with distance from residence to freeway. Self-report was used to control for numerous confounders, including tobacco smoke, SES, gas stoves, mildew, water damage, cockroaches and pets which did not substantially affect the association. Gauderman's study suggests that ambient air monitoring at the residence substantially increases sta-

tistical power to detect association of asthma with highway exposures.

Modeling of elemental carbon attributable to traffic near roadways based on ambient air monitoring of PM_{2.5} has recently emerged as a viable approach and a study using this method found an association with infant wheezing. The modeled values appear to be better predictors than proximity. Elevation of the residence relative to traffic was also an important factor in this study [66]. A 2007 paper reported on modeled NO₂, PM_{2.5} and soot and the association of these values with asthma and various respiratory symptoms in the Netherlands [67]. While finding modest statistically significant associations for asthma and symptoms, it is somewhat surprising that they found stronger associations for development of sensitization to food allergens.

Pediatric lung function and traffic-related air pollution

Studies of association of children's lung function with traffic pollutants have used a variety of measures of exposure, including: traffic density, distance to roadways, area (city) monitors, monitoring at the home or school and personal monitoring. Studies have assessed both chronic effects on lung development and acute effects and have been both cross-sectional and longitudinal. The wide range of approaches somewhat complicates evaluation of the literature.

Traffic density in school districts in Munich was associated with decreases in forced vital capacity (FVC), forced expiratory volume in 1 second (FEV₁), FEV₁/FVC and other measures, although the 2-kilometer (km) areas, the use of sitting position for spirometry and problems with translation for non-German children were limitations [68]. Brunekreef et al. [69] used distance from major roadways, considered wind direction and measured black smoke and NO₂ inside schools. They found the largest decrements in lung function in girls living within 300 m of the roadways.

A longitudinal study of children (average age at start = 10 years) in Southern California reported results at 4 [70] and 8 years [71]. Multiple air pollutants were measured at sites in 12 communities. Due to substantial attrition, only 42% of children enrolled at the start were available for the 8-year follow-up. Substantially lower growth in FEV₁ was associated with PM₁₀, NO₂, PM_{2.5}, acid vapor and elemental carbon at 4 and at 8 years. The analysis could not indicate whether the effects seen were reversible or not [72]. In 2007, it was reported from this same cohort that living within 500 m of a freeway was reported to be associated with reduced lung function [73].

A Dutch study [74] measured PM_{2.5}, NO₂, benzene and EC for one year at 24 schools located within 400 m of major roadways. While associations were seen between symptoms and truck traffic and measured pollutants, there was no significant association between any of the environmental measures and FVC < 85% or FEV₁ < 85%. Restricting the analysis to children living within 500 m of highways generally increased ORs.

Personal exposure monitoring of NO₂ as a surrogate for total traffic pollutants with 298 Korean college students found statistically significant associations with FEV₁, FEV₁/FVC, and forced expiratory volume between 25 and 75% (FEV₂₅₋₇₅), but not with FVC. The multivariate regression model presented suggests that FEV₂₅₋₇₅ was the outcome measure that most clearly showed an effect [75]. Cross-sectional studies of children in Korea [76] and France [77] also indicate that lung function is diminished in association with area pollutants that largely derive from traffic.

Time series studies suggest there are also acute effects. A study of 19 asthmatic children measured PM via personally carried monitors, at homes and at central site monitors. The study found deficits in FEV₁ that were associated with PM, although many sources besides traffic contributed to exposure. In addition, the results suggest that ability to see associations with health outcomes improves at finer scale of monitoring [78]. PM was associated with reduced FEV₁ and FVC in only the asthmatic subset of children in a Seattle study [79]. Studies have also seen associations between PM and self reported peak flow measurements [80,81] and asthmatic symptoms [82].

