Salt Mist Is the Right Seasoning for Ozone

Shipping ports face a newly discovered air pollution problem—the production of the ozone precursor nitryl chloride. Nitryl chloride was detected for the first time in the lowest part of the Earth’s atmosphere by a team from the National Oceanic and Atmospheric Administration (NOAA) that was monitoring air quality in Galveston Bay to understand why nearby Houston, Texas, has one of the worst air pollution problems in the nation. Salts in ocean mists were thought to be relatively inert until the connection to ozone was uncovered. “People never before thought that nitryl chloride was important,” says James Roberts, a NOAA research chemist and the team’s coordinator.

In the summer of 2006, the researchers used chemical ionization mass spectrometry to detect trace levels of airborne chemicals, including nitryl chloride. They found that when ship exhaust plumes rich in nitrogen oxides (NOₓ) meet ocean air at night, unexpectedly high levels of nitryl chloride form due to the NOₓ species dinitrogen pentoxide combining with chloride in sea mist. After the sun comes up, this buildup of photoactive nitryl chloride splits into chlorine atoms and nitrogen dioxide. These compounds then accelerate the production of ozone, a key component of smog.

The amount of nitryl chloride measured by Roberts and colleagues—as high as 650 ppt by volume, or about 15% of total reactive nitrogen species from ship exhaust—is much greater than that estimated by standard air pollution models, which have taken into account neither the heightened presence of nitryl chloride around ports nor its importance in forming ozone. “This preliminary study indicates that nitryl chloride chemistry could make a significant contribution—up to ten to thirty percent—to ozone production during the morning hours in Houston,” says Roberts. These results were published in the May 2008 issue of Nature Geoscience.

The study findings point to the need to control NOₓ emissions from fossil fuel combustion in coastal cities, says Roberts. He adds that one solution may be to require docked ships to use local electrical power, rather than burning diesel fuel, to generate electricity. Some ports have begun to pursue this course of action [see “Ports in a Storm,” EHP 114:A222–A231 (2006)]. The 10-year $750-million Middle Harbor Redevelopment Project proposed by the Port of Long Beach (California) would, among other pollution mitigation strategies, provide shoreside electricity for docked vessels.

About half the world’s population lives near coastlines where industrial pollution meets ocean air, Roberts says, so nitryl chloride could play a major role in air quality worldwide. The same reaction likely occurs inland, where chloride-containing aerosols drive the chemical reaction. Inland sources of chloride include natural soil salts such as calcium chloride and de-icing compounds spread on winter roads.

“We don’t know how widespread nitryl chloride is as a source of ozone pollution,” says Roberts. The health consequences of nitryl chloride itself are unknown. As for ozone, an April 2008 report by the National Research Council, Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution, links even short-term exposure with premature death.

“This is the first field measurement of nitryl chloride, and it’s very exciting,” says Barbara Finlayson-Pitts, a professor of chemistry at the University of California, Irvine. Experiments in her laboratory 20 years ago first showed that mixing sodium chloride with dinitrogen pentoxide in the dark generated nitryl chloride. “The data will be extremely useful for further development and application of air quality models for coastal urban areas,” she says.

—Carol Potera
Canyons Up the Pollution Ante

New York City and other megacities with populations of 10 million or more are grappling with air pollution associated with traffic congestion, which has been linked with numerous health problems. As part of a larger research effort to understand the costs and benefits of various strategies to reduce traffic congestion in the Big Apple, a new study focuses on how traffic emissions are dispersed within urban street canyons—streets that are lined with tall buildings on both sides.

Within these domains, large quantities of pollutants are released near the ground from motor vehicle exhaust, then trapped and concentrated within the canyon walls. Urban street canyons also tend to contain a lot of people, potentially making these areas high-risk zones for big cities.

“The combination of high population density and high traffic volume in urban areas such as New York City means that the health impact of traffic pollution can potentially be much larger than similar sources in less populated areas,” says Ying Zhou, a research fellow at the Harvard School of Public Health who coauthored a report in the April 2008 issue of *Atmospheric Environment* on urban street canyon modeling in Manhattan. “Street canyons can exacerbate the health impact of traffic pollutants, hence the need to understand their dispersion dynamics.”

Along with colleague Jonathan Levy, an associate professor of environmental health and risk assessment at the Harvard School of Public Health, Zhou has been working on a project to examine how proposed congestion pricing policies in New York City will affect air pollution. Several other cities, including London, Stockholm, Singapore, and three cities in Norway, have implemented congestion pricing systems that charge a fee for vehicles to enter a certain zone in the city, ostensibly to curb traffic in those zones.

“We sought to understand the degree of exposure and public health benefits of control strategies in urban street canyons similar to those in Manhattan, given the hypothesis that the same amount of emissions would have a greater population exposure in a street canyon setting,” says Levy. He and Zhou report that population exposure to traffic pollutants in New York’s urban street canyons can be up to 1,000 times higher than exposure to a similar amount of emissions in other urban settings. Additionally, pedestrians and daytime office workers received most of the population exposure in the midtown Manhattan area, emphasizing the importance of considering nonresidential exposures. In contrast, most studies to date have focused on residential exposures to roadway pollution.

