
General Discussion

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General discussion

SIR JAMES BEAMENT, F.R.S. (*Applied Biology, Cambridge University, U.K.*). It is not scientific for us to be speaking of pH, a ratio of ions, SO₂ or any other factor of this kind as ‘killing’ trees or fish. The actual causes of death of animals and plants are few and specific, and we must refer accurately to what may be contributory causes to changes in populations.

A widespread characteristic of the freshwater biota is the evolution of a remarkable range of dispersal mechanisms which enable the recolonization of an environment that has suffered a catastrophe and then recovered to a condition which will again support that biota. The one general exception to that are the fish, and it is therefore much more important to establish whether a body of water is capable of supporting fish than whether there are actually fish in it.

E. D. LE CREN (*Freshwater Biological Association, Ambleside, U.K.*). The last four papers mentioned effects on fish ‘reproduction’; the speakers should distinguish between toxic effects on the physiology of gonad development and spawning and survival of various stages between spawning, and what is generally called ‘recruitment’ by fishery biologists. For example, Dr Nyberg showed an example where salmon appeared to spawn successfully and O group parr were abundant, but there was heavy mortality during their first winter. In some unpolluted lakes there may be successful recruitment only once every few years, for example in some of the perch populations studied by Alm.

B. MORRISON (*Department of Agriculture and Fisheries for Scotland, Freshwater Fisheries Laboratory, Pitlochry, Perthshire, U.K.*). I would confirm the findings reported by Dr Ivar Muniz by saying that in Scotland we have on several occasions done experiments in which we have planted eggs taken from one pair of parent fish (Atlantic salmon, *Salmo salar*) and put a proportion of them into acid (pH 4.0–4.5) streams containing no salmonid populations, and the remainder into nearby streams where the pH range was slightly higher and trout were present. The eggs in the most acid streams failed to hatch, although some reached the eyed stage, whereas in one of the less acid streams a hatching success of 55% was observed.

M. H. UNSWORTH (*Institute of Terrestrial Ecology, Bush Estate, Penicuik, Midlothian, U.K.*). There seems to be a tendency to group together responses of aquatic organisms to acid pulses with responses to long-term shifts in acidity. This seems as unlikely to lead to understanding as the parallel case with plant responses to air pollution: we now recognize that there is no relation between plant sensitivity to high gas concentrations (acute injury) and to low concentrations (chronic injury). Is a division into ‘acute’ against ‘chronic’ responses useful for some aquatic organisms, and if so, can any of the speakers identify areas where our knowledge is particularly lacking?

K. MELLANBY (*Monks Wood Experimental Station, Abbots Ripton, Huntingdon, U.K.*). Some speakers have implied that the discharge of sulphur to the air by industry is a new phenomenon. Before 1914 in Britain we actually burned nearly three times as much coal as we do today. This

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produced a great deal of SO₂, about three million tonnes in 1912. This is about three quarters of the present day emission. There were no very high chimneys at that time, but though these successfully reduce local ground level concentrations they do not greatly affect the long range transmission because whatever the chimney height the gas is quickly mixed into the atmosphere. Did this pollution have any recognizable effects in Scandinavia?

A. KEDDIE (*Warren Spring Laboratory, Gunnels Wood Road, Stevenage, U.K.*). With reference to Professor Mellanby's point about sulphur dioxide emissions in the early 1900s, the best estimates we have for U.K. emissions around 1900 is between 2.6 and 3.0 million tonnes per annum. Emissions peaked at just under 6.0 million tonnes per annum in the late 1960s and early 1970s. The *percentage* increase in other European countries over the same period has almost certainly been much greater because of the higher degree of industrialization in the U.K. relative to other countries at the beginning of the century.

Trends in oxides of nitrogen and hydrocarbon emissions are also relevant. Between 1900 and about 1940 emissions of oxides of nitrogen from the U.K. are estimated to have been about half the current total of approximately 1.7 million tonnes per annum, since when there was a steady rise until the late 1970s. The situation for hydrocarbons is much less certain but a significant rise since about 1940 again seems likely.

