Blood, Poison and Death: Effects of Acid Deposition on Fish

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This might be the title of a murder mystery. In fact, there isn't any mystery. The negative effects of acidification on fish are clear and unambiguous, and the ecological consequences are clearest for fish. It is simply a matter of toxicology.

First I would like to set the stage. In the eastern third of the United States, most of the values for volume-weighted annual average pH of rainwater start with a four (Figure 1). This is about ten times as acid as estimated for rainwater pH in pre-industrial times. This means that "pure" rainwater in the eastern United States is acutely lethal to all native fish species. Not even a goldfish could survive in unbuffered rainwater. This is the central issue I will speak about.

"Acid Rain" implies the deposition of acid materials in wet deposition (rain, snow, fog, cloud) as well as in the dry deposition of dust (particles) and gases; scientists use "acid deposition" for this process, because it explicitly includes dry as well as wet deposition; indeed, deposition of acid materials in dry form is often equal to the amount deposited in wet form. Acid deposition is responsible for the loss of hundreds of fish populations in Europe and North America.

The effects of acid deposition on fish involve complex processes at different spatial scales, from atmospheric transport to cell membrane transport. Nevertheless, the interaction of these processes can be summarized in five major points, as follows.

Acid deposition effects on fish.

1) The source of the acid: fossil fuels.

The burning of fossil fuels releases sulfur and nitrogen oxides into the atmosphere, where they are converted to sulfuric and nitric acids. These acidic materials may be transported long distances in the atmosphere before they are deposited in wet or dry form on landscapes.

2) Landscapes differ in sensitivity, based on geology.

Whether or not acidic deposition produces negative effects on the animals living in streams

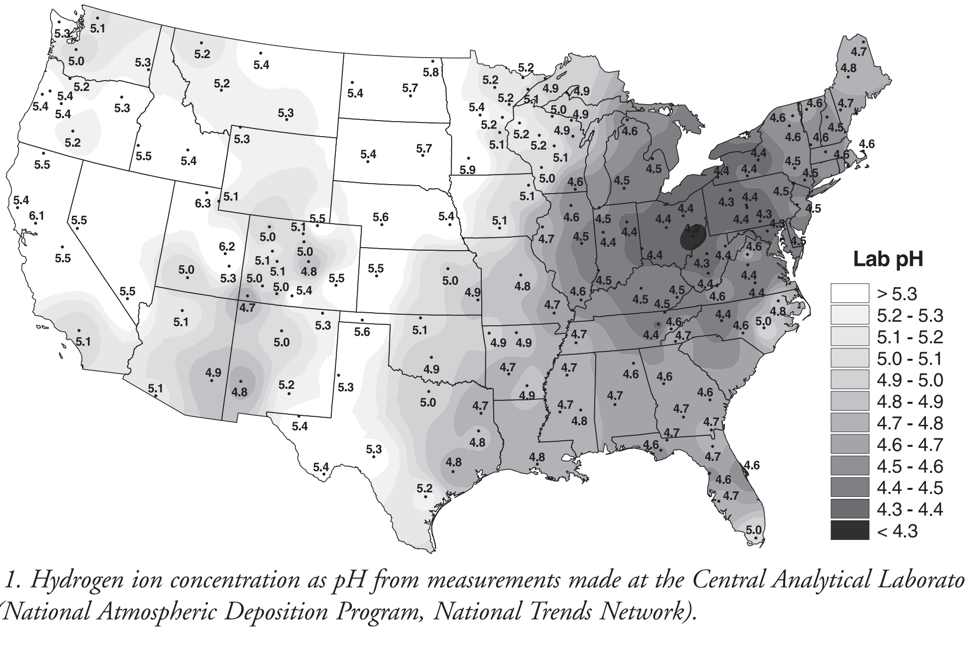

Figure 1. Hydrogen ion concentration as pH from measurements made at the Central Analytical Laboratory, 1999 (National Atmospheric Deposition Program, National Trends Network).

and lakes depends largely on the bedrock geology of their catchments. In landscapes underlain by limestone (carbonate bedrock), which provides substantial buffering of acidity, negative effects due to acidification are neither expected nor seen in water bodies. Basaltic, granitic, and siliciclastic (such as sandstone) bedrock types represent a series of decreasing levels of buffering capacity, such that modest amounts of acidic deposition produce conspicuous negative effects in sandstone catchments. Since buffering capacity ultimately depends on the weathering of acid-neutralizing material from the bedrock, hard bedrock types produce less buffering capacity for streams than soft bedrock types. Mountains by their very nature are more resistant to weathering than surrounding lowlands (that's why the mountains are still there), so mountain streams and lakes are usually the most sensitive to acidification due to the lower buffering capacity of their catchments. In contrast, large valley streams and lakes are often the recipients of upstream weathering products, and are often less sensitive to acidification as the result of their greater buffering capacity.