The study focused primarily on the “intake fraction,” a measure of the total population exposure per unit of emissions. “The intake fraction is essentially a function of what individuals are exposed to and how many individuals are exposed,” says Levy. “For example, even though cyclists may be breathing harder than pedestrians, there are many more pedestrians than cyclists passing through urban street canyons in Manhattan, and [pedestrians] move more slowly through the canyon.” Emissions controls in a given street canyon therefore would likely have a larger total health impact on pedestrians than on cyclists, he notes.

The authors speculate that the benefits of pollution controls targeted at urban environments probably have been underestimated because cost–benefit analyses of such controls have not considered either the spatial correlation between high population density and high source density or the dynamic effects of street canyons on pollutant dispersion. The benefits depend on the particular street configuration (with taller buildings and narrower streets amplifying exposures) and on the sizes of various subpopulations (who are affected differentially depending on where they are situated in the street canyon and at what time of day). The benefits per unit emissions are also slightly sensitive to traffic volume, but in an unexpected direction—having more traffic actually decreased the population exposure per unit of emissions (separate from its effect on total emissions) by increasing atmospheric turbulence and pollutant dispersion.

“This study adds to the growing literature on intake fractions for air pollutants and emphasizes that there may be settings that involve relatively small amounts of emissions but contribute substantially to population exposures,” says John Evans, a senior lecturer on environmental science at the Harvard School of Public Health. “Zhou and Levy point out that in urban street canyons mobile source intake fractions may be two to three orders of magnitude larger than in suburban and rural areas. With the rapid growth of megacities, urban mobile source emissions become increasingly important determinants of human exposures and health risks. Zhou and Levy’s study makes clear the need for greater emphasis on this issue.” —M. Nathaniel Mead
Not Immune to PFOS Effects?

Two recent studies have demonstrated that exposure to perfluorooctanoyl sulfonate (PFOS) can alter mammalian immune system function. In one study, PFOS induced immunotoxicity in test animals at exposures analogous to those reported in the U.S. population, raising the possibility that “if humans and mice share similarities in their immune systems, then people today could be immunocompromised due to current PFOS exposure,” says Jennifer Keller, a research biologist at the National Institute of Standards and Technology’s Hollings Marine Laboratory. However, resolving this question will require carefully controlled epidemiologic studies that include relevant immune system and infectious disease end points.

PFOS was removed from 3M’s Scotchgard™ stain repellent by the end of 2002, but the compound degrades slowly and is dispersed widely throughout the environment. Data from the National Health and Nutrition Examination Survey 2003–2004 documented that it was found in 99.9% of samples.

Research slated for publication in the 1 July 2008 issue of Toxicological Sciences documents that production of the immunoglobulin M (IgM) antibody decreased in B6C3F1 mice exposed to PFOS. This suggests the immune response to pathogens may be suppressed and could result in increased disease susceptibility, says lead author Margie Peden-Adams, a research assistant professor at the Medical University of South Carolina. She says these effects occurred at serum PFOS levels (91.5 ppb in male mice) that were 14 times lower than the average blood concentrations of occupationally exposed humans and in the upper range of concentrations reported for the general population, making this the lowest reported lowest-observable-effect level for PFOS. She adds that suppression of IgM production in female mice occurred at dose concentrations 10 times higher than in males.

The male mice’s natural killer (NK) cell population, an important part of the innate immune system for tumor and viral surveillance, was increased, but female NK cell function was not affected. Peden-Adams speculates that the increase could be a compensation for a decrease in the male mice’s populations of cytotoxic T cells, which also have functions related to killing tumor cells. Additional testing determined that the cause of the decreased IgM production is probably an issue with B cells, the cell type that differentiates into antibody-producing cells, she says.

Another study, published 1 May 2008 in Toxicological Sciences by Deborah Keil, an associate professor of clinical laboratory sciences at the University of Nevada at Las Vegas, also linked a reduction in IgM antibody response to PFOS exposure. This study, which evaluated the immune systems of B6C3F1 mouse pups whose mothers were exposed to the compound during pregnancy, showed that NK cell function was decreased in male pups. Both studies document that PFOS targeted antibody production and that males appeared more sensitive than females to the effects of PFOS, Keil says.

Peden-Adams says her group was quite surprised by their findings and that further research is needed to determine the effects’ mode of action in order to determine human risk. Her paper posits that sex differences in sensitivity may be related to levels of estrogen and testosterone, because these steroids are known to affect immune system function. “We know that PFOS does have some endocrine-disrupting activity,” she says, adding that some research suggests the chemical may be antiestrogenic.