There has been a substantial fall in U.K. sulphur dioxide emissions since the early 1970s, the figure for 1982 being about 4.0 million tonnes. There is also some indication of a more recent and gradual decline in emissions of oxides of nitrogen and hydrocarbons. Emissions of sulphur dioxide from some other European countries also exhibit a decline over the past 10 years but I have no information on oxides of nitrogen or hydrocarbons. It will be interesting to see whether these recent downward trends are reflected in precipitation pH and composition and in alleged effects.

N. CHRISTOPHERSEN (*Central Institute for Industrial Research, Oslo, Norway*). Freshwater acidification in Europe and North America is only observed in areas receiving anthropogenic sulphur and nitrogen compounds. This correlation is of course no proof of causality. But what could be the alternative explanations? Is there an alternative common 'denominator'?

If alternative explanations vary from site to site this would of course be highly coincidental. Freshwater acidification is presumably a modern phenomenon. What are we doing wrong now that we have not done before?

D. HAMMERTON (*Clyde River Purification Board, Rivers House, Murray Road, East Kilbride, Glasgow, U.K.*). Dr Howells stated earlier in this discussion that if acid precipitation was the main cause of damage to fisheries in Scandinavian rivers and lakes then one would need to explain the anomaly that damage only began to be observed many years after the onset of such precipitation. In response I wish to pose a question which I hope one of the soil experts can answer. Is it not likely, in at least some of the vulnerable areas of Scandinavia, Scotland or Canada, that the surface soil and rocks have a limited capacity to neutralize the acidity in rainfall and that after many years of continuous acid precipitation this capacity becomes exhausted resulting in a delayed acidification of the surface waters? If true this would also mean that a reduction in acid precipitation would not immediately restore the situation but would require substantial weathering to expose new layers of rock and soil.

I now wish to ask Dr Nyberg whether he considers that liming of streams is really a practicable proposition? Most of our acidified streams in Scotland are to be found in remote locations with no access by road and it seems to me that treatment would be technically difficult and very expensive. A particular difficulty is that lime could probably only be applied to a limited number of spots within a catchment and would only be effective at low flows in summer when the pH is normally at its highest. Could Dr Nyberg also give some estimate of the cost of liming in relation to fishery yields?

B. W. BACHE (*Macaulay Institute for Soil Research, Craigbuckler, Aberdeen AB9 2QJ, U.K.*). The variation in rock chemistry, and in soil properties (both depth and chemical composition) is so great that anything is possible. Many soils in cool moist areas naturally have acid peaty topsoils, and if the soil is shallow in depth and occurs on a hard rock formation (such as quartzite or some sandstones), that is both impermeable and contains little weatherable mineral, the effluent from that soil will have always been acid. At the other extreme, if water percolates through rotting rock that contains appreciable weatherable minerals (even granite), then there is almost infinite capacity for neutralization for both natural or man-made acidity. Somewhere in between there are certain to be situations where the acidity in rainfall provides the extra acid that the soil or rock cannot neutralize in the contact time available, and it will then produce a detectable effect in the surface waters. This could well be a delayed effect. In my own paper, I stressed that careful examination of soil, rock and hydrology is necessary to deduce what is occurring in a given situation. In many cases, reduction of acid precipitation will have no effect on acid waters, because it is not the cause of water acidity, but in cases where the extra acidity in precipitation may just be tipping the balance, one would expect its reduction to reduce the acidity of water, because neutralization is in most cases a time-dependent process. If, however, the neutralizing ability of the soil has been completely exhausted, then as you suggest a reduction in acid precipitation will not restore the situation.

GWYNETH D. HOWELLS (*Central Electricity Research Laboratories, Kelvin Avenue, Leatherhead, Surrey, U.K.*). Depletion of base materials from soils and rocks must have taken place throughout the world's history. Additional acid input could increase base transfer and the rate of weathering, which increases the calcium content of surface waters and decreases the base content of soils. There is evidence for the former, but studies of soils in Sweden and Canada have not shown the latter. Since soil and rock weathering is a continuous process, a reduction in acid precipitation could only change the rate, to a degree compatible with the changed input of H^+ .

