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

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Summary

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

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

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

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

Lancet 2007; 369: 571–77

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

See **Comment** page 535

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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

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 (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 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 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

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