Cancer and near highway exposures

As noted above, both the Six-Cities Study [27] and the American Cancer Society (ACS) Study [28] found associations between PM and lung cancer. Follow-up studies using the ACS cohort [29,37] and the Six-Studies cohort [83] that controlled for smoking and other risk factors also demonstrated significant associations between PM and lung cancer. The original studies were subject to intensive replication, validation, and re-analysis which confirmed the original findings [84].

The ASHMOG study [85] was designed to look specifically at lung cancer and air pollution among Seventh-day Adventists in California, taking advantage of their low smoking rates. Air pollution was interpolated to centroids of zip codes from ambient air monitoring stations. Highway proximity was not considered. The study found associations with ozone (its primary pollutant of consideration), PM₁₀ and SO₂. Notably, these are not the pollutants that would be expected to be substantially elevated immediately adjacent to highways.

A case control study of residents of Stockholm, Sweden modeled traffic-related NO₂ levels at their homes over 30 years and found that the strongest association involved a 20 year latency period [86]. Another case control study drawn from the European Prospective Investigation on Cancer and Nutrition found statistically significantly elevated ORs for lung cancer with proximity to heavy traffic (>10,000 cars per day) as well as for NO₂ and PM₁₀ at nearby ambient monitoring stations [87]. Nafstad et al. [88] used modeled NO₂ and SO₂ concentrations at the homes of over 16,000 men in Oslo to test associations with lung cancer incidence. The models included traffic and point sources. The study found small, but statistically significant associations between NO₂ and lung cancer. Problems that run through all these studies are weak measures of exposure to secondhand tobacco smoke, the use of main roads rather than highways as the exposure group and modeled rather than measured air pollutants.

A study of regional pollution in Japan and a case control study of more localized pollution in a town in Italy also found associations between NO₂ and lung cancer and PM and lung cancer [89,90]. On the other hand, a study that calculated SIRs for specific cancers across lower and higher traffic intensity found little evidence of an association with a range of cancers [91].

The plausibility of near-highway pollution causing lung cancer is bolstered by the presence of known carcinogens in diesel PM. The US EPA has concluded after reviewing the literature that diesel exhaust is "likely to be carcinogenic to humans by inhalation" [92]. An interesting study of UFP and DNA damage adds credibility to an association with cancer [93]. This study had participants bicycle in traffic in Copenhagen and measured personal exposure to UFP and DNA oxidation and strand breaks in mononuclear blood cells. Bicycling in traffic increased UFP exposure and oxidative damage to DNA, thus demonstrating an association between DNA damage and UFP exposure *in vivo*.

Policy and research recommendations

Based on the literature reviewed above it is plausible that gradients of pollutants next to highways carry elevated health risks that may be larger than the risks of general area ambient pollutants. While the evidence is considerable, it is not overwhelming and is weak in some areas. The strongest evidence comes from studies of development of asthma and reduction of lung function during childhood, while the studies of cardiac health risk require extrapolation from area studies of smaller and larger geographic scales and inference from toxicology laboratory investigations. The lung cancer studies, because they include pollutants such as O₃ that are not locally concentrated, are not particularly strong in terms of the case for near-high-

way risk. There is a need for lung cancer research that uses major highways rather than heavily trafficked roads as the environmental exposure.

While more studies of asthma and lung function in children are needed to confirm existing findings, especially studies that integrate exposure at school, home and during commuting, to refine our knowledge about the association, we would point to the greater need for studies of cardiac health and lung cancer and their association with near highway exposures as the primary research areas needing to be developed. Many of the studies of PM and cardiac or pulmonary health have focused on mortality. Near highway mortality studies may be possible, but would be lengthy if they were initiated as prospective cohorts. Other possibilities include retrospective case control studies of mortality, cross sectional studies or prospective studies that have end points short of mortality, such as biological markers of disease. For all health end points there is a need for studies that adequately address the possible confounding of SES with proximity to highways. There is good reason to think that property values decline near highways and that control for SES by, for example, income, may be inadequate.