3) The role of aluminum: metabolic poison.

Aluminum is the most abundant metal on the earth's surface, and the third most abundant element. It is non-toxic and insoluble under acid-neutral conditions, but very toxic to fish and other aquatic species under acidic conditions; unfortunately, the solubility of aluminum increases exponentially as pH falls below 5.6; its maximum toxicity occurs at about pH 5.0. The deposition of acids results in the release of aluminum from soils, which is carried in solution to streams and lakes. Both the aluminum and the hydrogen ion (derived from sulfuric and nitric acids) are toxic to fish, but in most streams and lakes the aluminum is the primary lethal agent; fish can survive more acidic conditions (i.e., lower pH) in the laboratory in the absence of aluminum.

4) Site of toxic action: fish gill

The site of the toxic action of both hydrogen ion and aluminum is the fish gill. The gill is a complex organ responsible for oxygen and carbon dioxide exchange, as well as maintaining the proper salt and water balance the fish's body. It is this later function which is always compromised by acid and aluminum stress; respiration is also compromised at higher concentrations of aluminum.

Freshwater fish maintain salt (sodium chloride) in their blood at concentrations similar to those in humans and most other vertebrates. The proper functioning of most body cells, and especially blood cells in this context, depends on keeping salt concentrations in body fluids within rather narrow limits. Since salt concentrations in the blood are much higher than the water in which they swim, fish constantly lose a small amount of sodium and chloride from the blood by passive diffusion across the thin skin of the gills. The lost sodium and chloride are replaced by an energy-requiring process (active transport) using biochemical "pumps" in the gill membranes which transport sodium and chloride from low concentration in the external stream water to higher concentration in the blood.

Aluminum and hydrogen ion poison the biochemical pumps which transport sodium and chloride into the body; they also weaken the junctions between gill cells, making them leak more sodium and chloride than they otherwise would. The rapid loss without replacement of sodium and chloride produces a cascade of negative physiological effects in the body.

It is a common misconception that stream acidification causes acidification of fish blood, with negative effects on oxygen transport; this does not occur at observed levels of acidification in nature which produce fish death.

5) The cause of death: circulatory collapse secondary to electrolyte imbalance.

The key indicators of incipient mortality under acute acid and aluminum stress are the concentrations of sodium and chloride in the blood plasma. When either or both (sodium and/or chloride) fall more than 30% below normal, death occurs within hours.

The proximal cause of death is ionic dilution of the blood plasma. This causes blood and body fluid disturbances which ultimately kill the fish through circulatory collapse.

Under normal conditions, plasma ionic (electrolyte) concentrations and body cell ionic concentration are in equilibrium. Under acute acid stress, ions are lost more rapidly from the blood plasma than from blood and muscle cells; as a result, there is an osmotically-driven shift of water to the cells from the plasma; blood plasma volume may drop as much as 30%; at the same time, the red cells swell due to the osmotically-driven shift of water from the plasma; the result is a doubling of blood viscosity. The heart is unable to circulate this much thicker blood at a rate sufficient to supply oxygen to body tissues, including the heart itself, so the fish dies of circulatory collapse secondary to ionic imbalance.

In short, the heart cannot supply its own tissues with sufficient oxygen to sustain cardiac activity. So, the fish has a lethal heart attack. Molecules released in Ohio give trout in Virginia heart attacks.

There are wide differences in the sensitivities of fish and other vertebrates to acidity. Acidification eliminates the most sensitive species first. The result is a "dose/response" relationship between acidity and the number of fish species, which looks like this (Figure 2):

This is the number of fish species in lakes with various pH classes in the Adirondacks. These are the lakes that still host fish. About a quarter of all the lakes are fishless in the Adirondacks; at least 20 percent are fishless because they are too acid to support any fish.

We see the same phenomenon in Virginia with a smaller sample size (Figure 3). As Senator Schumer mentioned today, acid rain has moved into Virginia.

Virginia and the Southern Appalachians have had a delayed response to acidification because the soils are thicker. Those soils retain sulfur. When that sulfur retention capacity is exceeded, all the sulfur that comes into the watershed exits to the stream or lake. The southeast is expected to have a slower recovery as well, because after the sulfur deposition is lowered, it will take some time to release the retained sulfur.

Another issue of importance in considering acidification in the northeast vs. the southeast is that lakes are numerous in the northeast and much less so in the southeast. Streams respond quickly to acid deposition, producing large acid episodes; episodes are usually of smaller magnitude in lakes because of dilution in the larger volume of lakes.

