Summer smog: Not just an urban problem

Last summer, New York City's air exceeded the federal 1-hour smog-ozone standard of 120 parts per billion on 42 occasions, or an average of about once every two days. Washington's summer air violated the standard an average of once every three days. Frequent ozone excesses also hit most other large U.S. cities. And this year, the National Weather Service's latest forecast envisions above-normal temperatures — the type that can cook up high levels of ozone, the primary irritant in smog — from July to October for one-third of the nation.

If that makes you long for a country stroll to breathe clean, fresh air, think again. A new study shows that unhealthy ozone levels plague rural air, too — at least in the eastern United States.

Though people have traditionally viewed ozone as an urban problem, Jennifer A. Logan, an atmospheric chemist at Harvard University, focused her analysis on data collected from 18 rural stations recording 24-hour ozone readings in 1978 and 1979. She sought to characterize not only the high-ozone trends but also the atmospheric processes that may have fostered them.

In the East, she found that high-ozone periods lasting up to three days tended to occur simultaneously or within a day or two of each other at sites hundreds of kilometers apart. Most such episodes appeared linked to the passage of large, slow-moving and persistent high-pressure weather systems, Logan reports in the June 20 JOURNAL OF GEOPHYSICAL RESEARCH.

These high-pressure systems tend to maintain a low-altitude boundary area — a well-mixed region of air extending about 2 kilometers from Earth's surface. The boundary area, explains Logan, "acts like a low lid," preventing pollutant dilution by restricting low-altitude air from mixing with the upper atmosphere. This gives hydrocarbons and nitrogen oxides entrained within it an especially good chance to interact with each other in ways that generate ozone, she says.

Logan's data show that peak ozone concentrations in rural areas rivaled or exceeded those reported in many urban areas. For instance, ozone peaks at the eastern sites topped 80 parts per billion (ppb) on 26 percent of the days between May and August in 1979 and on 39 percent of those days in 1978. Moreover, brief peaks commonly exceeded 120 ppb. This is well above the 40- to 60-ppb ozone level that can cause significant crop-yield losses. While all stations recorded similar median values — 30 to 40 ppb — from April to September of both years, the three western stations (in Arizona, Montana and Oregon) almost never exceeded 80 ppb. Logan suspects this difference stems primarily from the West's lower levels of hydrocarbons and nitrogen oxides.

Her data correlating acute ozone episodes with high-pressure areas hold open the prospect that weather forecasters might one day give farmers a couple days' warning before plant-choking ozone conditions arrive. Farmers would need such warnings to use chemical protectants now being developed to save plants from ozone damage, notes Joseph Miller at the Agriculture Department's air-quality program in Raleigh, N.C. His preliminary data indicate that the leading candidate protectant, best known as EDU, "was detrimental to plant growth" under all but extreme ozone concentrations.

Air-quality consultant Allen S. Lefohn of Helena, Mont., thinks Logan's most important contributions may be two observations that call into question the credibility of previous agricultural data. Logan observed that the 7-hour ozone-exposure period used in most crop studies — 9 a.m. to 4 p.m. — differs from the period of highest rural ozone levels, which ranges from midafternoon to near midnight. Moreover, she found that the practice of assessing ozone in terms of a 7- or even 12-hour seasonal average masks the brief peaks that may occur every few days and that could pose a far greater threat to crops. — *J. Raloff*

Pygmy paradox prompts a short answer

African Pygmies and some other diminutive individuals may be short on specialized cell receptors that interact with a hormone essential for normal growth, two new studies suggest. The reports offer tantalizing clues to the cause of this type of dwarfism. If confirmed, they may point the way to new forms of treatment for certain kinds of very short stature.

African Pygmies have long puzzled scientists studying growth disorders because these people have normal amounts of growth hormone but rarely grow taller than 4 feet 10 inches. U.S. physicians see a similar disorder in children who have normal growth-hormone levels yet lag far behind their peers in height. Two separate research teams now present evidence hinting that the trouble lies with the receptor that provides a docking site for human growth hormone circulating in the bloodstream.

Gerhard Baumann and Melissa A. Shaw of the Northwestern University Medical School in Chicago and Thomas J. Merimee of the University of Florida in Gainesville studied blood samples from 20 Pygmies living in Zaire's Ituri Forest. Because it is difficult to count the cell receptors directly, Baumann's group developed a method that detects complex molecules consisting of growth hormone attached to a binding protein, which is probably a fragment of a cell receptor that has broken off during the docking process, Merimee says. The research team found that Pygmies, compared with controls, have half the amount of this protein-hormone complex in their blood plasma. Control subjects in the study, reported in the June 29 NEW ENGLAND JOURNAL OF MEDICINE, were five non-Pygmy black Africans and seven U.S. whites, all of normal height.

The low levels of the protein-hormone complex suggest Pygmies have a shortage of growth-hormone receptors, Merimee says. If so, Pygmies would have trouble responding to their normal levels of growth hormone because many of the molecules couldn't find docking sites on the cell. That theory fits well with the observation that Pygmies grow slowly during childhood and show seriously stunted growth as teenagers, when a full supply of receptors is most crucial. Scientists have speculated that teenagers normally produce extra growth-hormone receptors during this time of rapid growth.

Some U.S. children appear to have the same disorder, scientists reported June 23 at the annual meeting of the Endocrine Society in Seattle. Jane L. Lynch and James R. Gavin III of the University of Oklahoma Health Sciences Center in Oklahoma City studied 26 white children who were more than two standard deviations shorter than their peers. A subset of these children had normal growth-hormone levels but showed "striking" deficits in the protein-hormone complex that Baumann's team measured, Gavin says. Like their African counterparts, such children reach adult heights well under 5 feet. These preliminary findings suggest the children have a receptor deficiency that prevents the growth hormone from doing its job, the Oklahoma researchers conclude.

Scientists are just beginning to unravel the complicated defects involved in such disorders. They have not ruled out the possibility that affected individuals have normal numbers of receptors that are flawed. In either case, both studies suggest that the cause of the growth disorder lies in the genes. Circumstantial evidence points to a genetic defect causing faulty production of growth-hormone receptors, Baumann says.

Gavin thinks the new findings should prompt researchers to pursue new therapies aimed at bypassing receptor problems. For example, scientists are beginning to consider clinical trials that would give patients a growth-promoting substance normally formed in cells after growth hormone interacts with the receptor. — *K.A. Fackelmann*