A Pollution Model Upgrade?  
Incorporating the Local Scale in National Models

Networks of fixed-site air monitors form the backbone of current efforts to predict health effects of air pollution. But there aren’t enough monitors on the ground to accurately account for local variations in ambient concentrations of air pollutants, even for those pollutants considered of most concern in many countries, such as fine particulate matter (PM_{2.5}). Experts have long struggled to develop better predictive models so epidemiologic studies can be made more accurate and affordable, and so health surveillance, policies, and regulations can be made more effective. A team of Canadian and U.S. researchers says it has made some incremental advances in this predictive science [EHP 119(8):1123–1129; Hystad et al.]. The researchers are using the results of their new study as part of the larger Carex Canada project to estimate cancer risks associated with known and suspected environmental and occupational carcinogens.

The researchers assembled readily available data from the National Air Pollution Surveillance monitoring network in Canada and devised models to predict ambient concentrations of five pollutants at a national scale while capturing within-city pollution variability. The five models generated results similar to those from models typically used for regional or city areas. They were able to predict 73% of the variability in readings taken at nitrogen dioxide (NO_{2}) monitors, 68% of the variability for 1,3-butadiene, 67% of that for ethylbenzene, 62% of that for benzene, and 46% of that for PM_{2.5}.

The NO_{2} and benzene model predictions were compared against predictions generated independently for seven cities using land-use regression (LUR) models keyed to numerous monitors in each city. The NO_{2} model predicted 43% of the variability in readings predicted by LUR models, compared with 18% using the common technique of simply interpolating between monitors using factors such as distance from the monitor, weather patterns, and known pollution sources. The within-city benzene model captured only 16% of variability predicted by LUR models, compared with 11% using interpolation.

Predictions from the five models were influenced most strongly by satellite data for PM_{2.5} and NO_{2}, vehicle emissions (represented by road type and location), industry emissions (from large point sources), and population density. Other factors in the current models included land use type, small industry point sources, railroad length, elevation, temperature, precipitation, and generalized predictive gradients representing dispersion from specific sources.

Other input might substantially improve the models if it were readily available and covered appropriate time intervals and geographic areas. Such input could include more monitoring data for these and other toxics, more detailed dispersion predictions, and emissions data for area sources such as wildfires and wood burning.

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All Diesel Exhaust Is Not the Same  
Engine Load Alters Toxicity

After decades of research on the adverse health effects of diesel engine exhaust (DEE), important information gaps still remain. Among these gaps are differences in the composition of DEE created when an engine operates under different load conditions (i.e., how hard the engine is working) as well as the health effects associated with various exhaust compositions. Some studies have evaluated exhaust products and health effects under one load condition or another, or with varying operating conditions during the same engine run cycle. But none have evaluated different load conditions in the same study, then looked at health effects caused by the separate exhaust products, according to the authors of a study that begins to address that knowledge gap [EHP 119(8):1136–1144; McDonald et al.].

The team analyzed the composition of DEE generated from a single-cylinder diesel generator operating under full (100%) or partial (55%) load. They also evaluated several health end points in two strains of mice that inhaled the two resulting exhaust products, which varied substantially in composition.

The particulate matter (PM) concentrations in the exhaust products were similar to those found in certain occupational and high ambient outdoor settings. Under partial load, the PM constituent had higher organic carbon, ammonium, sulfate, and nitrate mass, lower elemental (black) carbon mass, and smaller particle size, compared with full-load exhaust. Vapor phase partial-load exhaust had a greater mass of carbon monoxide and nonmethane volatile organic compounds, a higher percentage of naphthalenes, and a lower percentage of alkanes.

Male C57B1/6 mice exposed to full-load exhaust had significantly more lung inflammation, as indicated through heme oxygenase-1 expression, compared with mice exposed to partial-load exhaust. These mice also demonstrated greater susceptibility to viral lung inflammation and epithelial damage and were much slower in clearing the virus from their lungs. However, mice exposed to partial-load exhaust had lower levels of two other inflammation indicators, interferon-γ and tumor necrosis factor-α, than either control mice or those exposed to full-load exhaust.

In male ApoE−/− mice, one measure of cardiovascular toxicity—reduction in heart rate—occurred significantly faster following exposure to partial-load exhaust compared with full-load exposure. Partial-load exhaust also was linked with a rapid increase in T-wave area, another indicator of cardiovascular toxicity, which was not affected by exposure to full-load exhaust.

The researchers conclude that the typical practice of evaluating DEE health effects based solely on the PM mass concentration of the exhaust is misleading. Instead, they recommend researchers carefully analyze and describe the DEE compositions they use in their studies so different bodies of work can be more accurately compared.

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