Here are some fish modeling results for Virginia (Figure 4). About 30 percent of the probable historic trout streams in Virginia have become too acidic to support any fish (summing categories "chronically acidic", 6% and "episodically acidic", 24%). They don't support brook trout any more. Brook trout is the region's most

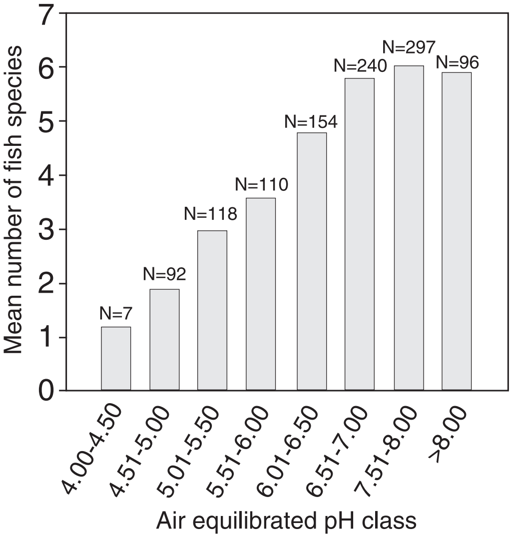

Figure 2. Acidity and number of fish species in Adirondacks waters (NY).

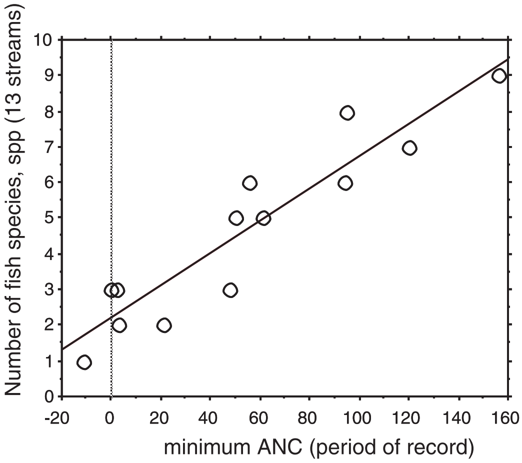

Figure 3. Thirteen streams, with Meadow Run (lowest ANC) showing only one species, reflecting recent loss of Blacknose Dace.

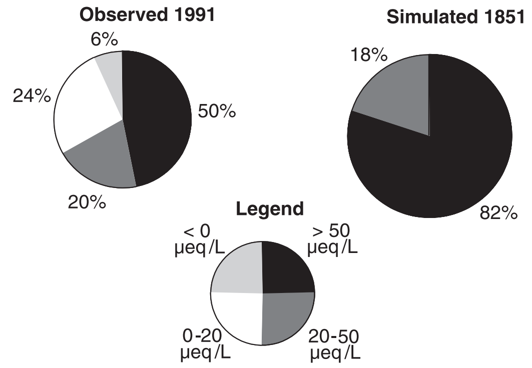

Figure 4. Percentage of streams in alkalinity classes.

acid tolerant species. When brook trout are gone, there are no fish left. By contrast, we estimated that there were no such streams among this population of about 300 Virginia trout streams in pre-industrial times.

This figure (Figure 5) defines the relationships among these four alkalinity classes and likely responses of brook trout populations in Virginia.

The "Southern Appalachian Man and the Biosphere" program (US Forest Service) estimated that, in the Southern Appalachians, there are about 30,000 miles of potential trout streams. Virginia has about 40 percent of the Southeast's trout stream miles. If one is willing to extrapolate from the 30% of Virginia's trout streams which are too acidic for any fish, then roughly 9000-10000 miles of the trout stream mile in the Southern Appalachians may have been lost as suitable fish habitat due to acidification. This must be regarded as a guess, since it is likely that some streams lost their fish populations before any records were made; streams which have become too acid now for fish may not have had fish in the past for other reasons.

This figure (Figure 6) shows further modeling results for Virginia trout streams. The pie diagram at the right is the distribution of streams in the four alkalinity classes, at the base or reference year of 1991. Three scenarios were modeled: continued acid deposition at 1991 levels, a 40% reduction relative to 1991, and a 70% reduction in acid deposition relative to 1991.

Even seventy percent reductions in sulfur deposition, relative to 1991, aren't good enough. There will still be more streams which become acidic and fishless in Virginia, and very probably in other parts of the southern Appalachians as well.

We conclude that reductions in sulfur greater than 70 percent will be needed just to stop further losses, and very substantial reductions will be needed for recovery.

Conclusions for the northeast suggest that reductions in sulfur deposition so far have not produced recovery from acidification, and acid rain is still a widespread problem. Substantial recovery of acidic streams like Hubbard Brook (NH) will occur in 25 years, if power plant SO_2 reductions are lowered by 80 percent. Lakes and streams in sensitive landscapes are still acidic.

The 1990 Clean Air Act amendments will not achieve chemical recovery that will permit biological recovery.

