Nitrogen oxides in polluted air

Environmental chemists have launched an intensive effort to monitor nitrogen oxides in polluted air and to determine their health effects on the human lung. And, although a cynic might wonder what took them so long and what good such studies are going to do now, the early results — presented at the recent meeting in Anaheim, Calif., of the American Chemical Society — are sobering, and certain to have at least some impact on future air quality standards.

Oxides of nitrogen are given off during all combustion processes, and indications are that the ambient levels of nitrogen oxides (NO_x) have increased markedly over urban areas. One form, nitrogen dioxide (NO_2), is responsible for both the sting and the yellowish-brown color of polluted air.

In order to determine how much NO_2 and other nitrogen oxides human activities have added to the air, researchers from the National Center for Atmospheric Research measured naturally occurring background levels of the chemicals. John Ritter, Donald Stedman and Thomas Kelley measured NO_x levels in rural Michigan and over a mountain top in Colorado with extremely sensitive equipment. Their results are dismaying: They found less than 0.25 parts per billion in the clean air, punctuated by high concentration surges from man-made sources. In other words, there is virtually no background level at all in clean air, and the existing nitrogen oxides are very likely man-made.

Another NCAR team, Barry J. Huebert and Allan Lazrus, measured nitric acid (HNO_3) and particulate nitrate over several remote areas, and found the levels in clean air to be about 0.1 parts per billion, or ten times lower than previously assumed.

Since NO_2 levels, for example during a smog alert, are often 4,000 to 8,000 times higher than "clean air background" figures, the immediate question becomes one of effect — are NO_2 levels of one or two parts per million damaging?

To help answer this, a research group at the Univerity of California at Irvine designed a clever system for growing human lung cells. Ronald E. Rasmussen, T. Timothy Crocker and G. Scott Samuelson place a single-celled layer of lung tissue on a porous membrane bathed continuously with nutrient broth. This technique more nearly recreates the moist, mucus-bound environment of normal lung tissue than did previous experimental set-ups, which allowed lung cells to dry out during NO_2 exposure.

More than 90 percent of the control cells (those cultured in the continuous-flow apparatus but exposed only to clean air) survived for eight hours. By contrast, more than 90 percent of the experimental cells died after eight hours of exposure to 0.12 parts per million of NO_2, a relatively common urban level of the pollutant.

This test is by no means an absolute measure of cell death in a healthy human lung. Like most experimental cells, these lung cells were derived from a line of cancerous cells, and their malignancy may or may not affect response to the pollutants. Also, by arranging them in a single, flattened layer, each cell was exposed to proportionately more NO_2 than are cells on the convoluted internal surfaces of the lung. But, says Rasmussen, industrialists and health researchers have "very different beliefs" regarding the effect of pollutants, and suggestive evidence such as this is needed to justify pollution control programs.

Another California study, this one on the intact lung itself, showed less dramatic effects. Male human volunteers were exposed for eight hour periods to NO_2 levels similar to those during a typical smog alert (1 or 2 ppm). Their exposure chambers were heated to simulate summer temperatures, and the subjects exercised periodically on stationary bicycles. James Hackney of the University of Southern California School of Medicine and the Environmental Health Service at Rancho Los Amigos Hospital at Downey found no clear evidence that such ambient levels were harmful for these short exposure periods. He observed slight changes in certain aspects of red blood cell biochemistry, but no significant change in lung function in the 16 volunteers.

This study did not, however, test the more relevant matter of long-term exposures or exposure effects on very young, old or ill subjects.

Despite the current paucity of meaningful data on human health effects of NO_x pollutants, mouse studies by Daniel Menzel and colleagues at the Duke University Medical Center in Durham, N.C., revealed mechanisms of lung damage and lung protection. Mice exposed to high levels (about 13 ppm) of NO_2 daily for three months showed significant pulmonary edema, which indicates tissue damage and decreased capacity to absorb oxygen. Menzel postulates that NO_2 prevents the proper production and metabolism of prostaglandins, hormones that, in the lungs, help regulate oxygen absorption and fluid balance. But by feeding the laboratory mice high levels of vitamin E, lung edema was prevented.

The specific damage caused by long-term low levels of NO_2 on the human lung remains to be explained, as well as the precise mechanism of protection by vitamin E. But it is perhaps significant that Menzel himself, who lives in a polluted urban area, is convinced enough of the protective phenomenon to take 200 units of vitamin E daily. □

Lung damage from other air pollutants

Nitrogen oxides can hurt the lungs, at least in experimental, high-level situations (see preceding article). But how about other common air pollutants, such as sulfur oxides and suspended particles? Research conducted in Japan and reported in the March 11 LANCET suggests that these chemicals can harm lungs too — in fact, even under long-term, real-life, moderately low-level conditions.

Koichi Nobutomo of Kyushu University in Fukuoka, Japan, launched a study in 1972 of 308 persons living in the industrial districts of Omuta City, Fukuoka, where coal mining and the metal and chemical industries are well established, and of 399 persons living in West Fukuoka, a residential area. None of the subjects suffered symptoms of chronic bronchitis or other respiratory ailments. (Residents who did had been excluded from the study.) Nor did any of the subjects work in occupations where they would come into contact with excessive air pollution. By these means well subjects living in two different geographic areas could be compared for the earliest possible effects of air pollution on their lungs.

Air pollution levels for the two geographic areas of interest were then obtained from air pollution monitoring stations in the areas. Readings showed that levels of sulfur oxides and suspended particles were several times higher in Omuta than in West Fukuoka. However, there was no significant difference between the two regions for nitrogen oxides. Air pollution levels for both regions were typical of those in many industrialized countries.

Finally, each subject was instructed that on a particular day he or she should cough sputum into a cup an hour after rising. Sputum samples from the subjects were collected and examined under a microscope by three independent observers to see whether the samples contained cells indicative of lung inflammation, notably phagocytes, neutrophils and lymphocytes. Sputum cell findings for the two groups of subjects were then compared, taking into account subjects' ages, sex and tobacco consumption differences.

Generally, those subjects living in the more polluted city of Omuta had more inflamed cells in their sputum than did subjects living in the less polluted West Fukuoka region. Thus, sulfur oxides and suspended particles appear capable of inflaming human lungs under long-term, moderate-level, real-life conditions.

What's more, the fact that none of the subjects had bronchitis or respiratory symptoms, but that many had lung inflammation, suggests that sputum analysis may provide a sensitive first warning of air pollution damage to lungs. □