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

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

Summary

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

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

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

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Department of Preventive Medicine, University of Southern California, 1540 Alcazar Street, Suite 220, Los Angeles, CA 90033, USA (WJ 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 Künzli 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

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 used 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 that completed the questionnaire. High socioeconomic status (23% of children, n=823) was defined as a parental

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 wepappendix

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 (FEV_1), and maximum midexpiratory flow rate (MMEF, also known as FEF_{25-75}). 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 values 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 categorisation distances 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 or values 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 FEV_1 values, we first fitted a regression model for observed FEV_1 (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 FEV_1 on the basis of this model and percent-predicted as observed divided by predicted FEV_1. 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, FEV_1, 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 FEV_1 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 FEV_1 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 socio-economic 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 FEV_1 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 FEV_1 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 between these six communities (data not shown).

	FVC (mL) difference (95% CI)	FEV1 (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 FEV_1 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 FEV_1 growth**

Furthermore, 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 FEV_1 growth were more pronounced.

See Online for webtables 1 and 2 and webfigure

	Regional pollutant effect*	p	Local freeway distance (m)						
			<500	p	500–1000	p	1000–1500	p	p for interaction†
1000–1800 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
$PM_{2·5}$	–110	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 FEV_1 growth from lowest to highest observed community–average concentration of the pollutant, specifically: per increase of 37·5 ppb ozone (1000–1800), 34·6 ppb of nitrogen dioxide, 9·6 ppb of acid vapour, 51·4 μg/m³ of PM10, 22·8 μg/m³ of PM2.5 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. PM_{10} =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 FEV_1 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 FEV_1 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 FEV_1 and MMEF than those who lived at least 1500 m from a freeway.

These deficits in average FEV_1 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 FEV_1 (p=0·013 relative to >1500 m) and 93·4% (89·1–97·7) for MMEF (p=0·006).

		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)	–69 (–160 to 22)
	500–1,000 m	–12 (–61 to 37)	–54 (–151 to 43)	–42 (–125 to 41)
	1000–1500 m	–30 (–80 to 21)	–81 (–181 to 19)	–52 (–137 to 33)
FEV_1	**Freeway distance**			
	<500 m	–23 (–73 to 28)	–121 (–219 to –23)	–98 (–182 to –15)
	500–1000 m	–32 (–78 to 14)	–93 (–183 to –4)	–61 (–137 to 15)
	1000–1500 m	–34 (–81 to 14)	–78 (–170 to 14)	–44 (–122 to 34)
MMEF	**Freeway distance**			
	<500 m	–57 (–169 to 56)	–230 (–432 to –28)	–173 (–327 to –19)
	500–1000 m	–92 (–195 to 10)	–105 (–289 to 79)	–12 (–152 to 128)
	1000–1500 m	–45 (–150 to 60)	–151 (–340 to 38)	–106 (–250 to 38)

*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 of follow-up**

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 FEV_1 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 FEV_1 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 are associated with diminished lung-function

development 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 study[29] 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-workers[32] 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 FEV_1, MMEF, and FVC. Chronic airway inflammation could produce our reported deficits in increased MMEF and FEV_1. 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

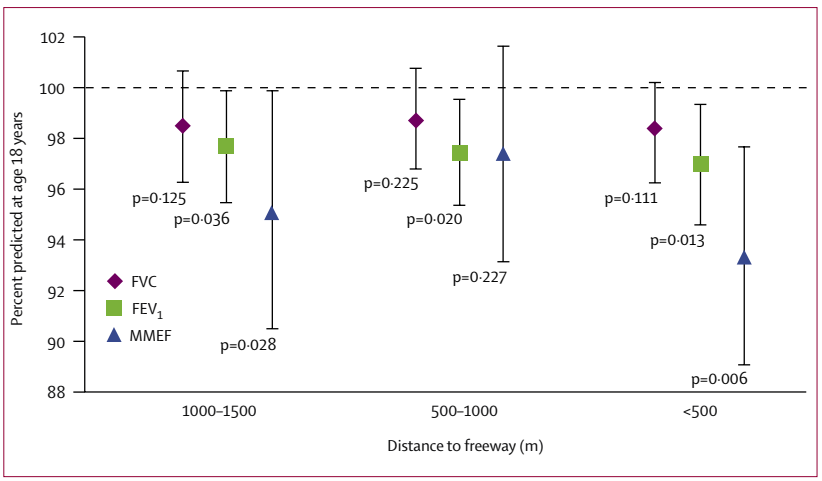

Figure: **Percent-predicted lung function at age 18 years versus residential distance from a freeway**
The horizontal line at 100% corresponds to the referent group, children living >1500 m from a freeway.

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.

