# Linking Emissions to Admissions

## $PM_{2.5}$ and Respiratory Health

It may seem self-evident that living close to a busy street with its many automotive pollutants can have adverse effects on respiratory health, but documenting such a relationship with sufficient rigor to be considered scientifically valid has proven to be an elusive undertaking due to the many seemingly incompatible variables involved. In this month's issue, Canadian researchers from the University of Toronto led by David Buckeridge (now affiliated with Stanford University) report the results of their study of the relationship between proximity to vehicle emissions and respiratory health [*EHP* **110:293–300**]. Their unique approach of using a geographic information system (GIS) to model that relationship could serve as a basis for future studies, and adds a convincing piece of evidence suggesting that chronic exposure to urban air can cause respiratory disease.

**Car exhaust really *is* bad to breathe.** A new study provides hard evidence of a relationship between exposure to vehicle emissions and adverse effects on respiratory health.

The authors chose to study exposure to a single pollutant emitted by vehicles: particulate matter smaller than 2.5 microns ($PM_{2.5}$), which had been linked to detrimental respiratory effects in previous research. They examined the correlation between exposure to $PM_{2.5}$ emissions and respiratory hospitalization while controlling for socioeconomic status. Their study area was the socioeconomically diverse southeastern section of Toronto, which had been divided into 334 "enumeration areas" during the 1991 Canadian census. The Toronto enumeration areas, each the geographic area covered by one census canvasser, had a median population of 400.

By incorporating a wide range of data, including population information, traffic volume, vehicle type mix, and distances of residences from streets, the scientists were able to develop a refined GIS exposure model to estimate the average daily exposure to $PM_{2.5}$ in each enumeration area. The GIS model allowed them to account for the critical fact that exposure drops off dramatically with increasing distance from the street, falling by about half within 10 meters of the roadway.

They used statistical analysis to cross-reference the exposure estimates with hospitalization rates for three diagnostic groups—all respiratory conditions, a subset of respiratory conditions known to be related to $PM_{2.5}$ exposure (asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, and upper respiratory tract infection), and genitourinary conditions, a type of condition chosen as a control because the authors assumed they would not be associated with exposure to vehicle emissions.

The data showed no correlation between socioeconomic status and residential proximity to busy streets (and thus higher $PM_{2.5}$ exposure). The researchers did find that exposure to $PM_{2.5}$ at the enumeration area level had a significant positive correlation with hospital admission rates for the subset of respiratory diagnoses. They also found a weaker correlation between $PM_{2.5}$ exposure and hospitalization for all respiratory conditions, and no such correlation with hospitalization for genitourinary conditions.

While the use of hospital admission rates is a valid measure of health effects, the authors point out that these rates probably give a conservative estimate of the health impact of exposure compared with other markers such as disease prevalence estimates or self-reported health status data. Given that most of the respiratory conditions they found to be associated with $PM_{2.5}$ exposure are typically chronic and often ambulatory in nature, and thus do not necessarily involve admission to the hospital, it seems likely that the link may be even stronger than that documented in this study.

The study results are limited by factors such as traffic flow fluctuations (such as rush hour), indoor air quality, meteorologic conditions, or exposure to other pollutants present in automobile exhaust, and that exposure data did not account for individual activity (such as times that people were at home to be exposed). The authors suggest that future research include these potential confounders, along with other refinements in exposure modeling and analysis. **–Ernie Hood**

# The Paths of Chlorpyrifos

## Quantifying Aggregate Exposures

Chlorpyrifos, once one of the most widely used pesticides in the United States, was banned from home and garden use in June 2000 after federally mandated risk assessments concluded that children are more sensitive to the pesticide than previously estimated. Under the Food Quality Protection Act, U.S. Environmental Protection Agency pesticide exposure risk assessments must now use aggregate exposure estimates that account for all exposure routes, including inhalation, ingestion, and dermal absorption. But comprehensive measurements of how one person is exposed via all three routes are rare; few studies have combined direct pesticide concentration measurements from different sources with exposure estimates. In this issue, Yaohong Pang of the University of Georgia at Athens and colleagues present results from just such a study on residential chlorpyrifos exposures—the National Human Exposure Assessment Survey (NHEXAS) in Maryland [*EHP* **110:235–240**]. During NHEXAS–Maryland, researchers were able to quantify aggregate chlorpyrifos exposure by multiple routes among a sample population as well as uncover the surprising dominance of one particular route of exposure.