G. ABRAHAMSEN (*Norwegian Forest Research Institute, Aas-NLH, Norway*). I should like to comment on the discussion between Dr Howells and Dr Christophersen. I agree with Dr Christophersen in thinking that Dr Howells should have included reductions in the emissions as a measure of reducing lake acidity. General knowledge of soil chemistry and processes, modelling of these and also experimental studies all indicate that the deposition of acid air pollutants has had an effect on freshwater acidity. The effect is likely to be largest in areas with shallow acid soil as those typical in parts of southern Norway. The magnitude of change in H^+ concentrations in small lakes and brooks in these areas is probably of the order of $10\text{--}20\ \mu\text{mol l}^{-1}$. This means that in lakes with a present pH of 4.5 the actual pH-change has not been very large. On the other hand, some freshwater scientists claim that lake pH has changed much more than this

as a result of the acid deposition: sometimes as much as 2 or even 3 pH-units. If these large pH-changes turn out to be true, I know of no explanation for how they can be produced by the atmospheric depositions. In this case there must be additional factor(s) to account for the major change in lake pH.

I agree with Dr Rosenqvist in thinking that changes in the frequency of forest fires are important in this connection. Forest fires were much more frequent and extensive in previous times than they are today. A fire is likely to reduce soil acidity – and thereby adjacent lake acidity – for several reasons:

- (i) accumulated N (included from biological fixation) and S will be volatilized and partly removed from the soil–plant system;
- (ii) organic acids will be transformed to water and CO₂;
- (iii) the ash remaining on the soil will have a lime effect.

To my mind the research within this area should be more open to include all possible sources for lake acidification and not only concentrate on the effects of the deposition of air pollutants.

GWYNETH D. HOWELLS. Dr Abrahamsen suggests considering emission reduction – but presumably to show that changes in surface water pH are quantitatively inconsistent with changes in emissions – which was the reason why I did not include it among the options listed. I agree with his argument for research to identify or quantify other factors which may have brought about the change in surface water quality.

P. F. CHESTER (*C.E.G.B. Central Electricity Research Laboratories, Kelvin Avenue, Leatherhead, Surrey, U.K.*). A number of points made in this Discussion can be tested against data obtained on the 700 lakes in southernmost Norway, discussed by Dr Muniz. Figure 1 shows that pH, *per se*, is not a good discriminator between good and fishless lakes. The concentration of Ca²⁺ relative to that of H⁺ is clearly better, although the ratio may only be a surrogate for the interactions between Al, Ca²⁺ and H⁺ described by Dr Howells.

There is a systematic relation between fishery status and cation balance (Ca²⁺:H⁺, figure 2 and Ca²⁺:Al, not shown) at all altitudes. However, there is little systematic difference in excess sulphate between good and fishless lakes at any altitude. Even though the concentrations of sulphate, H⁺ and Al all decrease with increasing altitude, the incidence of low-ratio, fishless, lakes increases with altitude (from 27% below 200 m to 90% above 1000 m). To improve the fishery status of these lakes it would seem sufficient to improve the cation balance (Ca²⁺:H⁺, Ca²⁺:Al) rather than necessarily to bring about a positive alkalinity. It is not clear that a change in excess sulphate would have this effect.

The results also serve to illustrate the pitfall of inferring changes with time or concentration from synoptic data without an understanding of spatially-related differences.

N. A. SØRENSEN (*Institute of Organic Chemistry, N.T.H. Trondheim, Norway*). I appreciate the analysis that Dr Chester has done with the S.N.S.F. data on 700 lakes at Sorlandet, but I should like to draw attention to a specific point in one of his figures. It showed a curve for high altitude lakes that contained just under 1.0 mg SO₄²⁻ l⁻¹. These lakes belong to the high percentage lacking in fish.