Because of the incomplete development of the science regarding the health risks of near highway exposures and the high cost and implication of at least some possible changes in planning and development, policy decisions are complicated. The State of California has largely prohibited siting of schools within 500 feet of freeways (SB 352; approved by the governor October 2, 2003). Perhaps this is a viable model for other states or for national-level response. As it is the only such law of which we are aware, there may be other approaches that will be and should be tried. One limitation of the California approach is that it does nothing to address the population already exposed at schools currently cited near freeways and does not address residence near freeways.

Conclusion

The most susceptible (and overlooked) population in the US subject to serious health effects from air pollution may be those who live very near major regional transportation route, especially highways. Policies that have been technology based and regional in orientation do not efficiently address the very large exposure and health gradients suffered by these populations. This is problematic because even regions that EPA has deemed to be in regional PM "attainment" still include very large numbers of near highway residents who currently are not protected. There is a need for more research, but also a need to begin to explore policy options that would protect the exposed population.

Abbreviations

UFP = ultra fine particles

BC = black carbon

NO₂ = nitrogen dioxide

NO_x = oxides of nitrogen

CO = carbon monoxide

PM = particulate matter

PM_{2.5} = particulate matter less than 2.5 um

PM₁₀ = particulate matter less than 10 um

PPAH = particle bound polyaromatic hydrocarbons

EC = elemental carbon

VOC = volatile organic compounds

SO₂ = sulfur dioxide

ACS = American Cancer Society

SES = socioeconomic status

EPA = Environmental Protection Agency

OR = odds ratio

FEV₁ = forced expiratory volume in 1 second

FEV₁/FVC = ratio of FEV₁ and forced vital capacity

FEV₂₅₋₇₅ = forced expiratory volume between 25 and 75

FVC = forced vital capacity

ug/m³ = micrograms per cubic meter of air

m = meters

um = micrometers

veh/d = vehicles per day

veh/h = vehicles per hour

Competing interests

The author(s) declare that they have no competing interests.

Authors' contributions

DB took the lead on the manuscript. He co-wrote the background and wrote the sections on asthma, lung function and cancer and the conclusions. JLD wrote the section on air pollutants near roadways and contributed substantially to the background. CR wrote the section on cardiovascular health. All authors participated in editing and refining the manuscript and all read it multiple times, including the final version.

Acknowledgements

We thank Wig Zamore for useful insights into the topic. The Jonathan M Tisch College of Citizenship and Public Service partially supported the effort of Doug Brugge and Christine Rioux. Figure 1 was reproduced with permission of the publisher.

