Air of suspicion in Morgantown

Particles from air samples taken during a recent five-month period in Morgantown, W. Va., have been shown to cause chromosomal changes in bacteria, a federal public health agency reported last week. This finding of mutagenic activity—usually taken as the first indication that a substance has the potential to cause cancer—in the air of a rural area calls into question the concept of nonpolluted areas as safer than industrialized regions and suggests the necessity of routine monitoring of air samples for similar health effects.

The findings, which are still under investigation, were the result of a bit of serendipity. Tong-man Ong and co-workers from the National Institute for Occupational Safety and Health (NIOSH) in Morgantown, while developing a method of testing for mutagenicity in the air of the workplace, decided last June to use local air samples as a baseline. Their first sample, however, which was taken from the roof of their laboratory, produced five to seven times the number of chromosomal changes in bacteria that a control sample did. (Their method consists of pumping air through a filter at a constant rate and for a specific period of time, dissolving the particles collected on the filter and using the standard Ames test—exposing a bacterial culture to the solution—to test for mutagenicity.) Additional samples taken from a school, two residential areas and a business area during the next five months showed similar results. NIOSH officials, who are responsible only for health matters in the workplace, had been keeping state and county health departments informed of the findings, and on Oct. 31 released a memo describing their results. The health agencies will manage further investigations.

While not currently viewed by health officials as a hazard, the results are surprising because the 30,000-population town is considered unpolluted. Located about 60 miles south of Pittsburgh, the nearest industrial center, Morgantown has only a glass factory, two small, university-run coal-burning power plants and a larger plant outside the town. (Ironically, hearings are to be held Nov. 10 to discuss building a coke plant and a synfuels plant in the area; the recent findings may have a negative effect, say some observers.) This situation, say NIOSH officials, suggests that the source may be long-range transport of pollutants — the export via prevailing winds of pollution from other areas. If "pollution exporting" — considered a major factor in the cause of acid rain—is also the cause of mutagenic air in a "clean" region, it may strengthen current efforts to change the Clean Air Act to deal with the phenomenon, says a spokesman for the regional office in Philadelphia of the Environmental Protection Agency.

Moreover, the findings raise the possibility that such results may be more common than previously thought. Similar testing of air in cities such as Chicago, Houston and New York has shown mutagenic activity, says Ong. But such tests in those areas are not routine, he says, and have never been conducted in an area not suspected of heavy pollution. And because of differences in techniques, adds West Virginia health director George Pickett, the results of tests in urban areas cannot readily be compared with the Morgantown results. "I strongly suspect that if you ran the Ames test in other places you'd find the same thing, but I don't know," says Carl Beard of the West Virginia Air Pollution Control Commission. "Nationally, these are questions that need to be answered."

As for health effects, "that's the $64 million one," says Pickett. "I'm not alarmed. As a scientist and an epidemiologist, my curiosity is very high right now. I'm not scared, but I may be later if we find something." It is possible, he suggests, that similar or even higher levels of mutagenic activity might have been found in the area had such tests been conducted routinely during the past 20 years. He points out, however, that although there is a high correlation between a positive Ames test in bacteria and chromosomal changes in mammals, very little is known about the relationship between health and long-term exposure to pollutants. "The next step, if we find something," he says, "is to test it in a mammalian system."

In order to start pinning down some answers, Pickett's agency, with the county health department and the Air Pollution Control Commission, has asked NIOSH to "fingerprint" by electron microscopy both the original air samples and samples taken from the local power plants. Comparison of trace metals and other constituents in the samples may determine the source of mutagenic activity. In addition, says Pickett, NIOSH will test air samples collected by an existing state-wide network operated by the Air Pollution Control Commission. In this way, he says, any common pattern or revealing trend may be detected. To double-check the method used by NIOSH, says spokesman George Bochanski, EPA scientists are examining the data and have asked NIOSH to use their technique on known samples taken from other parts of the region. □

Sodium-excreting hormone found

Demonstrating that there is more than one way to discover a hormone, Kenneth A. Gruber, Janice M. Whitaker and Vardaman M. Buckalew of the Bowman Gray School of Medicine in North Carolina report in the Oct. 23 NATURE the isolation of a substance with potent effects on both the cardiovascular system and the kidneys. The factor, which inhibits the enzyme (Na^+ and K^+) ATPase, plays an active role in salt excretion. It is similar in structure and effect to digoxin, a powerful drug used to stimulate the heart, and may play an important role in hypertension.

Hormones are customarily found by grinding up large amounts of tissue believed to produce the substance in question, running the puree through separation procedure, measuring the different fractions for biological activity, pooling active fractions and then cycling the product through more separation procedures until it is pure enough for identification.

In this case, instead of starting with tissue, the researchers exploited the notion that pharmacology can imitate life, and hunted for a natural correlate of digoxin. They injected the drug into goats, isolated the resultant antibody, used this antibody to capture the digoxin-like substance in the plasma of dogs and measured the presence of the antibody-hormone complex. When the dogs were fed large amounts of salt, higher concentrations of the hormone were found, indicating a positive role in salt excretion.

The researchers named their find endoxin, for endogenous digoxin. A similar procedure has been used by other researchers to isolate a morphine-like compound and a Valium-like compound.

The newly discovered hormone is evidently excreted by the hypothalamus and plays an important role in hypertension. A year and a half ago, the Bowman Gray researchers and a group from the University of Iowa found that if they destroyed the endoxin-excreting area of rats' brains and fed the rats a lot of salt, the animals failed to excrete the salt yet did not develop hypertension as expected. "This was the first evidence that salt itself is not the cause of hypertension. Salt may play a role, but there's a missing factor," says Gruber.

The researchers expect that the discovery of the hormone will aid in monitoring digoxin therapy. Since too much of the drug can be lethal, doses are carefully checked against blood concentration. But present monitoring techniques are not terribly accurate. Gruber believes this inaccuracy is due to mistakenly measuring the natural hormone and assuming it is digoxin. "Endoxin has three times the activity," explains Gruber. "If you assume what is measured is digoxin, you may be giving a toxic dose."

The hormone makes rats hypersensitive to hypertension-inducing agents, says Gruber. Its presence, recently documented in humans, may eventually prove of use in predicting which people are most likely to develop stress-induced hypertension, the researchers believe. □

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