



## The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age

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### Abstract

**Background:** Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown.

**Methods:** In this prospective study, we recruited 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The rate of attrition was approximately 10 percent per year. The communities represented a wide range of ambient exposures to ozone, acid vapor, nitrogen dioxide, and particulate matter. Linear regression was used to examine the relationship of air pollution to the forced expiratory volume in one second (FEV1) and other spirometric measures.

**Results:** Over the eight-year period, deficits in the growth of FEV1 were associated with exposure to nitrogen dioxide ( $P=0.005$ ), acid vapor ( $P=0.004$ ), particulate matter with an aerodynamic diameter of less than  $2.5 \mu\text{m}$  (PM2.5) ( $P=0.04$ ), and elemental carbon ( $P=0.007$ ), even after adjustment for several potential confounders and effect modifiers. Associations were also observed for other spirometric measures. Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV1 attained at the age of 18 years. For example, the estimated proportion of 18-year-old subjects with a low FEV1 (defined as a ratio of observed to expected FEV1 of less than 80 percent) was 4.9 times as great at the highest level of exposure to PM2.5 as at the lowest level of exposure (7.9 percent vs. 1.6 percent,  $P=0.002$ ).

**Conclusions:** The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV1 as children reach adulthood.

## Diesel Exhaust and Asthma: Hypotheses and Molecular Mechanisms of Action

Environ Health Perspectives 110(suppl 1):103-112 (2002).

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### Abstract:

Several components of air pollution have been linked to asthma. In addition to the well-studied criteria air pollutants, such as nitrogen dioxide, sulfur dioxide, and ozone, diesel exhaust and diesel exhaust particles (DEPs) also appear to play a role in respiratory and allergic diseases. Diesel exhaust is composed of vapors, gases, and fine particles emitted by diesel-fueled compression-ignition engines. DEPs can act as nonspecific airway irritants at relatively high levels. At lower levels, DEPs promote release of specific cytokines, chemokines, immunoglobulins, and oxidants in the upper and lower airway. Release of these mediators of the allergic and inflammatory response initiates a cascade that can culminate in airway inflammation, mucus secretion, serum leakage into the airways, and bronchial smooth muscle contraction. DEPs also may promote the expression of the TH2 immunologic response phenotype that has been associated with asthma and allergic disease. DEPs appear to have greater immunologic effects in the presence of environmental allergens than they do alone. This immunologic evidence may help explain the epidemiologic studies indicating that children living along major trucking thoroughfares are at increased risk for asthmatic and allergic symptoms and are more likely to have objective evidence of respiratory dysfunction.

## Characterizing the Range of Children's Pollutant Exposure During School Bus Commutes

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(continued)

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**Abstract:**

To determine the range of children's exposures during their bus commutes, especially those conditions leading to high exposures, real-time and integrated measurements of pollutant concentrations were conducted inside five conventional diesel school buses, as well as a diesel bus outfitted with a particulate trap and a bus powered by natural gas. Measurements were made during 20 bus commutes on a Los Angeles Unified School District bus route from South Central Los Angeles to the west side of LA, with additional runs on a second urban route, a rural/suburban route, and to test the effect of window position. Children's school bus commutes in Los Angeles appear to expose them to significantly higher concentrations of vehicle-related pollutants than ambient air concentrations and frequently higher concentrations than those measured on roadways. Concentrations of diesel vehicle-related pollutants such as black carbon and particle-bound PAHs were significantly higher on board conventional diesel buses when windows were closed. This was due to the intrusion of the bus's own exhaust, as demonstrated through the use of a tracer gas added to each bus's exhaust. When windows were open, increased ventilation rates markedly reduced this effect, although high peak concentrations were then observed when following other diesel vehicles. On-board concentrations of vehicle-related pollutants were also significantly higher on the urban routes compared to the rural/suburban route, indicating the importance of surrounding traffic density. Other related exposure scenarios such as bus loading and unloading, and time spent waiting at bus stops, were shown to make relatively insignificant contributions to children's exposure, due to the generally lower concentrations and the short times spent at those activities compared to bus commutes. Results from this study show that minimizing commute times, using the cleanest buses for the longest routes, and reducing bus caravanning and idling time will reduce children's exposure to bus-related pollutants.

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## **Children's Exposure to Diesel Exhaust on School Buses**

Environment & Human Health, Inc. February 2002

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**Abstract:**

In the United States nearly 600,000 school buses transport 24 million students to school daily. Each year buses travel 4.3 billion miles as children take nearly 10 billion school bus rides. In Connecticut, 387,000 students ride to school each day on 6,100 buses. If rides average 30 minutes in each direction, students will spend 180 hours on buses each year. Collectively, U.S. children spend 3 billion hours on school buses.

A vast majority of U.S. school buses are powered by diesel fuel. Diesel exhaust is comprised of very fine particles of carbon and a mixture of toxic gases. Federal agencies have classified diesel exhaust as a probable human carcinogen. Benzene, an important component of the fuel and exhaust, is designated to be a known human carcinogen. Components of diesel exhaust are genotoxic, mutagenic, and can produce symptoms of allergy, including inflammation and irritation of airways. There is no known safe level of exposure to diesel exhaust for children, especially those with respiratory illness.