If one averages over the four to five rain chemistry monitors in the area and make a moderate

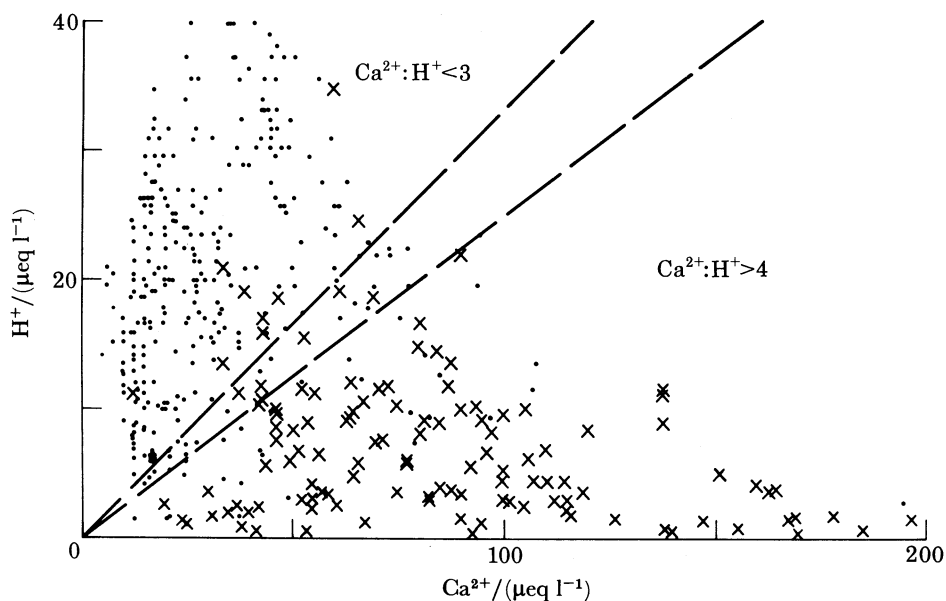


FIGURE 1 (Chester). Chemical balance in fishless lakes (●) and in good or overpopulated lakes (×) in Sorlandet.

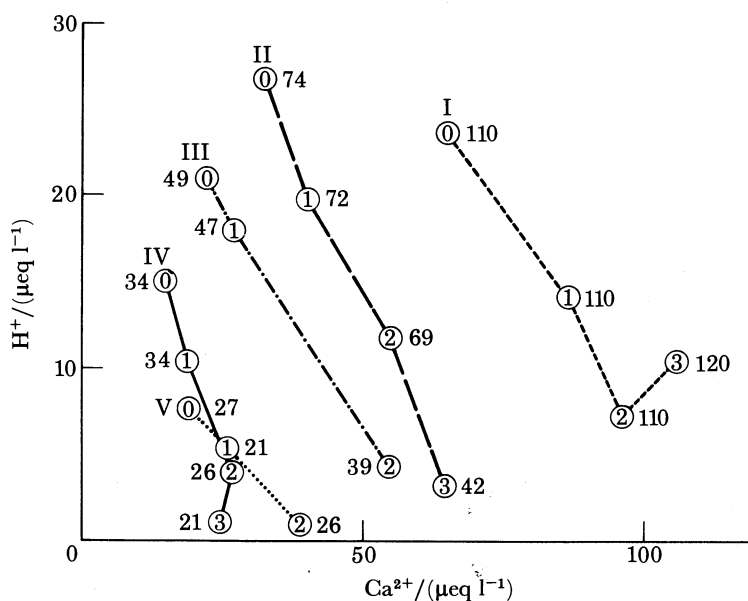


FIGURE 2 (Chester). Average H^+ against Ca^{2+} for Sorlandet lakes in altitude bands I (0–250 m) to V (1000 m+) by fishery category. Average ex SO_4^{2-} is also shown ($\mu eq\ l^{-1}$). (0: fishless; 1: sparse fish; 2: good fishery; 3: overpopulated.)

addition for evapo-transpiration, the sulphate concentration should be in the 3–5 mg $SO_4\ l^{-1}$ range without addition for dry deposition or weathering.