The reason we still have fish in the eastern third of the country is that most landscapes can buffer at least some of the acidity in the rain. However, it has become clear in the last ten years that the buffering capacity of some landscapes has

Brook Trout Category	**ANC Class**	**ANC Range meq L^{-1}**	**Brook Trout Response**
Suitable	Not acidic	> 50	Reproducing brook trout populations expected where habitat suitable
Indeterminate	Indeterminate	20-50	Extremely sensitive to acidification; brook trout response variable
Marginal	Episodically acidic	0-20	Sub-lethal and/or lethal effects on brook trout possible
Unsuitable	Chronically acidic	< 0	Lethal effects on brook trout probable

Figure 5. Brook trout categories, ANC classes, and ANC thresholds for brook trout responses to acidification in forested, headwater catchments in western VA (Bulger et al., 2000).

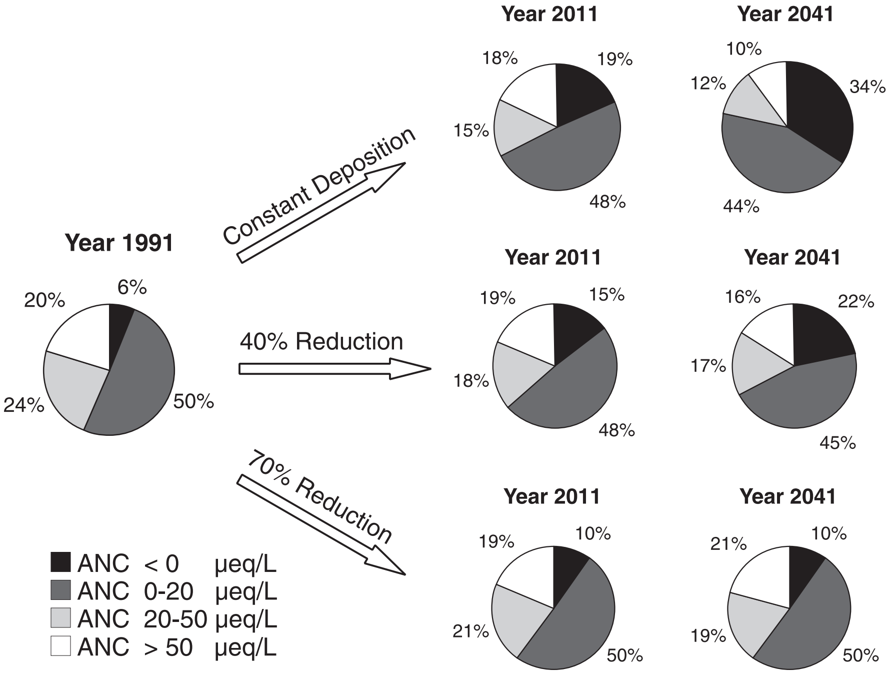

Figure 6. Percentage of streams in alkalinity classes for additional Virginia trout streams.

been substantially reduced, and so acidification will continue without further reductions in emissions.

Can we imagine a recovery? Chemical recovery of streams and lakes is dependent on the rate at which acid deposition is lowered, and the rate of local weathering of bedrock, and is likely to be highly variable.

However, once chemical recovery has occurred and stream conditions reach a pH of about 6, biological recovery is likely to occur on relatively short time scales. In streams, large invertebrates may recover in as little as three years. They are very good colonizers: many are the young stages of insect species that fly as adults.

In lakes, functional zooplankton communities may recover in about 10 years. However, from post-liming studies and recovery from experimental acidification studies, there are no examples where the exact zooplankton communities were restored, but all the zooplankton community functions appeared to be in place after about 10 years.

Fish comminutes might recover to some degree in perhaps 10 to 20 years. Once conditions are suitable for fish, they could be reintroduced rather than waiting for natural colonization events. In terms of the recovery of species composition, the last species in the stream may be the first one back. The most sensitive species, the first to go, will be the last to come back.

QUESTIONS

AUDIENCE: I am sorry if I missed this. You mentioned an 80 percent reduction in sulfate deposition and I would like to know for what baseline and also what has to happen to nitrogen depositions.

MR. BULGER: All the models that I am talking about were based on sulfur deposition, especially in the southeast. I didn't know what to do with nitrogen deposition.

AUDIENCE: The base year for the 80 percent reduction in the northeast, do you know what the base year is for the sulfate reductions in the northeast?

MR. BULGER: Substantial recovery of streams like Hubbard Brook, which is a pretty acidic stream, will occur in 25 years if power stations drop SO_2 emissions by a decrease of 80 percent relative to 1990.

ACKNOWLEDGMENTS

I would like to thank the Center for Environmental Information for the invitation to speak here today. I also thank the US National Park Service, Shenandoah National Park, and the Hubbard Brook Research Foundation for their support and especially for the opportunity to collaborate with many fine scientists, many of whom are authors of the following references.

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