New Acid Rain Threat Identified

Acid rain's primary threat to aquatic life has generally been seen as its ability to lower the pH of water. But a study released this week by the New York City-based Environmental Defense Fund (EDF) highlights what its authors contend is an equally deadly but ignored impact of acid rain — nitrate-induced suffocation and light starvation of aquatic species.

Though nitrates are a nutrient, when their levels become excessive they can cause "algal blooms" — essentially an overgrowth of algae and other phytoplankton. These plants quickly consume most of the dissolved oxygen in water, suffocating other aquatic species. Algal overgrowth also clouds the water, preventing necessary light from filtering down to plants on the seafloor.

The EDF study focused on the effects of excess nitrogen on the Chesapeake Bay — the largest U.S. estuary and a major spawning ground for East Coast fisheries from Maine to North Carolina. Until now, says Michael Oppenheimer, one of the study's authors, the Environmental Protection Agency (EPA) and others have largely ignored or greatly underestimated acid rain's role in overloading the Chesapeake and other waters with nitrogen. EDF's calculations, based on data from a range of federal and state agencies, including the EPA's Chesapeake Bay Program, indicate that acid deposition contributes at least one-quarter of the nitrogen entering the bay as a result of human activity. That makes acid deposition second only to fertilizer runoff as a source of nitrogen pollution.

Moreover, says Diane Fisher, who led the EDF study, "Our analyses show that acid rain is a significant problem in coastal waters up and down the *entire* eastern seaboard." Affecting primarily brackish and salt water, it "will continue to grow until nitrogen oxides (NO_x) emissions are controlled," she adds. In fact, efforts already underway to control nitrogen emissions (from sources other than acid rain) into many waterways "could be largely negated if NO_x emissions [from acid deposition] are not further reduced," her study says.

To counter the acid rain nitrate problem, EDF recommends that:

- states in the Chesapeake Bay watershed implement NO_x limits for electric power plants and factories and beef up motor vehicle inspection programs. (Unlike the sulfur dioxide component of acid rain, in which electric power plants contribute 70 percent of the pollution, motor vehicles are the major source of acid rain's nitrates. However, federal law prevents states from setting motor vehicle standards for NO_x.)
- the federal government adopt regulations to reduce industrial and vehicular NO_x emissions by at least 40 percent — a level that matches nitrogen reductions from other sources now being planned for the Chesapeake. (The Clean Air Act reauthorization legislation, pending in the House and Senate, would reduce allowable NO_x emissions from cars by about 60 percent.) EDF particularly endorses energy-efficiency measures to reduce NO_x.
- all governmental bodies acknowledge acid rain's contribution to the deteriorating quality of East Coast waters, and work at reducing nitrogen from sources besides acid rain — especially from sewage treatment plants and fertilizer and manure storage.

The EDF study wins high praise from William C. Baker, president of the Annapolis, Md.-based Chesapeake Bay Foundation. "The Chesapeake Bay is dying," he says, and this new report not only "brings to light an important new source of nitrogen — a major pollutant in the Chesapeake Bay" — but also points to where those involved in saving it must direct their attentions.

Though EPA spokesman Dave Cohen says the EDF scientists "seem to [offer] fairly compelling evidence regarding nitrogen loading" into the Chesapeake from acid rain, he adds that his agency's scientists were "surprised" at the size of the nitrogen contribution being attributed to this source and will therefore begin re-evaluating the data themselves. If EDF's 25 percent figure is accurate, he says, "then acid rain will be making a greater, more harmful contribution to the bay than we [at EPA] had suspected."

— J. Raloff

Linking body fat to neurons and energy

Obesity may involve disturbances of the body's autonomic nervous system that cause excessive storage of energy, scientists report this week. They expect their new finding to help undermine the attitude that putting on excessive pounds is "all in the mind," as accumulating evidence links aberrations in basic metabolism to obesity.

The latest study revealed only "weak" associations between body fat and the autonomic nervous system and thus needs to be duplicated. But knowledge that the nervous system could be directly involved will help physicians identify different types of obesity and individualize treatment, says Hugh R. Peterson of the University of Louisville in Kentucky. He says the new results complement two recent studies showing that inheriting a slow metabolism can lead to obesity (SN: 3/5/88, p.152). "We're probably looking at a metabolic phenomenon related to the autonomic system that is important [in controlling weight]," he said in an interview.

The autonomic nervous system is responsible for many of the body's unsung functions, such as temperature regulation and heart rate. It is divided into the sympathetic and parasympathetic systems, which can be distinguished by hormones and other factors. In general, sympathetic nerves respond during stress and activate energy-using responses such as utilization of stored fat, while the parasympathetic system usually conserves energy through reactions that include slowing the heart rate.

It appears that activity levels of both systems are inversely related to increasing fat in humans, says Peterson. He and other researchers from the university, the Veterans Administration Medical Center in Louisville and the National Institute of Environmental Health Sciences in Research Triangle Park, N.C., report their results in the April 28 NEW ENGLAND JOURNAL OF MEDICINE. Earlier animal studies found that decreased sympathetic activity may cause excessive fat storage while increased parasympathetic activity may prompt overeating — both leading to obesity.

But in their study of 56 healthy but overweight men, the authors found to their surprise that both systems appear to be depressed. The depression in parasympathetic activity may be the body's attempt to slow weight gain, and responsible for different weight plateaus, Peterson says. "It's likely that [the two systems] do not turn on and off as an entire unit, and that they turn on and off in different organs at different rates," he says. Because the observed decreases in activity were small, Peterson says more studies must be done to confirm the relationship — including studies during weight gain and loss, to learn whether the autonomic changes are the causes or effects of obesity.

Scientists still have only a partial picture of obesity, says Peterson. "It looks as though an explanation of human obesity in terms of psychosocial factors is inadequate," he says. "But I don't think we've reached the point where we can say those factors are not important."

— D.D. Edwards