In 1995–1996, Pang and the other NHEXAS–Maryland researchers measured chlorpyrifos concentrations in indoor air, carpet dust, exterior soil, and diet samples from 80 people over age 10 who lived in Baltimore and the surrounding counties. None of the

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participants used pesticides for six months before or during the study. The team combined the measured chlorpyrifos concentrations in each medium with self-reported time spent indoors at home, time and frequency in contact with carpet, frequency of contact with soil, and amounts of pesticide in the diet samples to derive the exposure to chlorpyrifos for each medium as well as the average daily aggregate exposure.

They found that aggregate daily exposures for chlorpyrifos ranged from 13.5 ng/day to 12,821.0 ng/day, with a mean daily aggregate exposure of 1,390.0 ng/day. Inhalation of indoor air accounted for 76.1% of the aggregate exposure to the population, while solid food intake contributed 22.8% of the population exposure. The importance of the inhalation pathway was somewhat surprising to the authors because chlorpyrifos is not very volatile. They also point out that little is known about how much chlorpyrifos is actually absorbed through inhalational exposure.

The distribution of chlorpyrifos concentrations in each medium varied by over three orders of magnitude. This variation and the concentrations for indoor air, carpet dust, and soil measured in this study were in the same range as those measured in earlier comparable studies. The aggregate exposure estimates determined in this study are lower than some previous exposure estimates, according to the authors. But some of the previous estimates reflected exposures following pesticide applications and those for young children, whose crawling and mouthing behaviors could lead to higher exposures.

The study indicates that a single short-term measurement of exposure may not yield an accurate estimate of an individual's long-term exposure, but that knowing the relationship between short-term exposure measurements and long-term exposure could be used to improve the efficiency of future epidemiologic study designs. The study also showed that multiple environmental media are important contributors to aggregate exposure, so epidemiologists should account for both dietary and nondietary exposure in their assessments. Finally, better quantification of aggregate exposure to pesticides will help environmental health scientists to evaluate the utility of biological markers of exposure for future epidemiologic studies. **–Rebecca Renner**

# How Earplugs Can Help Your Heart

## Health Effects of Noise Pollution

Noise exposure can lead to small increases in blood pressure readings and possibly even increases in cardiovascular disease prevalence, conclude researchers from the National Institute of Public Health and the Environment in Bilthoven, the Netherlands, after filtering their way through more than 500 studies of health effects potentially linked with noise exposure [***EHP* 110:307–317**]. But the limited number of health end points investigated and the lack of consistent methodologies have left large gaps in our knowledge of noise impacts, says lead researcher Elise E. M. M. van Kempen.

Noise is suspected of causing a number of adverse physiologic effects, ranging from hearing loss to myocardial infarction. Studies conducted to date suggest there is little risk of permanent hearing damage from noise below 70 decibels, which is the level typically found in a car or office setting. A jackhammer operates at about 100 decibels, and a jet taking off generates about 130 decibels. Other health end points studied have included elevated blood pressure, angina pectoris, ischemic heart disease, use of antihypertensive or cardiovascular drugs, and consultation with a doctor (for unspecified reasons).

Of the 500-plus studies the team reviewed, 43 contained data adequate for inclusion in a meta-analysis. The studies were conducted around the world from 1970 to 1999, covered a variety of occupational and community settings, and contained study populations ranging in size from 46 to 35,150. Studies in occupational settings tended to cover higher noise levels (up to 116 decibels) that were actually measured, while studies in community settings tended to cover lower levels (up to 80 decibels) that were estimated, not measured.

Based on results from nine pertinent studies, the team found a small but significant link between occupational noise exposure and hypertension. Exposure to air traffic noise in community settings also showed a small but significant link with hypertension, but only one study covered that scenario. A few other studies in community settings showed statistically insignificant links between road traffic noise exposure and myocardial infarction and ischemic heart disease, and between air traffic noise exposure and angina pectoris, the use of cardiovascular drugs, and consultation with a doctor.

To gain a better understanding of potential health effects from exposure to higher noise levels, which tens of millions of people routinely encounter, van Kempen and her colleagues recommend that researchers undertake additional studies that address the many shortcomings identified in existing studies. For instance, most studies did not investigate confounding factors such as an individual's weight or smoking and alcohol consumption habits. Nor did researchers look at other pollutants associated with particular occupational and community settings. Noise levels often were simply estimated or, if measured, did not necessarily reflect the exposure of a particular individual, but rather that of the immediate area around the noise detector. Blood pressure measurements often were based on just one reading. Long-term health effects were poorly evaluated. And studies that were conducted but not published, which the team concludes has been a likely occurrence, skew the data base by reducing its size and altering the weight given to certain findings. For instance, the team concludes that studies that found a relatively small effect from noise exposure have been published less often than might be expected. **–Bob Weinhold**

**The sound of sickness?** Loud noise can raise blood pressure and may lead to even greater heart problems.

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