When Good Buses Go Bad

Diesel Exhaust Linked to Neighborhood Air Pollution

Mass transit can help reduce air pollution if it replaces enough cars. But not everyone reaps the rewards, as a Harvard-led team discovered after studying diesel emissions in Boston's Roxbury neighborhood [*EHP* 109:341–347]. Jonathan Levy and colleagues from the Harvard School of Public Health collaborated with a local advocacy organization called Alternatives for Community and Environment to begin characterizing pollution patterns in the Dudley Square area using a novel combination of methods and resources. This study lays the groundwork for future epidemiologic assessments of traffic-related respiratory symptoms.

The Dudley Square terminal is one of the busiest Massachusetts Bay Transportation Authority bus stops in Boston. The neighborhood surrounding this bus terminal sees numerous diesel-powered vehicles, with 1,150 buses and trucks garaged within a 1.5 mile radius of Dudley Square (including half of the Massachusetts Bay Transportation Authority bus fleet). Some neighborhood residents suspect the horde of diesel-powered vehicles may be contributing to the community's high asthma hospitalization rate, which is nearly five times the state average (9.8 hospitalizations per 1,000 people versus 2.1 per 1,000 people). The current study was intended as a community-based pilot project to gain insight into whether the vehicles may be contributing significantly to air pollution exposures experienced by Roxbury residents.

The team used three portable monitors to test a 1-mile-radius area around the terminal for two important diesel combustion byproducts—fine particulates and particle-bound polycyclic aromatic hydrocarbons (PAHs). To cover the neighborhood effectively, the researchers sent trained high school students out on sorties with the portable monitors to measure particulates and PAHs at 1-minute intervals during the morning hours of July and August 1999. The readings varied widely, from 0.3 to 340.0 nanograms per cubic meter for PAHs and from 2.0 to 347.0 micrograms per cubic meter for particulates less than 2.5 micrometers in diameter.

Mass asthma authority? Residents living around Roxbury's Dudley Square station suspect diesel-powered buses may be part of the reason for the neighborhood's unusually high incidence of asthma.

While taking their readings, the students documented their position every five minutes so the data could be plugged into maps developed through a geographic information system. They also tracked temperature and humidity, which might be expected to affect pollutant concentrations. The team then used statistical methods and data from fixed-site PAH monitors in the neighborhood to evaluate spatial and temporal patterns of air pollution in the neighborhood data.

The team found temporal patterns in air pollution (higher levels during rush hour and on weekdays) that suggested a link with transportation sources. Through statistical models, the team found a significant link between higher PAH concentrations and proximity to the bus terminal. They also found slightly—though not significantly—higher concentrations of PAHs and particulates along bus routes and high-traffic roads in the neighborhood. Higher humidity and temperature each coincided with higher particulate concentrations throughout the neighborhood, and higher humidity coincided with higher PAH concentrations.

Levy cautions that the study was not designed to investigate links with specific health problems in the neighborhood, and the researchers can't develop any conclusions from this study about specific health risks, including the high asthma rates in the neighborhood, because they did not collect pertinent health data. It also was not a comprehensive study, given budget constraints, and the lack of a comprehensive monitoring network made it difficult to disentangle spatial and temporal effects. Nevertheless, Levy says, the study was able to gather key exposure information on Roxbury residents and provides a template for more comprehensive analysis. **–Bob Weinhold**

Red Flag for Fish

Brevetoxin Induces More—And Worse—Effects

In the Gulf of Mexico, population explosions of the marine dinoflagellate *Gymnodinium breve* are responsible for chronic, persistent "red tides," so named for the reddish-brown color the blooms give the affected waters. *G. breve* produces brevetoxins, potent neurotoxins that kill finfish and accumulate in filter-feeding animals such as shellfish. Because brevetoxins are fat-soluble and bioaccumulate in organisms, adverse effects might also be expected to occur in the eggs and developing embryos of fish and higher vertebrates. So researchers Karen L. Kimm-Brinson and John S. Ramsdell of the Center for Coastal Environmental Health and Biomolecular Research in Charleston, South Carolina, decided to study the toxins' developmental effects in aquatic species [*EHP* 109:377–381]. They microinjected eggs of medaka fish with one of the most common and potent brevetoxins, PbTx-1, and witnessed significant developmental abnormalities that impaired both hatching success and survival of medaka larvae.

Double trouble. *G. breve* toxins affect not only the larval life stages of fish but also their ability to spawn successfully.

Left to right: Richard Howard; Karen L. Kimm-Brinson & John S. Ramsdell

The researchers designed their approach to reproduce the transfer of contaminant from mother fish to egg. Fertilized eggs collected from laboratory-bred females were microinjected with varying amounts of PbTx-1 reconstituted in a droplet of triolein fish oil. The egg microinjection method had already been used to characterize the adverse effects of known bioaccumulants such as ciguatoxin (a related polyether toxin also produced by a dinoflagellate) and the pesticide DDT in medaka fish. But comparable data for brevetoxin had not been reported until this study.