The Centers for Disease Control and Prevention (CDC) estimates that 4.5 million U.S. children have asthma. This figure includes nearly 44,500 school-aged children in Connecticut diagnosed with the illness. Diesel exhaust can adversely affect children with underlying respiratory illness such as asthma, bronchitis, and infections. Diesel emissions may enhance the effects of some allergens among sensitive individuals. Children's airways are not yet fully developed and have a smaller diameter than those of adults. If airways are inflamed or constricted by asthma, allergies or infections, diesel exhaust may make breathing more difficult.

Fine particulate concentrations (PM<sub>2.5</sub>) measured on buses in this study were often 5-10 times higher than average levels measured at the 13 fixed-site PM<sub>2.5</sub> monitoring stations in Connecticut. Levels of fine particles were often higher under certain circumstances: when buses were idling with windows opened, when buses ran through their routes with windows closed, when buses moved through intense traffic, and especially when buses were queued to load or unload students while idling.

This study concludes that the laws intended to control air pollution in the U.S. and Connecticut must be strengthened to protect the health of children in several important respects. First, fixed monitoring facilities do not capture the variability in air pollution experienced by children. Second, air quality indoors and within vehicles is not regulated by EPA or the State of Connecticut, while Americans spend on average between 80-90% of their time indoors. Third, tougher diesel regulations adopted by EPA last year are insufficient to protect health. Under the new provisions, they will be phased in between 2006-2010. This delay means that children may be exposed to increasing levels of diesel exhaust for nearly a decade, as truck and bus traffic are likely to continue their steady rate of increase. Fourth, Connecticut is already beyond compliance with federal air quality standards for ozone, which may exacerbate respiratory illnesses. Given the limited monitoring facilities and extended averaging periods

allowed by current law, state “compliance” with federal standards offers little assurance of sufficient health protection. Fifth, routine emissions testing for school buses is not required by federal law, and school buses are specifically exempted from testing in Connecticut. Sixth, Connecticut adopted idling regulations, limiting idling time to 3 minutes, however, few know of the restriction, and it is neither monitored nor enforced.

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## Air Pollution–Associated Changes in Lung Function among Asthmatic Children in Detroit

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### Abstract:

In a longitudinal cohort study of primary-school-age children with asthma in Detroit, Michigan, we examined relationships between lung function and ambient levels of particulate matter  $\leq 10\mu\text{m}$  and  $\leq 2.5\mu\text{m}$  in diameter (PM<sub>10</sub> and PM<sub>2.5</sub>) and ozone at varying lag intervals using generalized estimating equations. Models considered effect modification by maintenance corticosteroid (CS) use and by the presence of an upper respiratory infection (URI) as recorded in a daily diary among 86 children who participated in six 2-week seasonal assessments from winter 2001 through spring 2002. Participants were predominantly African American from families with low income, and >75% were categorized as having persistent asthma. In both single-pollutant and two-pollutant models, many regressions demonstrated associations between higher exposure to ambient pollutants and poorer lung function (increased diurnal variability and decreased lowest daily values for forced expiratory volume in 1 sec) among children using CSs but not among those not using CSs, and among children reporting URI symptoms but not among those who did not report URIs. Our findings suggest that levels of air pollutants in Detroit, which are above the current National Ambient Air Quality Standards, adversely affect lung function of susceptible asthmatic children.

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## Diesel Exhaust Inhalation Causes Vascular Dysfunction and Impaired Endogenous Fibrinolysis

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### Abstract:

**Background:** Although the mechanisms are unknown, it has been suggested that transient exposure to traffic-derived air pollution may be a trigger for acute myocardial infarction. The study aim was to investigate the effects of diesel exhaust inhalation on vascular and endothelial function in humans.

**Methods and Results:** In a double-blind, randomized, cross-over study, 30 healthy men were exposed to diluted diesel exhaust (300  $\mu\text{g}/\text{m}^3$  particulate concentration) or air for 1 hour during intermittent exercise. Bilateral forearm blood flow and inflammatory factors were measured before and during unilateral intrabrachial bradykinin (100 to 1000 pmol/min), acetylcholine (5 to 20  $\mu\text{g}/\text{min}$ ), sodium nitroprusside (2 to 8  $\mu\text{g}/\text{min}$ ), and verapamil (10 to 100  $\mu\text{g}/\text{min}$ ) infusions 2 and 6 hours after exposure. There were no differences in resting forearm blood flow or inflammatory markers after exposure to diesel exhaust or air. Although there was a dose-dependent increase in blood flow with each vasodilator ( $P < 0.0001$  for all), this response was attenuated with bradykinin ( $P < 0.05$ ), acetylcholine ( $P < 0.05$ ), and sodium nitroprusside ( $P < 0.001$ ) infusions 2 hours after exposure to diesel exhaust, which persisted at 6 hours. Bradykinin caused a dose-dependent increase in plasma tissue plasminogen activator ( $P < 0.0001$ ) that was suppressed 6 hours after exposure to diesel ( $P < 0.001$ ; area under the curve decreased by 34%).

**Conclusions:** At levels encountered in an urban environment, inhalation of dilute diesel exhaust impairs 2 important and complementary aspects of vascular function in humans: the regulation of vascular tone and endogenous fibrinolysis. These important findings provide a potential mechanism that links air pollution to the pathogenesis of atherothrombosis and acute myocardial infarction.