Industries Tally Air Pollution Poorly

Companies in the United States that annually spew 100 tons or more of smog-forming volatile organic chemicals (VOCs) must provide the federal government with a quantitative breakdown of those emissions. These inventories—best estimates of releases from various processes, operations, and plumbing—often fall somewhere between fantasy and fraud, a new study reports.

Most policy makers "assume these numbers are gospel" and use them to fine-tune smog-control strategies, observes Ronald C. Henry of the University of Southern California in Los Angeles, who led the study. The faulty inventories can hurt public and polluter alike, he argues, if they lead to costly process- or pollution-control changes that provide little smog relief.

The state of Texas employed Henry to help home in on the sources and conditions most responsible for Houston's continuing smog-ozone problem—the nation's second worst. He applied a sophisticated mathematical formula to the data collected over 6 months by an automated air-sampling monitor. This device provides hourly parts-per-billion readings of more than 50 different industrial smog-fostering VOCs in Houston air.

By correlating the hourly VOC readings with wind direction and speed in this leading petrochemical hub, Henry could project not only where many VOCs originated but also "how many [different industrial] sources there must have been and what the composition of their emissions was." This approach works, he explains, because each source tends to emit its own signature mix of VOCs over time, though quantities may vary. In a test in Atlanta, the technique successfully apportioned VOCs to their largely vehicular sources.

By comparing the chemical signatures detected and the reports from each Texas firm upstream of the monitor, Henry's team found that, with a few notable exceptions, industrial inventories appear very "inaccurate" in terms of location, composition, and emission rates. The researchers describe their findings in the June 24 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES.

For instance, the second largest oil refinery in Texas is located a few miles southeast of the monitor. While the refinery's own inventory tagged it as a large VOC source, Henry notes that "we didn't see anything—even though that refinery was running full tilt." On the other hand, his team found a potent source of ethylbenzene, a compound often emitted by refineries, although no ethylbenzene sources were reported upwind.

Air pollution analysts "have worried for many years about the suspected large inaccuracies in these emissions inventories," observes Charles W. Lewis of the Environmental Protection Agency in Research Triangle Park, N.C.

Until now, however, analysts have lacked evidence to challenge those inventories. Henry says that his new study finally provides "the smoking gun."

While he can't rule out some fraud, Henry suspects that most inaccuracies were unintentional. After all, he notes, companies were as likely to err in ways that made themselves look bad as in ways that improved their image.

Indeed, notes Steve Bromberg of EPA, assembling an emission inventory "is still really more of an art than a science." Ironically, most firms consider the inventories "scut work" for junior engineers, notes Jim Price of the Texas Natural Resource Conservation Commission in Austin.

The increasing use of VOC inventory data in computer models that simulate air quality "presents some of the most severe requirements on the accuracy of these inventories," points out Glen Cass of the California Institute of Technology in Pasadena. Indeed, he notes, some firms may simply report the allowed, rather than the actual, emissions. Studies like Henry's could spur moves to improve models' accuracy, Cass believes, "by raising questions that can be answered by direct emissions measurements."

Indeed, Lewis says, "we've got to get people to take these [inventories] more seriously and begin making more actual measurements rather than trying to calculate them on paper." —*J. Raloff*

Parkinson's disease gene mutation found

After announcing last November that they were closing in on a mutant gene that causes Parkinson's disease (SN: 11/30/96, p. 348), scientists have captured their quarry. Unexpectedly, the normal form of the gene encodes a protein implicated in Alzheimer's disease, another neurodegenerative disorder.

While only a small percentage of the millions of people worldwide with Parkinson's disease may have the mutant gene, researchers hope its discovery will provide a clue to the origins of the remaining cases.

"This is a great step forward for Parkinson's disease research that will really unlock the disorder's pathogenesis in the same way that finding [Alzheimer's] mutations unlocked the pathogenesis of that disease," predicts John Hardy of the Mayo Clinic in Jacksonville, Fla.

Parkinson's disease results from the gradual death of brain cells, particularly those that produce the neurotransmitter dopamine. Such cells often control movement, which explains why tremors are a typical symptom of the disease.

Scientists have had few clues to what causes Parkinson's disease, although the brain cells of patients contain mysterious lumps called Lewy bodies. Some investigators believe Lewy bodies trigger cell death, while others suspect they are a by-product of that destruction.

In one Italian and three Greek families plagued by Parkinson's disease, researchers have now traced the origin of the illness to a mutation in the gene for a protein called alpha-synuclein. Mihael H. Polymeropoulos of the National Human Genome Research Institute in Bethesda, Md., and his colleagues report their finding in the June 27 SCIENCE.

"The next step is to figure out what the normal function of alpha-synuclein is and whether it aggregates in the Lewy bodies," says study coauthor Lawrence I. Golbe of the Robert Wood Johnson Medical School in Piscataway, N.J. Alpha-synuclein may play a role in synapses, connections that allow brain cells to communicate with one another.

The researchers suggest that mutant forms of alpha-synuclein may aggregate abnormally and thus kill cells or cause other proteins to do so. If so, Parkinson's disease could resemble Alzheimer's, which many investigators now attribute to the accumulation in the brain of a protein fragment called beta-amyloid.

"There are all these nebulous connections [between the two diseases]," says Hardy. For example, relatives of people with Alzheimer's have a higher than average risk of Parkinson's and vice versa. Moreover, the brains of Alzheimer's patients often exhibit Lewy bodies. Finally, researchers discovered several years ago that alpha-synuclein is a major component of the amyloid plaques that develop in Alzheimer's disease.

Investigators note that current therapies for Parkinson's disease address only symptoms. The finding of a mutant gene now "sets the stage to develop research ideas aimed at the cause and not just the symptoms," says Polymeropoulos. —*J. Travis*