References

1. **American Housing Survey for the United States: 2003 Series H150/03** [<http://www.census.gov/hhes/www/housing/ahs/ahs03/ahs03.html>]. Accessed May 2007.
2. Massachusetts Fact Book; 2004.
3. Chambers LA: **Classification and extent of air pollution problems**. In *Air Pollution Volume 1*. 3rd edition. Edited by: Stern AC. Academic Press, NY; 1976.
4. Rogge WF, Hildemann LM, Mazurek MA, Cass GR, Simoneit BRT: **Sources of fine organic aerosol. 2. Noncatalyst and catalyst-equipped automobiles and heavy-duty diesel trucks**. *Environmental Science Technology* 1993, **27**:636-651.
5. Graedel TE, Hawkins DT, Claxton LD: **Atmospheric Chemical Compounds: Sources, Occurrence, and Bioassay**. Academic Press, Inc., New York, NY; 1986.
6. Shi JP, Khan AA, Harrison RM: **Measurements of ultrafine particle concentration and size distribution in the urban atmosphere**. *The Science of the Total Environment* 1999, **235**:51-64.
7. Zhu Y, Hinds WC, Kim S, Sioutas C: **Concentration and size distribution of ultrafine particles near a major highway**. *Journal of the Air and Waste Management Association* 2002, **52**(9):1032-1042.
8. Zhu Y, Hinds WC, Kim S, Shen S, Sioutas C: **Study of ultrafine particles near a major highway with heavy-duty diesel traffic**. *Atmospheric Environment* 2002, **36**:4323-4335.
9. Zhang KM, Wexler AS, Zhu Y, Hinds WC, Sioutas C: **Evolution of particle number distribution near roadways. Part II: the 'Road-to-Ambient' process**. *Atmospheric Environment* 2004, **38**:6655-6665.
10. Sioutas C, Delfino RJ, Singh M: **Exposure assessment for atmospheric ultrafine particles (UFP) and implications in epidemiologic research**. *Environmental Health Perspectives* 2005, **113**(8):947-955.
11. Hitchens J, Morawska L, Wolff R, Gilbert D: **Concentrations of submicrometre particles from vehicle emissions near a major road**. *Atmospheric Environment* 2000, **34**:51-59.
12. Morawska L, Thomas S, Gilbert D, Greenaway C, Rijnders E: **A study of the horizontal and vertical profile of submicrometer particulates in relation to a busy road**. *Atmospheric Environment* 1999, **33**:1261-1274.
13. Fischer PH, Hoek G, van Reeuwijk H, Briggs DJ, Lebret E, van Wijnen JH, Kingham S, Elliott PE: **Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam**. *Atmospheric Environment* 2000, **34**:3713-3722.
14. Roorda-Knape MC, Janssen NAH, De Hartog JJ, van Vliet PHN, Harssema H, Brunekreef B: **Air pollution from traffic in city districts near major motorways**. *Atmospheric Environment* 1998, **32**:1921-1930.
15. Janssen NAH, van Vliet PHN, Aarts F, Harssema H, Brunekreef B: **Assessment of exposure to traffic related air pollution of children attending schools near motorways**. *Atmospheric Environment* 2001, **35**:3875-3884.
16. National Research Council, Committee on Research Priorities for Airborne Particulate Matter: **Research priorities for airborne particulate matter, IV: continuing research progress**. National Academy Press, Washington, DC; 2004.
17. US Environmental Protection Agency: **Air quality criteria for particulate matter** Research Triangle Park; 2004.
18. Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD: **Long-term exposure to air pollution and incidence of cardiovascular events in women**. *The New England Journal of Medicine* 2007, **356**:447-458.
19. Riediker M, Cascio WE, Griggs TR, Herbst MC, Bromberg PA, Neas L, Williams RW, Devlin RB: **Particulate matter exposure in cars is associated with cardiovascular effects in healthy young men**. *American Journal of Respiratory and Critical Care Medicine* 2004, **169**:934-940.