A third study using a different mouse strain found similar effects for perfluorooctanoic acid (PFOA). The study, published in the May 2008 issue of EHP by research biologist Robert Luebke of the U.S. Environmental Protection Agency National Health and Environmental Effects Research Laboratory, showed that adult female C57BL/6J mice exposed to higher concentrations of PFOA also exhibited a reduction in IgM production. —Kellyn S. Betts
BUILT ENVIRONMENT

Turn Up the Heat for Respiratory Health

In the dog days of summer it’s hard to think about turning up the thermostat, but keep one thing in mind as the cooler months approach: people with chronic obstructive pulmonary disease (COPD) whose houses are cold in cold weather are more likely to suffer poorer respiratory health than those whose houses are warmer, according to a study published online 26 March 2008 ahead of print in the European Journal of Public Health. Turning up the heat might therefore lead to more benefits than just warm feet.

“This is the first time a direct relationship has been found between the number of hours a house is warm and respiratory health status—in this case that of patients with COPD,” explains first author Liesl Osman, a research development advisor with the Nuffield Department of Clinical Medicine, Oxford University. “And it would seem that this relationship is most marked for smokers.”

The researchers monitored the temperature of the living rooms and bedrooms of 148 COPD patients in Aberdeen, Scotland, for 1 week between October and May (all of which are cold months in Scotland). They tracked how often each living room was kept at a temperature of at least 21°C (69.8°F)—which the U.K. government recommends be maintained for at least 9 hours per day for maximal energy efficiency—and how long each bedroom maintained a temperature of at least 18°C (64.4°F). The subjects also completed questionnaires to determine their respiratory and general health status.

Overall, people who lived in homes in which the living room temperature was more typically at least 21°C for at least 9 hours per day had significantly better respiratory health (that is, fewer exacerbations of their underlying COPD or respiratory infections) than subjects who lived in homes where this temperature was maintained less often.

“Unfortunately,” Osman adds, “we also saw that over fifty percent of the households involved in this study were colder than recommended.” No association was seen between respiratory health scores and bedroom temperatures.

In a post hoc analysis, the protective effect of warmth was statistically significant for smokers but did not reach significance for nonsmokers. “Prolonged cold weakens our resistance to infection,” explains Osman. “Perhaps this is accentuated in smokers with COPD, resulting in overall poorer respiratory health.”

Philippa Howden-Chapman, director of the Housing and Health Research Programme at the University of Otago in Wellington, New Zealand, also points out that cold homes could be the cause of poorer health in people with other underlying diseases. “[We have] shown that increasing warmth leads to improvements in health for most respiratory diseases, and our recently completed Housing, Heating and Health Study found warmer temperatures improved the respiratory symptoms of children with asthma,” she says. The literature also suggests that coronary conditions are affected by indoor temperatures, particularly in temperate countries.

“The key question that needs to be answered is whether improvements in home heating will actually bring out the benefits to health [that may be] predicted using data from this cross-sectional study,” remarks Anna Hansell, a clinical fellow at Imperial College London. Howden-Chapman agrees: “This is a cross-sectional study, so causal conclusions really shouldn’t be drawn from it, though the results are very interesting. The next step would be to [look at] daily measures to see whether short-term exposure to cold influences COPD symptoms. This would also help determine the precision of [these] long-term measures.”

The United Kingdom has social programs to help people with low incomes or poorer housing increase the energy efficiency of their homes, which can help conserve heat. Given today’s sky-high oil and gas prices, the results of this study could mean such schemes are more needed than ever. –Adrian Burton

CA Nail Salon Workers Surveyed

Most of the 35,000 nail salons in California employ Asian immigrants who are routinely exposed to potentially harmful compounds in nail care products—some of which are unregulated—including carcinogens and endocrine disruptors. In a study published online 14 May 2008 ahead of print in the Journal of Community Health, investigators collected preliminary descriptive data from Vietnamese nail salon workers in Alameda County to help inform future targeted health interventions and reduce occupational exposure. A majority of the workers expressed concern about chemical exposures in the workplace, and many reported acute symptoms such as skin and eye irritation, breathing difficulties, and headaches associated with solvent exposure. The authors note that although nail care products are thought to contain low levels of harmful compounds, their prevalence in salons means exposure is likely continuous, and the chronic effects are unknown. The U.S. EPA and partner agencies are reaching out to salon workers, many of whom do not speak English. The 2007 report Protecting the Health of Nail Salon Workers, which offers guidance on minimizing exposures, is available in English, Vietnamese, and Korean.

Quick Hit to the Vascular System

Although governments around the world are banning smoking in public areas in response to warnings of scientists about the harmful health effects of secondhand smoke (SHS), many people are still exposed daily to SHS, even if only for brief periods. A new study published in the 6 May 2008 issue of the Journal of the American College of Cardiology suggests that exposure to SHS in real-world settings for as little as 30 minutes can adversely affect the body’s vascular system, even in young and otherwise healthy lifelong nonsmokers. The harm was twofold: SHS exposure not only injured blood vessels but also impaired the ability of endothelial progenitor cells to repair the damaged vessels. The study further showed that the harmful response to SHS exposure persisted for at least 24 hours, much longer than previously believed.

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