

# Children's Exposure to Diesel Exhaust on School Buses

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**John Wargo, Ph.D., David Brown, Sc.D.**

*Center for Children's Environmental Health, Yale University; Environmental Research Institute, University of Connecticut*

## **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 geno-toxic, 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 (PM2.5) measured on buses in this study were often 5-10 times higher than average levels measured at the 13 fixed-site PM2.5 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.

# Characterization of Fine Particle and Gaseous Emissions During School Bus Idling

Environmental Science & Technology Volume 41 Issue 14 Page 4972-9 July 2007

**Kinsey JS, Williams DC, Dong Y, Logan R.**

*United States Environmental Protection Agency, Office of Research and Development, National Risk Management Research Laboratory*

## **Abstract:**

The particulate matter (PM) and gaseous emissions from six diesel school buses were determined over a simulated waiting period typical of schools in the northeastern U.S. Testing was conducted for both continuous idle and hot restart conditions using a suite of on-line particle and gas analyzers installed in the U.S. Environmental Protection Agency's Diesel Emissions Aerosol Laboratory. The specific pollutants measured encompassed total PM-2.5 mass (PM  $\leq$  2.5 microm in aerodynamic diameter), PM-2.5 number concentration, particle size distribution, particle-surface polycyclic aromatic hydrocarbons (PAHs), and a tracer gas (1,1,1,2,3,3,3-heptafluoropropane) in the diluted sample stream. Carbon monoxide (CO), carbon dioxide, nitrogen oxides (NO(x)), total hydrocarbons (THC), oxygen, formaldehyde, and the tracer gas were also measured in the raw exhaust. Results of the study showed little difference in the measured emissions between a 10 min post-restart idle and a 10 min continuous idle with the exception of THC and formaldehyde. However, an emissions pulse was observed during engine restart. A predictive equation was developed from the experimental data, which allows a comparison between continuous idle and hot restart for NO(x), CO, PM<sub>2.5</sub>, and PAHs and which considers factors such as the restart emissions pulse and periods when the engine is not running. This equation indicates that restart is the preferred operating scenario as long as there is no extended idling after the engine is restarted.

# Traffic-related Air Pollution and the Development of Asthma and Allergies During the First 8 years of Life

American Journal of Respiratory and Critical Care Medicine Volume 181 Number 6 Page 596-603 Mar 2010

**Gehring U, Wijga AH, Brauer M, Fischer P, de Jongste JC, Kerkhof M, Oldenwening M, Smit HA, Brunekreef B.**

*Institute for Risk Assessment Sciences, Utrecht University, The Netherlands*

## **Abstract:**

**RATIONALE:** The role of air pollution exposure in the development of asthma, allergies, and related symptoms remains unclear, due in part to the limited number of prospective cohort studies with sufficiently long follow-ups addressing this problem.

**OBJECTIVES:** We studied the association between traffic-related air pollution and the development of asthma, allergy, and related symptoms in a prospective birth cohort study with a unique 8-year follow-up.

**METHODS:** Annual questionnaire reports of asthma, hay fever, and related symptoms during the first 8 years of life were analyzed for 3,863 children. At age 8, measurements of allergic sensitization and bronchial hyperresponsiveness were performed for subpopulations ( $n = 1,700$  and  $936$ , respectively). Individual exposures to nitrogen dioxide (NO<sub>2</sub>), particulate matter (PM<sub>2.5</sub>), and soot at the birth address were estimated by land-use regression models. Associations between exposure to traffic-related air pollution and repeated measures of health outcomes were assessed by repeated-measures logistic regression analysis. Effects are presented for an interquartile range increase in exposure after adjusting for covariates.

**MEASUREMENTS AND MAIN RESULTS:** Annual prevalence was 3 to 6% for asthma and 12 to 23% for asthma symptoms. Annual incidence of asthma was 6% at age 1, and 1 to 2% at later ages. PM<sub>2.5</sub> levels were associated with a significant increase in incidence of asthma (odds ratio [OR], 1.28; 95% confidence interval [CI], 1.10-1.49), prevalence of asthma (OR, 1.26; 95% CI, 1.04-1.51), and prevalence of asthma symptoms (OR, 1.15; 95% CI, 1.02-1.28). Findings were similar for NO<sub>2</sub> and soot. Associations were stronger for children who had not moved since birth. Positive associations with hay fever were found in nonmovers only. No associations were found with atopic eczema, allergic sensitization, and bronchial hyperresponsiveness.