The conclusion must be that in the district of Sorlandet, absorption of sulphate is a dominant process. Dr Goldstein and Professor Kramer briefly mentioned that adsorption of sulphate occurs in soils rich in iron and aluminium oxides. If this is so then Sorlandet must have experienced extensive natural acidification for centuries. It is ironic that in Sorlandet which, from official accounts, is being destroyed by large depositions of foreign sulphate, a very high percentage of fishless lakes is lower in sulphate than most other lakes reported in the literature.

These waters have such low contents of dissolved salts that they fall into the category of ordinary distilled water. One of the best experiments at S.N.S.F. was to put one group of brown trout into purified water; that group succumbed much earlier than any of the groups in acidified water.

R. A. SCRIVEN (*Central Electricity Research Laboratories, Kelvin Avenue, Leatherhead, Surrey, U.K.*). We have heard about the ways in which fish populations of lakes have been estimated by asking anglers, using questionnaires, etc. and there must surely be considerable uncertainties involved. We have also heard of the uncertainties involved with measuring pH and adopting one pH number to represent a lake in space and time. Much of the data in the lectures we have just heard presented fish populations plotted against lake pH where all these uncertainties are compounded. Would the authors please give us some idea of the overall accuracy of their data and the effect that the uncertainties could have on their conclusions?

J. J. MAGNUSON (*Laboratory of Limnology, The University of Wisconsin, Madison, U.S.A.*). As Dr Scriven knows there is much uncertainty in the measurement and reporting of pH measured in soft waters. Also pH is used in my paper only as a correlate with a number of related causal factors such as metal toxicity, calcium deficiencies, and ecological changes to other portions of the aquatic system. Thus it should not be taken as a precise number.

TABLE 1

	number of studies	pH
field experiments		
population loss	2	4.5–5.0
recruitment failure	1	5.0–5.5
fish kill	1	5.9
laboratory experiment		
lethal		
adult mortality		
acute (2 days)	3	3.5–3.8
chronic	2	4.4–4.5
egg mortality	5	4.0–6.1
fry mortality	6	4.0–6.1
Sublethal		
reduced production viable eggs	1	5.1
reduced growth	1	6.5
behavioural avoidance	1	4.5
field experiment		
adult mortality	3	4.4–5.1
egg mortality	1	4.5–4.6

Likewise records of the presence or absence of fish can be biased because the sampling effort may differ and because the recruitment of young fish terminates well before the adult fish reach old age and die. For example a fish that has a lifespan of 10 years may not successfully produce a new year class of fishes but will still remain in the lake for 5–7 years after failure of recruitment occurs. Also, other factors than discussed here influence the year class strengths of fishes such as weather, changing abundance of predators or food. These also induce variability into field estimates of the effects of acidification on the loss of fish. Many field studies have used experimental fishing to access populations and anglers are a remarkably good source of data.

To counter these problems with variability, bias, and imprecision, I have only given the pH at which fish are lost or reproduction fails in strata 0.5 pH units wide. Also I have relied on redundancy of results from three different scientific approaches to identify the strongest scientific evidence, namely field correlations in space and time, laboratory experiments and manipulative field experiments. I gave the example of the fathead minnow to point out how the different kinds of data reinforce each other and allow the conclusion that a loss of fatheads will occur in lakes with a pH less than about 6.0.

Perhaps another example will help. For brook trout (Bakers table 5–14 in the effects volume of the Critical Assessment Document) the range of pH values at which detrimental effects have been observed are given in table 1.

The lowest value in this table (pH 3.5) is for mortalities after only 2 days of exposure. The highest value (pH 6.5) is for sublethal effects on body growth. Each type of evidence (observation, laboratory experiments and field experiments) indicates that at pH 4.5–5.0 major lethal events occur: population loss, chronic death of adults, eggs and fry. When matched against the expected pH of 4.3–4.9 for culturally acidified waters which are sensitive to acidification, higher levels of precision may not be required for broad decision making. For example, a decision to allow sensitive waters containing brook trout to decline to a pH of 4.3–4.9 will in my judgement result in the loss of brook trout from these waters.