The results in this study were striking, say the researchers. The lowest observable effect on embryos occurred at a PbTx-1 dose concentration between 0.1 and 0.9 nanogram (ng) per egg—equal to 0.1–0.9 parts per million (ppm)—which induced excessive tail and body twitching in the early stages of development. The range of 0.1–3.0 ng/egg caused curvature of the spine, which became more pronounced with increased dose concentration. The most deformed of those embryos hatched out in an abnormal, head-first fashion and could not swim well enough to survive. Doses between 3.1 and 4.0 ng/egg produced malpositioned eyes and hernias in the brains and brain membranes of embryos. Eggs injected with concentrations of PbTx-1 above 4.0 ng failed to hatch.

Blooms of *G. breve* are toxic to fish at a concentration as low as 200,000 cells per liter, which produces approximately 5 ppm brevetoxin. At this 5 ppm concentration, Kimm-Brinson and Ramsdell concluded that abnormal developments similar to those in the lab experiment might occur if the animal had a bioaccumulation and egg-transfer factor of one-fifth the toxin in the water. This one-fifth bioaccumulation and egg transfer factor is well within the range found for other fat-soluble contaminants.

The results identify a new spectrum of adverse effects from red tides, which often persist from late fall to early spring, the breeding season of many Gulf fish species. Although adverse effects on larval life stages had been suspected, this new research suggests that the spawning success of fish also may be compromised. Further, sublethal exposure to brevetoxins throughout the life of many marine organisms may cause developmental toxicity to manifest in subsequent generations as with DDT, polychlorinated biphenyls, and other contaminants that accumulate in fatty tissues. Because the developmental processes of lower and higher vertebrates are similar, the potential exists for similar cumulative risks to marine mammals and even to humans. **–Carla Burgess**

Pb Affects BP

Closing In on Contributing Factors

Among the negative health effects of lead absorption are demonstrated increases in blood pressure and the risk of developing chronic high blood pressure, or hypertension. In this issue, a collaboration between Korean and American researchers provides a more detailed picture of this relationship as well as new evidence that variations in the gene for the vitamin D receptor (VDR), which is known to modify the toxicokinetics of lead, influence blood pressure [*EHP* 109:383–389].

Byung-Kook Lee of Soonchunhyang University in Korea, Brian S. Schwartz of the Johns Hopkins School of Hygiene and Public Health, and their colleagues examined Korean workers who worked at facilities where lead was used. The researchers assessed lead absorption in the workers by measuring lead in blood, the tibia, and soft tissues. They then correlated these concentrations with measures of blood pressure and hypertension. In addition, they looked at the influence of two obvious gene candidates, those for the VDR and those for the enzyme δ-aminolevulinic acid dehydratase (ALAD). Both genes have been shown to modify the toxicokinetics of lead—that is, its absorption, distribution, metabolism, and excretion. For example, ALAD genotype is known to influence blood lead concentrations, and VDR genotype is known to influence lead concentrations in blood, bone, and soft tissues. Each gene is also polymorphic, meaning that several variants (polymorphisms) of the gene exist in a population. Possession of a particular polymorphism could alter the way a person's body responds to absorbed lead.

The researchers explored this relationship in 798 lead workers and 135 control subjects. For all three measures of lead, higher concentrations were associated with higher systolic blood pressure. There were no associations between the lead biomarkers and diastolic blood pressure, but bone lead was a predictor of hypertension status. This last finding is particularly important, say the authors, because it suggests that lead has a chronic, cumulative influence on hypertension risk.

An entirely new finding was that variations in VDR genotype affected all of the blood pressure measures. Lee and colleagues examined the *Bsm*I polymorphism of the VDR, which has three genotypes—*bb*, *Bb*, and *BB*. Workers who carried the *B* allele of the *Bsm*I polymorphism (in either the *BB* or *Bb* combination) had higher systolic blood pressure, diastolic blood pressure, and prevalence of hypertension than did workers who did not carry the *B* gene allele (that is, those with the *bb* combination). In fact, the risk of hypertension was doubled among subjects with the VDR *B* allele. The researchers also found that the *B* variant modified the effects of age on systolic blood pressure: workers with the *BB* or *Bb* genotype had greater increases in blood pressure with increasing age. By contrast, variations in the ALAD gene produced no noticeable differences in blood pressure measures.

The authors suggest that if lead influences blood pressure by directly interacting with the proteins coded for by the VDR gene, the different genotypes would have produced different effects on the measured relationships between lead concentrations and blood pressure. Since this was not the case, it is likely that lead and VDR genotype each have an independent influence on blood pressure. Exactly how variations in the VDR gene affect blood pressure is not clear, but the authors point out that the VDR plays a critical role in intestinal calcium regulation and that there is a demonstrated interrelationship between calcium, lead, and blood pressure. One possibility is that the VDR could influence the absorption of calcium and lead from the gastrointestinal tract or the release of calcium and lead from bones. **–Hakon Heimer**

PhotoDisc

Off the cuff. Scientists already knew that lead absorption is linked with high blood pressure. New research now points toward genetic factors that may influence whether a lead-exposed person will develop hypertension.