20. Hoffmann B, Moebus S, Stang A, Beck E, Dragano N, Möhlenkamp S, Schermund A, Memmesheimer M, Mann K, Erbel R, Jockel KH, Heinz Nixdorf RECALL Study Investigative Group: **Residence close to high traffic and prevalence of coronary heart disease**. *European Heart Journal* 2006, **27**:2696-2702.
21. Ruckerl R, Greven S, Ljungman P, Aalto P, Antoniadou C, Bellander T, Berglund N, Chrysohoou C, Forastiere F, Jacquemin B, von Klot S, Koenig W, Kuchenhoff H, Lanki T, Pekkanen J, Perucci CA, Schneider A, Sunyer J, Peters A: **Air pollution and inflammation (IL-6, CRP, fibrinogen) in myocardial infarction survivors**. *Environmental Health Perspectives* 2007, **115**:1072-1080.
22. Schwartz J, Itonjua A, Suh H, Verrier M, Zanobetti A, Syring M, Nearing B, Verrier R, Stone P, MacCallum G, Speizer FE, Gold DR: **Traffic related pollution and heart rate variability in a panel of elderly subjects**. *Thorax* 2005, **60**:455-461.
23. Adar SD, Gold DR, Coull BA, Schwartz J, Stone P, Suh H: **Focused exposures to airborne traffic particles and heart rate variability in the elderly**. *Epidemiology* 2007, **18**:95-103.
24. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA: **Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study**. *The Lancet* 2002, **360**:1203-1209.
25. Peters A, von Klot S, Heier M, Trentinaglia I, Hormann A, Wichmann HE, Lowel H: **Exposure to traffic and the onset of myocardial infarction**. *The New England Journal of Medicine* 2004, **351**:1861-70.
26. Pope CA, Dockery DW: **Health effects of fine particulate air pollution: lines that connect**. *Journal of Air and Waste Management* 2006, **56**(6):709-742.
27. Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE: **An association between air pollution and mortality in six U.S. cities**. *New England Journal of Medicine* 1993, **329**:1753-9.
28. Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Hath CW: **Particulate air pollution as a predictor of mortality in a prospective study of US adults**. *American Journal of Respiratory and Critical Care Medicine* 1995, **151**:669-674.
29. Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD: **Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution**. *Journal of the American Medical Association* 2002, **287**:1132-1141.
30. Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN: **Ambient air pollution and Atherosclerosis in Los Angeles**. *Environmental Health Perspectives* 2005, **113**:201-206.
31. Peters A: **Particulate matter and heart disease: Evidence from epidemiological studies**. *Toxicology and Applied Pharmacology* 2005:477-482.
32. Wheeler A, Zanobetti A, Gold DR, Schwartz J, Stone P, Suh H: **The relationship between ambient air pollution and heart rate variability differs for individuals with heart and pulmonary disease**. *Environmental Health Perspectives* 2006, **114**:560-566.
33. Chuang K, Chan C, Chen N, Su T, Lin L: **Effects of particle size fractions on reducing heart rate variability in cardiac and hypertensive patients**. *Environmental Health Perspectives* 2005, **113**:1693-1697.
34. Chan C, Chuang K, Shiao G, Lin L: **Personal exposure to submicrometer particles and heart rate variability in human subjects**. *Environmental Health Perspectives* 2004, **112**:1063-1067.
35. Brauer M, Hoek G, van Vliet P, Meliefste K, Fischer P, Gehring U, Heinrich J, Cyrys J, Bellander T, Lewne M, Brunekreef B: **Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems**. *Epidemiology* 2003, **14**:228-239.