**CONCLUSIONS:** Exposure to traffic-related air pollution may cause asthma in children.

# Traffic-related Particulate Matter and Acute Respiratory Symptoms Among New York City Area Adolescents

Environmental Health Perspectives May, 7 2010

Patel MM, Chillrud SN, Correa JC, Hazi Y, Feinberg M, Kc D, Prakash S, Ross JM, Levy D, Kinney PL.  
*Columbia University College of Physicians and Surgeons*

## Abstract:

Background: Exposure to traffic-related particulate matter has been associated with adverse respiratory health outcomes in children. Diesel exhaust particles (DEP) are a local driver of urban fine particulate matter (PM<sub>2.5</sub>); however, evidence linking ambient DEP exposure to acute respiratory symptoms is relatively sparse, and susceptibilities of urban and asthmatic children are inadequately characterized.

Objectives: To examine associations of daily ambient black carbon (BC) concentrations, a DEP indicator, with daily respiratory symptoms among asthmatic and nonasthmatic adolescents in New York City (NYC) and a nearby suburban community.

Methods: BC and PM<sub>2.5</sub> were monitored continuously outside 3 NYC high schools and 1 suburban high school for 4-6 weeks, and daily symptom data were obtained from 249 subjects (57 asthmatics, 192 nonasthmatics) using diaries. Associations between pollutants and symptoms were characterized using multilevel generalized linear mixed models, and modification by urban residence and asthma status were examined.

Results: Increases in BC were associated with increased wheeze, shortness of breath, and chest tightness. Multiple lags of nitrogen dioxide (NO<sub>2</sub>) exposure were associated with symptoms. For several symptoms, associations with BC and NO<sub>2</sub> were significantly larger in magnitude among urban subjects and asthmatics. PM<sub>2.5</sub> was not consistently associated with increases in symptoms.

Conclusions: Acute exposures to traffic-related pollutants such as DEP and/or NO<sub>2</sub> may contribute to increased respiratory morbidity among adolescents, and urban residents and asthmatics may be at increased risk. The findings provide support for developing additional strategies to reduce diesel emissions further, especially in populations susceptible because of environment or underlying respiratory disease.

## DNA Damage in Rats After a Single Oral Exposure to Diesel Exhaust Particles

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Pernille Høgh Danielsen, Lotte Risoma, Håkan Wallinb, Herman Autrupc, Ulla Vogelb, Steffen Lofta and Peter Møllera.

*Institute of Public Health, Department of Occupational and Environmental Health, University of Copenhagen, Denmark*

*National Research Centre for the Working Environment, Copenhagen, Denmark*

*Institute of Public Health, Department of Environmental Medicine, Aarhus University, Denmark*

## Abstract:

The gastrointestinal route of exposure to particulate matter is important because particles are ingested via contaminated foods and inhaled particles are swallowed when removed from the airways by the mucociliary clearance system. We investigated the effect of an intragastric administration by oral gavage of diesel exhaust particles (DEP) in terms of DNA damage, oxidative stress and DNA repair in colon epithelial cells, liver, and lung of rats. Eight rats per group were exposed to Standard Reference Material 2975 at 0.064 or 0.64 mg/kg bodyweight for 6 and 24 h. Increased levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine lesions were observed at the highest dose after 6 and 24 h in all three organs. 8-Oxo-7,8-dihydro- 2'-deoxyguanosine is repaired by oxoguanine DNA glycosylase 1 (OGG1); upregulation of this repair system was observed as elevated pulmonary OGG1 mRNA levels after 24 h at both doses of DEP, but not in the colon and liver. A general response of the antioxidant defence system is further indicated by elevated levels of heme oxygenase 1 mRNA in the liver and lung 24 h after administration. The level of bulky DNA adducts was increased in liver and lung at both doses after 6 and 24 h (DNA adducts in colon epithelium were not investigated). In summary, DEP administered via the gastrointestinal tract at low doses relative to ambient

exposure generates DNA damage and increase the expression of defence mechanisms in organs such as the lung and liver. The oral exposure route should be taken into account in risk assessment of particulate matter.

