Acidification of Norwegian lakes

SIR—Brakke *et al.*[1] claim to have quantified acidification of Norwegian lakes by deposition of H_2SO_4 using a multiple linear regression equation relating strong acidity (as defined by Gran titration) to the independent variables TOC, SO_4^{2-}, and Ca^{2+}. Such quantification is not valid, however, because these three variables are not independent. Natural cycles of bases and acids (for example Ca^{2+} and TOC) in watersheds are perturbed by inputs of acid countering the effect of such input. Thus changes in acidity may be considerably less than predicted.

Henriksen, an author of ref. 1, has shown that concentration of Ca^{2+} and other base cations can significantly increase above background levels because of acidic deposition[2]. Estimated increases are highly variable, but an increase equivalent to 40 per cent of estimated anthropogenic SO_4^{2-} input was put forward as an estimated regional mean value for southern Norway[2].

Organic acids (as represented by TOC) and SO_4^{2-} are not independent variables either. The same chemical principles dictating that inputs of H_2SO_4 increase dissolution of mineral bases dictate that inputs of H_2SO_4 decrease dissolution of terrestrial humic acids[3]. This *p*H-dependent solubility, along with other interactions[4], results in replacement of humicacids by H_2SO_4 without the expected corresponding large change in acidity which is equivalent to estimated change in SO_4^{2-}.

Brakke *et al.*[1] cite palaeolimnological data for southern Norway and state that "these results suggest some reduction of TOC during acidification". Actually, these data indicate that pre-industrial era levels of TOC were 2–3 times greater than present levels and loss of TOC was concomitantly accompanied by a surprisingly small change in *p*H[5]. A compilation of palaeolimnological data show that 9 of 10 acidic (*p*H <5.5) lakes examined in southern Norway were always acidic[6].

A similar compilation, which also includes water chemistry data, indicates that 12 of 14 acidic lakes in the northeastern US were acidic in pre-industrial times. Ten of these 12 lakes are now clear water and SO_4^{2-}-dominated and yet most show little or no measurable acidification. These data, combined with reports of widespread loss of humic colour in the Adirondacks as well as in southern Scandinavia, plus theoretical considerations and experimental data, led the National Academy of Sciences report to suggest that organic acid buffering is important. Norton, an author of ref. 1, is co-author of this report[7].

Reported values for conductivity in Tables 1 and 2 (ref. 1) are erroneously low. For example, conductivity for Lake G-21 in Table 2 is reported to be 2.36 μS (rather than μS cm^{-1}) when conductivity for H^+ alone is calculated to be 11.06 μS cm^{-1}.

Organic acids principally influence *p*H as weak acids not strong acids. Yet the authors assume that organic acidity is independent of *p*H (as is shown in the earlier discussed multiple linear regression equation) and their running a linear regression of anion deficit on TOC: DIFF = 4.06 TOC − 0.96.

Brakke *et al.*[1] incorrectly quote the results presented by myself and Frink[3], "lakes were acidic simply because of processes occurring in acid soils". But we concluded, "acidification by acid rain is superimposed upon long-term acidification". An example was given where acid rain was believed to be the principal cause of acidification; however, we felt that "natural soil formation was often more important than acid rain in determining the acidity of lakes and streams".

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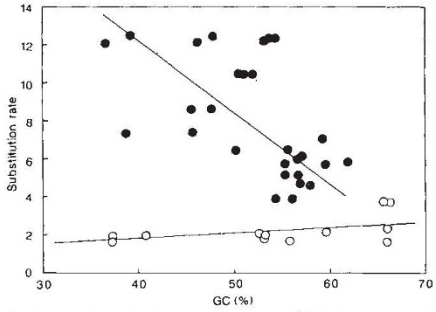
1. Brakke, D.F., Henriksen, A. & Norton, S.A. *Nature* **329**, 432–434 (1987).
2. Henriksen, A. *Acid Rain Res. Rep.* no. 1 (NIVA, Oslo, 1982).
3. Krug, E.C. & Frink, C.R. *Science* **221**, 520–525 (1983).
4. Krug, E.C., Isaacson, P.J. & Frink, C.R. *J. Air Pollut. Control Ass.* **35**, 109–114 (1985).
5. Davis, R.B., Anderson, D.S. & Berge, F. *Nature* **316**, 436–438 (1985).
6. Scruton, D.A., Elner, J.K. & Howell, G.D. *Can. Tech. Rep. Fish. Aquat. Sci.* no. 1521 (Dept. Fish. Ocean, St John's, Newfoundland, 1987).
7. Charles, D.F. & Norton, S.A. in *Acid Deposition: Long Term Trends* 335–431, 482–506 (National Academy, Washington, DC, 1986).

Sequence-based phylogeny in eukaryotic genomes

SIR—Jared Diamond[1] compares the most parsimonious primate phylogeny deduced from DNA sequences of the η-globin gene with that deduced from data on hybridization of total single-copy DNA. The two methods yield the same branching pattern. "A moment's reflection shows why this conclusion was inevitable", he says, pointing to the fact that both methods actually measure the same thing, namely the number of mismatches in heteroduplex DNA, and that the two methods will converge on the same result as the size of the sequenced fragments increases.

His conclusion is obviously true when the length of the compared sequences approaches the total length of a genome. When studying the evolution of eukaryotic genomes, however, we are far from this ideal, and one must make a choice of sequences to compare. This choice, for two different reasons, could strongly influence the final result.

First, hybridization data reflect the number of mismatches in heteroduplexes prepared from bulk DNA, the majority of which is non-coding. Mutations occurring during evolution in non-coding DNA have, in general, been neutral as far as their selective value is concerned. Therefore, they could have been indiscriminately accepted and fixed.



Relationship between the rates[3] [(silent substitutions/year × site) × 10^{-9}] of the silent codon substitutions in several primate (open circles) and rodent (black circles) genes and their G+C content (from the EMBL sequence data bank).

The situation is different in coding sequences of DNA (the major component of data banks). Selection would have prevented the fixation of some mutations, so that the apparent rate of accumulation of mutations is lower in the coding than in the non-coding part of the genome. Hybridization and sequencing data will only give convergent results if the ratio of these two rates of accumulation of mutations has been constant during evolution. But there are examples showing this not always to be the case[2].

A second problem, which is both more serious and only recently recognized, is caused by variation in the rate of accumulation of mutations[3] with the chromosomal localization of the DNA sequence. In primates, GC-rich sequences located in Giemsa-negative chromosomal bands (see ref. 4) accumulate neutral mutations with approximately the same rate as the AT-rich sequences located in Giemsa-positive chromosomal bands. In rodents, however, while GC-rich sequences evolve at a rate similar to that of the equivalent human sequences, AT-rich sequences evolve four times as fast as their human equivalent (see figure). It is likely that the high rate of accumulation of mutations in AT-rich sequences in rodents as well as their compositional bias is caused by less efficient DNA repair in these regions of their genome[5].

Whatever their origin, differences in the rate of accumulation of mutations cause sequence-based phylogeny to be dependent on the choice of sequence