36. Brunekreef B, Holgate ST: **Air pollution and health.** *Lancet* 2002, **360**:1233-1242.
37. Jerrett M, Finkelstein M: **Geographies of risk in studies linking chronic air pollution exposure to health outcomes.** *Journal of Toxicology and Environmental Health* 2005, **68**:1207-1242.
38. O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwartz J: **Workshop on Air Pollution and Socioeconomic Conditions. Health, wealth, and air pollution: advancing theory and methods.** *Environmental Health Perspectives* 2003, **111**:1861-1870.
39. Jerrett M, Burnett RT, Ma R, Pope CA, Krewski D, Newbold KB, Thurston G, Shi Y, Finkelstein N, Calle EE, Thun MJ: **Spatial Analysis of Air Pollution and Mortality in Los Angeles.** *Epidemiology* 2005, **16**(6):727-736.
40. Finkelstein M, Jerrett M, Sears MR: **Environmental inequality and circulatory disease mortality gradients.** *Journal of Epidemiology and Community Health* 2005, **59**:481-487.
41. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J: **A case-control analysis of exposure to traffic and acute myocardial infarction.** *Environmental Health Perspectives* 2007, **115**:53-57.
42. Lipfert FW, Wyzga RE, Baty JD, Miller JP: **Traffic density as a surrogate measure of environmental exposures in studies of air pollution health effects: Long-term mortality in a cohort of US veterans.** *Atmospheric Environment* 2006, **40**:154-169.
43. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ: **Cardiovascular mortality and long-term exposure to particulate air pollution – Epidemiological evidence of general pathophysiological pathways of disease.** *Circulation* 2004, **109**:71-77.
44. Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC, Tager I: **Air pollution and cardiovascular disease: a statement for healthcare professionals from the expert panel on population and prevention science of the American Heart Association.** *Circulation* 2004, **109**:2655-2671.
45. Sun Q, Wang A, Jin X, Natanzon A, Duquaine D, Brook RD, Aguinaldo JG, Fayad Z, Fuster V, Lippman M, Chen LC, Rajagopalan S: **Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model.** *Journal of the American Medical Association* 2005, **294**:3003-3010.
46. Sandhu RS, Petroni DH, George WJ: **Ambient particulate matter, C-reactive protein, and coronary artery disease.** *Inhalation Toxicology* 2005, **17**:409-413.
47. Oberdorster G: **Pulmonary effects of inhaled ultrafine particles.** *International Archives of Occupational and Environmental Health* 2001, **65**:1531-1543.
48. Delfino RJ, Sioutas C, Malik S: **Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health.** *Environmental Health Perspectives* 2005, **113**:934-946.
49. Venn A, Lewis S, Cooper M, Hubbard R, Hill I, Boddy R, Bell M, Britton J: **Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study.** *Occupational & Environmental Medicine* 2000, **57**:152-158.
50. Waldron G, Pottle B, Dod J: **Asthma and the motorways – One district's experience.** *Journal of Public Health Medicine* 1995, **17**:85-89.
51. Lewis SA, Antoniak M, Venn AJ, et al: **Secondhand smoke, dietary fruit intake, road traffic exposures, and the prevalence of asthma: A cross-sectional study in young children.** *American Journal of Epidemiology* 2005, **161**:406-411.
52. English P, Neutra R, Scaif R, Sullivan M, Waller L, Zhu L: **Examining associations between childhood asthma and traffic flow using a geographic information system.** *Environmental Health Perspectives* 1999, **107**:761-767.
53. Heinrich J, Topp R, Gerring U, Thefeld W: **Traffic at residential address, respiratory health, and atopy in adults; the National German Health Survey 1998.** *Environmental Research* 2005, **98**:240-249.
54. Van Vliet P, Knappe M, de Hartog J, Janssen N, Harssema H, Brunekreef B: **Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways.** *Environmental Research* 1997, **74**:122-132.
55. Venn AJ, Lewis SA, Cooper M, Hubbard R, Britton J: **Living near a main road and the risk of wheezing illness in children.** *American Journal of Respiratory and Critical Care Medicine* 2001, **164**(12):2177-2180.