Conflict of interest statement
We declare that we have no conflict of interest.

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Webtable

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

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

	n	Mean number of PTFs	Children with								
			8-years' follow-up				Race/Ethnic origin (%)				
			n	(%)	Female (%)	Asthma (%)	NHW	HW	AA	Asian	Other
Riverside	329	6·0	123	37·4	50·5	14·6	36·5	42·0	12·5	2·4	6·7
Atascadero	278	6·8	117	42·1	48·9	22·3	75·2	14·8	1·1	1·1	7·9
Alpine	308	6·2	121	39·3	50·1	12·9	75·0	18·8	0·0	0·3	5·8
Long Beach	320	6·1	141	44·1	47·5	13·9	32·2	24·7	18·4	15·3	9·4
San Dimas	293	6·4	117	39·9	50·2	15·3	50·2	32·4	3·1	9·2	5·1
Santa Maria	310	5·7	100	32·3	49·4	14·6	25·2	62·9	1·0	4·5	6·5
Lake Elsinore	306	6·0	104	34·0	50·0	12·5	64·3	25·8	2·3	2·0	5·6
Mira Loma	319	5·9	118	37·0	50·2	12·3	51·7	42·3	1·6	0·9	3·5
Upland	283	6·9	150	53·0	52·7	13·7	66·4	17·3	4·3	8·5	3·5
Lancaster	315	5·5	110	34·9	52·1	14·7	52·1	29·8	9·2	2·2	6·7
Lompoc	281	6·3	113	40·2	47·0	10·3	55·2	28·1	5·7	5·3	5·7
Lake Arrowhead	335	6·2	131	39·1	51·3	14·6	73·1	20·0	0·3	0·9	5·7
Overall	3677	6·2	1445	39·3	49·9	14·3	54·4	30·2	5·0	4·4	6·0

NHW=Non-Hispanic whites. HW=Hispanic whites. AA=African American. PFT=pulmonary-function test.

Webtable 1: **Participants' characteristics by community**

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

	n	Residential distance to dearest								Model–based pollution from							
		Freeway (m)				Major non-freeway road (m)				Freeways (quartile*)				Major non-freeway roads (quartile*)			
		<500	500–1000	1000–1500	>1500	<75	75–150	150–300	>300	4th	3rd	2nd	1st	4th	3rd	2nd	1st
Riverside	329	103	66	61	99	46	45	90	148	190	123	14	2	149	138	41	1
Atascadero	278	83	60	46	89	11	8	15	244	0	70	155	53	4	17	58	199
Alpine	308	81	54	42	131	41	9	31	227	14	135	141	18	21	43	73	171
Long Beach	320	54	64	54	148	55	79	78	108	264	54	2	0	311	9	0	0
San Dimas	293	47	145	83	18	45	47	62	139	282	8	1	2	169	114	9	1
Santa Maria	310	44	74	58	134	25	47	104	134	0	7	73	230	18	191	64	37
Lake Elsinore	306	12	17	7	270	32	33	50	191	1	41	184	80	17	27	103	159
Mira Loma	319	9	30	45	235	20	37	57	205	11	304	2	2	12	43	212	52
Upland	283	4	0	0	279	53	52	62	116	4	2	85	192	83	100	60	40
Lancaster	315	3	35	31	246	52	24	91	148	0	21	108	186	48	127	128	12
Lompoc	281	0	0	0	281	5	21	33	222	..	..	..	..	4	26	88	163
Lake Arrowhead	335	0	0	0	335	0	0	0	335	..	..	..	..	..	..	..	..
Total	3677	440	545	427	2265	385	402	673	2217	766	765	765	765	836	835	836	835

* There is no major freeway within Lompoc or Lake Arrowhead, and no major non-freeway road within Lake Arrowhead.