## **School Bus Pollution and Changes in the Air Quality at Schools: A Case Study**

Journal of Environmental Monitoring 11(5): 1037-42 May 2009

**Li C, Nguyen Q, Ryan PH, Lemasters GK, Spitz H, Lobaugh M, Glover S, Grinshpun SA**  
*Department of Environmental Health, University of Cincinnati*

### **Abstract:**

Millions of children attending US schools are exposed to traffic-related air pollutants, including health-relevant ultrafine aerosols generated from school buses powered with diesel fuel. This case study was established in a midwestern (USA) metropolitan area to determine the concentration and elemental composition of aerosol in the vicinity of a public school during morning hours when the bus traffic in and out of the adjacent depot was especially intense. Simultaneous measurements were performed at a control site. The ambient aerosol was first characterized in real time using a particle size selective aerosol spectrometer and then continuously monitored at each site with a real-time non-size-selective instrument that detected particles of 20 nm to >1 microm. In addition, air samples were collected with PM<sub>2.5</sub> Harvard Impactors and analyzed for elemental composition using the X-ray fluorescence technique (for 38 elements) and thermal-optical transmittance (for carbon). The measurements were conducted during two seasons: in March at ambient temperature around 0 degrees C and in May when it ranged mostly between 10 and 20 degrees C. The particle number concentration at the test site exhibited high temporal variability while it was time independent at the control site. Overall, the aerosol particle count at the school was 4.7 +/- 1.0 times (March) and 2.2 +/- 0.4 times (May) greater than at the control site. On some days, a 15 min-averaged particle number concentration showed significant correlation with the number of school bus arrivals and departures during these time intervals. On other days, the correlation was less than statistically significant. The 3 h time-averaged particle concentrations determined in the test site on days when the school buses operated were found to be more than two-fold greater (on average) than those measured on bus-free days at the same location, and this difference was statistically significant. Overall, the data suggest a possible association between the number of detected aerosol particles and the school bus traffic intensity. Analysis of the filter samples collected at the school site between 6:00 and 9:00 AM revealed higher concentrations of elemental carbon as compared to the control site (2.8 +/- 0.9 times in March and 3.1 +/- 1.1 times in May). The data collected in this case study suggest that school buses significantly contribute to exposure of children to aerosol pollutants (including diesel exhaust particles) in the school.

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

New England Journal of Medicine 2004;351(11):1057

**W. James Gauderman, Ph.D., Edward Avol, M.S., Frank Gilliland, M.D., Ph.D., Hita Vora, M.S., Duncan Thomas, Ph.D., Kiros Berhane, Ph.D., Rob McConnell, M.D., Nino Kuenzli M.D., Fred Lurmann, M.S., Edward Rappaport, M.S., Helene Margolis, Ph.D., David Bates, M.D., and John Peters, M.D.**

*From the Department of Preventive Medicine, University of Southern California, Los Angeles; Sonoma Technology, Petaluma, Calif.; Air Resources Board, State of California, Sacramento; and the University of British Columbia, Vancouver, B.C., Canada.*

### **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 (FEF1) 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.

## **Health Effects of Real-world Exposure to Diesel Exhaust in Persons With Asthma**

Respiratory Report Health Effects Institute (138)5-109 Pages 111-23 February 2009

**Zhang JJ, McCreanor JE, Cullinan P, Chung KF, Ohman-Strickland P, Han IK, Järup L, Nieuwenhuijsen MJ**

*University of Medicine and Dentistry of New Jersey-School of Public Health*

### Abstract:

Many people, including people with asthma, experience short-term exposure to diesel exhaust (DE\*) during daily activities. The health effects of such exposures, however, remain poorly understood. The present study utilized a real-world setting to examine whether short-term DE exposure would (1) worsen asthma symptoms, (2) augment airway inflammation, or (3) increase oxidative stress burdens. The study also examined exposure-response relations for several DE components and the contribution of background asthma severity to individuals' respiratory responses to DE exposure. Sixty people participated in the study; 31 had mild asthma and 29 had moderate asthma. Each participant completed an exposure and a control session. Some effects on lung function were statistically significant. Compared with control sessions, forced expiratory volume in the first second (FEV1) was reduced 3.0% to 4.1%, and forced vital capacity (FVC) was reduced 2.8% to 3.7% in the 5 hours immediately after the exposure sessions. The changes in lung function indices (FEV1, FVC, and forced expiratory flow during the middle half of the FVC [FEF25-75]) were most consistently associated with UFP and EC exposures, whereas the changes in EBC pH were most consistently associated with NO2 exposure. However, our findings cannot be taken as demonstrating a causal association with any measured pollutant, because the measured pollutant concentrations may simply represent the entire roadside diesel-traffic exposure that comprises not only the pollutants measured in this study but also other pollutants in the complex DE mixture and resuspended coarse particles from road dust, engine debris, and tire debris. The effects of exposure appeared to be larger in the more severe asthmatic group for most outcomes measured. In conclusion, short-term exposure to urban roadside diesel traffic led to consistent and significant reductions in lung function, accompanied by airway acidification and neutrophilic inflammation. Our findings help to explain the epidemiologic evidence on diesel traffic health effects in persons with asthma.