56. Venn A, Yemaneberhan H, Lewis S, Parry E, Britton J: **Proximity of the home to roads and the risk of wheeze in an Ethiopian population.** *Occupational and Environmental Medicine* 2005, **62**:376-380.
57. McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, Kunzli N, Gauderman J, Avol E, Thomas D, Peters J: **Traffic susceptibility, and childhood asthma.** *Environmental Health Perspectives* 2006, **114**:766-772.
58. Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, von Mutius E: **Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children.** *European Respiratory Journal* 2003, **21**:956-963.
59. Ryan PH, LeMasters , Biswas P, Levin L, Hu S, Lindsey M, Bernstein DI, Lockett J, Villareal M, Hershey GKH, Grinshpun SA: **A comparison of proximity and land use regression traffic exposure models and wheezing in infants.** *Environmental Health Perspectives* 2007, **115**:278-284.
60. Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B: **Traffic-related air pollution near busy roads: The East Bay children's respiratory health study.** *American Journal of Respiratory and Critical Care Medicine* 2004, **170**:520-526.
61. Ong P, Graham M, Houston D: **Policy and programmatic importance of spatial alignment of data sources.** *Am J Public Health* 2006, **96**:499-504.
62. Hwang BF, Lee YL, Lin YC, Jaakkola JJ, Guo YL: **Traffic related air pollution as a determinant of asthma among Taiwanese school children.** *Thorax* 2005, **60**:467-473.
63. Migliaretti G, Cadum E, Migliore E, et al: **Traffic air pollution and hospital admissions for asthma: A case control approach in a Turin (Italy) population.** *International Archives of Occupational and Environmental Health* 2005, **78**:164-169.
64. Lweguga-Mukasa JS, Oyana TJ, Johnson C: **Local ecological factors, ultrafine particulate concentrations, and asthma prevalence rates in Buffalo, New York, neighborhoods.** *Journal of Asthma* 2005, **42**:337-348.
65. Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R: **Childhood asthma and exposure to traffic and nitrogen dioxide.** *Epidemiology* 2005, **16**:737-743.
66. Ryan PH, LeMasters GK, Biswas P, Levin L, Hu S, Lindsey M: **A comparison of proximity and land use regression traffic exposure models and wheezing in infants.** *Environmental Health Perspectives* 2007, **115**:278-284.
67. Brauer M, Hoek G, Smit HA, de Jongste JC, Gerritsen J, Postma DS, Kerkhof M, Brunekreef B: **Air pollution and development of asthma, allergy and infections in a birth cohort.** *European Respiratory Journal* 2007, **29**:879-888.
68. Wjst M, Reitmeir P, Dodd S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF, von Mutius E: **Road traffic and adverse effects on respiratory health in children.** *British Medical Journal* 1993, **307**:596-307.
69. Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knappe M, van Vliet P: **Air pollution from truck traffic and lung function in children living near motorways.** *Epidemiology* 1997, **8**:298-303.
70. Gauderman WJ, McConnell , Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J: **Association between air pollution and lung function growth in Southern California Children.** *American Journal of Respiratory and Critical Care Medicine* 2000, **162**(4 Pt 1):1383-1390.
71. Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J: **The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age.** *New England Journal of Medicine* 2005, **351**:1057-67.
72. Merkus PJFM: **Air pollution and lung function.** *New England Journal of Medicine* 2005, **351**:2652.
73. Gauderman WJ, Vora H, McConnell R, Berhane K, Gilliland F, Thomas D, Lurmann F, Avol E, Kunzli N, Jarrett M, Peters J: **Effect of exposure to traffic on lung development from 10 to 18 years of age: A cohort study.** *The Lancet* 2007, **369**:571-577.
74. Janssen NA-H, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, Fischer P: **The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyper-**