***Webtable 2*: Number of study participants within categories of four traffic indicators**

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

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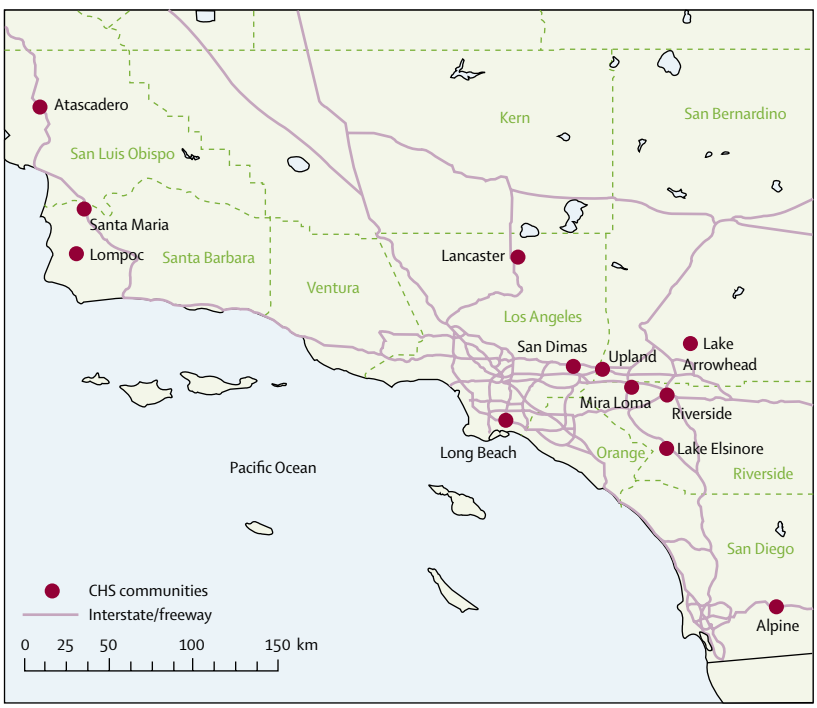
Correspondence to:
Dr W James Gauderman
jimg@usc.edu



Webfigure: **Location of the 12 Children's Health Study communities and the major freeways (purple lines) in southern California.**

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

Details of exposure assessment

Traffic exposures were assigned to each child on the basis of the residence at study entry. Residence addresses were standardised and their locations geocoded by use of the TeleAtlas database and software (Tele Atlas Inc., Menlo Park, CA, www.na.teleatlas.com). We used ERSI ArcGIS version 8.3 (ESRI, Redland, CA www.esri.com) software to calculate the distance from each residence to the nearest freeway, defined as an interstate freeway, US highway, or restricted-access highway, and to the nearest major non-freeway road, which included other types of highways and large roads. Yearly average daily traffic volumes were obtained from the California Department of Transportation Highway Performance Monitoring System for the year 2000. To obtain model-based estimates of traffic-related pollution exposure, we used the CALINE4 line-source air-quality dispersion model, separately for freeways and non-freeway roads.[1] The main model inputs included roadway geometry, traffic volumes, meteorological conditions (wind speed and direction, atmospheric stability, and mixing heights), and vehicle emission rates. We used the CALINE4 model to predict nitrogen dioxide concentrations derived from freeways and non-freeways at each child's home. Categories of exposure were then formed on the basis of quartiles of the within-community distribution of child-specific predictions, specifically based on cutpoints 0·6, 1·9, and 7·1 parts per billion (ppb) from freeways, and 1·5, 2·6, and 5·3 ppb from non-freeway roads. We also used the CALINE4 model to predict concentrations of other traffic-related pollutants, including oxides of nitrogen, elemental carbon, and carbon monoxide. However, predictions for each of these pollutants were almost perfectly correlated (around 0·99) with predictions of nitrogen dioxide. Thus, our model-based concentrations should be viewed as general measures of traffic-related pollution rather than this pollutant specifically. For both distance and model-based traffic indicators, within-community deviations from the corresponding community mean of the indicator were used in the health models to assess local (rather than between-community) effects.

Air-pollution monitoring stations were established in each of the 12 study communities and provided continuous monitoring data from 1994 to 2003. Each station measured average hourly concentrations of ozone, nitrogen dioxide, and particulate matter with aerodynamic diameter less than 10 μm (PM_{10}). Stations also collected 2-week integrated filter samples for measuring acid vapour and $PM_{2.5}$ mass and chemistry. Acid vapour included both inorganic (nitric, hydrochloric) and organic (formic, acetic) acids. For statistical analysis, we used total acid calculated as the sum of nitric, formic, and acetic acid concentrations. Hydrochloric acid was excluded from this sum, because concentrations were very low and close to the detection limit. In addition to measurement of $PM_{2.5}$ mass, we measured concentrations of elemental carbon and organic carbon, using the NIOSH 5040 method.[2] We calculated yearly averages on the basis of 24 h (PM_{10}, nitrogen dioxide) or 2-week ($PM_{2.5}$, elemental carbon, organic carbon, acid) average concentrations. For ozone, we calculated the yearly average of the 1000–1800 h (8 h daytime) average. Long-term mean pollutant concentrations (between 1994 and 2000 for cohort 1 and 1996 and 2003 for cohort 2) were also calculated for use in the statistical analysis of the lung-function outcomes. The distribution and correlation structure of these pollutants across communities, and their effect on lung function development, have been previously reported.[3–5] In this paper, we used community-average pollutant concentrations in models of local traffic exposure to investigate their combined effects and to explore the possibility that traffic effects vary according to regional air quality.

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