Mapping the benefits of acid-rain controls

On Oct. 1, the National Acid Precipitation Assessment Program (NAPAP) will enter the ranks of history, winding up its 10-year, $535 million study of acid rain. NAPAP issued its swan song, a three-volume "integrated assessment" of the program's findings, at a technical briefing last week in Washington, D.C. The document concludes that acidic pollutants have changed the landscape in certain especially vulnerable regions, and predicts where and when future benefits might accrue from tightened controls on fossil-fuel emissions.

It reveals, for example, that an estimated 14 percent of Adirondack lakes are acidic, as are 12 to 14 percent of the streams in the Mid-Atlantic states – acid rain's two aquatic hotspots. About 75 percent of the low-pH Adirondack lakes and 50 percent of low-pH Mid-Atlantic streams have acidified since preindustrial times, primarily due to sulfates from fossil-fuel combustion, says Robert S. Turner of Oak Ridge (Tenn.) National Laboratory, who summarized aquatic findings at the briefing. In contrast, he notes, Florida's many low-pH lakes and streams come by their acidity naturally.

The integrated assessment concludes that without new controls on sulfur dioxide (SO_2), which converts to sulfates in the atmosphere, another 4 percent of Adirondack lakes will acidify before the region's waters begin to recover. But the assessment indicates that few if any additional lakes in the region will suffer if the nation halves its SO_2 emissions, Turner says. That change corresponds to the 10-million-ton annual SO_2 reduction called for in Clean Air Act amendments pending in Congress.

NAPAP projects that existing controls would restore most Adirondack lakes to conditions suitable for fish by around 2030 – but that reducing annual SO_2 emissions by 8 million to 12 million tons could speed that recovery by 20 years.

Mid-Atlantic waterways are more sensitive to acid deposition because area soils have nearly reached their acid-neutralizing capacity. Indeed, NAPAP data indicate that a 50 percent reduction in SO_2 emissions would just maintain the status quo in that region.

Moreover, the assessment reports results of new supercomputer simulations using NAPAP's Regional Acid Deposition Model (RADM), indicating that cutting SO_2 emissions by 50 percent would not yield a commensurate decrease in acid deposition. These simulations identify a distinct "nonlinearity" between emissions and acid rain, explains Robin L. Dennis of the National Oceanic and Atmospheric Administration in Research Triangle Park, N.C.

RADM simulations of the effects of hourly weather changes on air chemistry indicate that "deposition reductions will be less than emission reductions" over most of the East, Dennis says.

Some acid rain analysts find fault with the integrated assessment. Although NAPAP produced "a lot of good work," the final document ignores or downplays some of its more significant findings, says James J. MacKenzie of the World Resources Institute in Washington, D.C.

Deborah A. Sheiman of the Natural Resources Defense Council in Washington, D.C., speaks more critically. "We knew in 1980 what they're telling us now: What causes acid rain, how to control it, what are the effects," she says. By unnecessarily "studying the problem to death," she charges, NAPAP "became a convenient excuse for political inaction."

Such criticism is like saying, "We landed on the moon and found it's still there," counters Christopher Bernabo, NAPAP's first director and now a consultant in Washington, D.C. "Getting there is quite a lot different than knowing it's there," he argues. And along the way, NAPAP advanced the science of measuring acid rain, monitoring its effects and forecasting its damage, Bernabo says.

Prior to NAPAP's landmark National Surface Water Survey, "not only did we not know how many acid [water] systems there were, we didn't even know how many lakes were out there," adds NAPAP Deputy Director John L. Malanchuk. Now, he says, many scientists believe this inventory "will revolutionize the way we do synoptic surveys."

Researchers "are already using RADM for climate-change applications on a regional scale," Malanchuk says, and the EPA will probably use the model to gauge effects of pollution controls.

Bernabo thinks NAPAP's most lasting legacy, however, will prove organizational. The focus required for the program, he says, not only taught government agencies how to collaborate effectively but also showed them how "to make the transition from curiosity-driven research to policy-driven research." – *J. Raloff*

Diabetes marker pegged as brain enzyme

For nearly a decade, diabetes researchers have studied a protein known as 64K, produced in tiny amounts by the insulin-secreting beta cells of the pancreas. Antibodies to 64K circulate in the blood of about 80 percent of people who later develop insulin-dependent (Type I) diabetes or who already have the disease, which appears to involve a misguided immune-system attack on beta cells (SN: 6/18/88, p.389).

But attempts to use the presence of 64K antibodies as a widespread screening test for Type I diabetes have failed because of difficulties in identifying and purifying the relatively rare pancreatic protein they target.

Now, scientists report that 64K appears identical to an abundant and easily isolated enzyme of the central nervous system, known as glutamic acid decarboxylase, or GAD. Since other researchers already have cloned one form of the enzyme, the finding may speed efforts to develop an inexpensive screening test for Type I diabetes. Michele Solimena of Yale University adds that the work may also provide new information about the causes of the disease, which affects up to 1 million people in the United States.

Solimena, with colleagues from Yale and the University of Milan in Italy, proposed an association between GAD and 64K in the May 31 NEW ENGLAND JOURNAL OF MEDICINE. They noted that GAD, which helps synthesize the neurotransmitter GABA, comes under antibody attack in a rare autoimmune disease known as stiff-man syndrome. A significant number of the estimated 300 people with the muscle-stiffening disorder also suffer Type I diabetes. Moreover, 64K antibodies in diabetics without stiff-man syndrome show similarities to those targeting GAD, they found.

Now, in the Sept. 13 NATURE, Solimena and co-workers from Yale and the University of California, San Francisco, report that the two compounds exhibit identical chemical behavior, indicating they are one and the same.

Solimena notes that antibodies against the full GAD molecule cannot directly cause diabetes because GAD lies inside cells, away from immune-system components that might be activated to kill beta cells. He speculates that GAD fragments may migrate to the surface of beta cells to trigger such wholesale destruction. He adds that 64K's identification as a neuronal compound does not indicate that Type I diabetics are prone to neurological disorders – in part because pancreatic antibodies would have to cross the blood-brain barrier to cause such damage.

But the discovery does emphasize the similarity between nerve and endocrine cells, researchers note. At the European Congress of Cell Biology, held last week in Florence, Pietro De Camilli of Yale and his colleagues reviewed their findings that beta cells and other hormone-secreting cells possess storage compartments similar to those used by some neurons to hold neurotransmitters. They say this suggests beta cells may secrete GABA, possibly to help regulate blood sugar. – *R. Cowen*