- responsiveness, and respiratory symptoms in Dutch school children. *Environmental Health Perspectives* 2003, **111**:1512-1518.
75. Hong Y-C, Leem J-H, Lee K-H, Park D-H, Jang J-Y, Kim S-T, Ha E-H: **Exposure to air pollution and pulmonary function in university students.** *International Archives of Occupational and Environmental Health* 2005, **78**:132-138.
 76. Kim HJ, Lim DH, Kim JK, Jeong SJ, Son BK: **Effects of particulate matter (PM10) on pulmonary function of middle school children.** *Journal of the Korean Medical Society* 2005, **20**(1):42-45.
 77. Penard-Morand C, Charpin D, Raheison C, Kopferschmitt C, Cailaud D, Lavaud F, Annesi-Maesano I: **Long-term exposure to background air pollution related to respiratory and allergic health in schoolchildren.** *Clinical and Experimental Allergy* 2005, **35**:1279-1287.
 78. Delfino RJ, Quintana PJE, Floro J, Gastanaga VM, Samimi BS, Klienman MT, Liu LJ, Bufalino C, Wu C, McLaren CE: **Association of FEV1 in asthmatic children with personal and microenvironment exposure to airborne particulate matter.** *Environmental Health Perspectives* 2004, **112**:932-941.
 79. Koenig JQ, Larson TV, Hanley QS, Rebolledo V, Dumler K, Checkoway H, Wang SZ, Lin D, Pierson WE: **Pulmonary function changes in children associated with fine particulate matter.** *Environmental Research* 1993, **63**:26-38.
 80. Van der Zee SC, Hoek G, Boezen HM, Schouten JP, van Wijnen JH, Brunekreef B: **Acute effects of urban air pollution on respiratory health of children with and without chronic respiratory symptoms.** *Occupational and Environmental Medicine* 1999, **56**(12):802-813.
 81. Pekkenen J, Timonen KL, Ruuskanen J, Reponen A, Mirme A: **Effects of ultrafine and fine particulates in urban air on peak expiratory flow among children with asthmatic symptoms.** *Environmental Research* 1997, **74**:24-33.
 82. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, Thomas DC: **A study of twelve Southern California communities with differing levels and types of air pollution: Prevalence of respiratory morbidity.** *American Journal of Respiratory and Critical Care Medicine* 1999, **159**(3):760-767.
 83. Laden F, Schwartz J, Speizer FE, Dockery DE: **Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six-cities study.** *American Journal of Respiratory and Critical Care Medicine* 2006, **173**:667-672.
 84. Health Effects Institute: **Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution mortality.** *Final Version; Boston, MA* 2000.
 85. Beeson WL, Abbey DE, Knutsen SF: **Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: Results from the ASHMOG study.** *Environmental Health Perspectives* 1998, **106**:813-823.
 86. Nyberg F, Gustavsson P, Jarup L, Bellander T, Berglind N, Jakobsson R, Pershagen G: **Urban air pollution and lung cancer in Stockholm.** *Epidemiology* 2000, **11**:487-495.
 87. Vineis P, Hoek G, Krzyzanowski M, Vigna-Tagliani F, Veglia F, Airoidi L, Autrup H, Dunning A, Garte S, Hainaut P, Malaveille C, Matullo G, Overvad K, Raaschou-Nielsen O, Clavel-Chapelon F, Linseisen J, Boeing H, Trichopoulou A, Palli D, Peluso M, Krogh V, Tumino R, Panico S, Bueno-De-Mesquita HB, Peeters PH, Lund EE, Gonzalez CA, Martinez C, Dorronsoro M, Barricarte A, Cirera L, Quiros JR, Berglund G, Forsberg B, Day NE, Key TJ, Saracci R, Kaaks R, Riboli E: **Air pollution and risk of lung cancer in a prospective study in Europe.** *International Journal of Cancer* 2006, **119**:169-174.
 88. Nafstad P, Haheim LL, Oftedal B, Gram F, Holme I, Hjermmann I, Leren P: **Lung cancer and air pollution: A 27-year follow up of 16 209 Norwegian men.** *Thorax* 2003, **58**:1071-1076.
 89. Choi K-S, Inoue S, Shinozaki R: **Air pollution, temperature, and regional differences in lung cancer mortality in Japan.** *Archives of Environmental Health* 1997, **52**:160.
 90. Biggeri A, Barbone F, Lagazio C, Bovenzi M, Stanta G: **Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources.** *Environmental Health Perspectives* 1996, **104**:750-754.
 91. Visser O, van Wijnen JH, van Leeuwen FE: **Residential traffic density and cancer incidence in Amsterdam, 1989-1997.** *Cancer Causes & Control* 2004, **15**:331-339.
 92. US Environmental Protection Agency: **Health Assessment Document for Diesel Engine Exhaust.** Washington, DC. 2002.
 93. Vinzents PS, Meller P, Sorensen M, Knudsen LE, Hertel O, Jensen FP, et al.: **Personal exposure to ultrafine particulates and oxidative DNA damage.** *Environmental Health Perspectives* 2005, **113**:1485-1490.

Publish with **Bio Med Central** and every scientist can read your work free of charge

"BioMed Central will be the most significant development for disseminating the results of biomedical research in our lifetime."

Sir Paul Nurse, Cancer Research UK

Your research papers will be:

- available free of charge to the entire biomedical community
- peer reviewed and published immediately upon acceptance
- cited in PubMed and archived on PubMed Central
- yours — you keep the copyright

Submit your manuscript here:
http://www.biomedcentral.com/info/publishing_adv.asp

