

Trialkyl lead in rain in the Black Forest

SIR—We have recently suggested that high concentrations of trialkyl lead, R_3Pb^+ , as detected, for example, in rainwater samples from the Black Forest, may be harmful to the forests of Central Europe¹. The theory has been seriously questioned by M.H. Unsworth and R.M. Harrison². We wish to provide some new and supplementary data, which we believe can help to remove their doubts.

The most serious questions posed by the authors concerned the bioassay used for quantitation of R_3Pb^+ . In continuing our study for several months of this year on behalf of the government of Baden-Württemberg, two independent methods have been employed for the determination of R_3Pb^+ in rain water. In addition to the tubulin assay, all samples were submitted to a chemical separation procedure³ which was also used for the determination of some of the concentration values cited by Unsworth and Harrison. The results of both assays were the same within the limits of experimental error. Again, four of the samples collected at two locations in the Black Forest had trialkyl lead concentrations higher than 0.2 μM .

Unsworth and Harrison were concerned about the likelihood of the occurrence of such high concentrations of R_3Pb^+ in rain. The concentrations are indeed 100–1,000 times higher than those reported by others. So far, however, analytical data on trialkyl lead in rain are scarce. The few data known are from locations with a typical sea climate, which may not be comparable to locations on the Continent. Moreover, the occurrence of trialkyl-lead-polluted rain seems to be confined to small areas, as shown by the analytical results obtained from one of our sampling stations which, although located only 100 km east of the Feldberg, has never given R_3Pb^+ values above the limit of detection. Sampling at this site alone would have led to different conclusions. Finally, the Antwerp data mentioned by Unsworth and Harrison were one-sample measurements, and therefore statistically insignificant. In 1984, it took us 4 months of uninterrupted observation before we recorded a single toxic event.

Unsworth and Harrison argue that the low concentrations of R_3Pb^+ in the atmosphere cannot account for the high concentrations of R_3Pb^+ in rain. For the undegraded compound R_3Pb , Harrison and co-workers reported atmospheric concentrations at rural sites of up to 230 $ng\ m^{-3}$. After degradation, the distribution of R_3PbX can be expected to remain unchanged, at least for some period of time. However, values of R_3PbX were reported to be as low as 2 $ng\ m^{-3}$. So where is the rest? The loss is most easily explained by

the high instability of salts when dispersed in air. They may be captured by polar media, such as surfaces of particulate matter, and would therefore be analysed together with the inorganic lead. Indeed, in most of the studies of atmospheric lead pollution, the term "inorganic lead" stands for the lead fraction removed by filters. R_3PbX could be washed off the surface of such particles, on contact with rain or fog. In our example, 230 ng of R_3PbX would be dissolved in about 10 ml ($10\ g\ m^{-3}$ is the typical atmospheric humidity in Europe). The resulting concentration would be about $10^{-7}\ M$, which is within the range of R_3PbX found in the Black Forest. This value does not consider the R_3PbX that may be taken up by rain on its way from clouds to the surface.

Fortunately, we do not disagree on controversial theories but on a finding that can be examined experimentally. Therefore, we suggest that further discussion should be postponed until our results have been confirmed by others. Twin samples were taken of most of the rainwater samples collected in 1985 in the Black Forest. They are waiting to be analysed.

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An anniversary for the lipid bilayer

SIR—The year 1985 marked the sixtieth anniversary of the proposal by Gorter and Grendel¹ that biological membranes consist of a double layer of lipid molecules, as appropriately mentioned by Bretscher². In 1925 E. Gorter, a pediatrician with a strong interest in chemical pathology, and F. Grendel, a chemist in the laboratory of the University Children's Hospital at Leiden, The Netherlands, derived their cell membrane model from observations made with rather simple experimental techniques. The lipid extracted with ace-

tone from a known amount of erythrocytes was spread as a monolayer on a water surface. The ratio of monolayer lipid area to total erythrocyte surface turned out to be close to 2:1 for red blood cells from different mammalian species. Thus the investigators concluded with the suggestion that in the cell membrane of erythrocytes sufficient lipid is present for constituting a molecular bilayer. The structural model is the more remarkable because it was derived without any modern instrumental method such as infrared spectrometry, gas chromatography or electron spin resonance. Already 10 years ago, the Gorter-Grendel model was considered by Bretscher and Raff³ a major breakthrough in cell biology, comparable to that of the double DNA helix. In 1934 Danielli and Davson⁴ improved on the model by allowing for proteins to be additional components of plasma membranes. The fluid mosaic model proposed in 1972 by Singer and Nicolson⁵, viewing functional biological membranes as bidimensional solutions of integral proteins in a liquid phospholipid matrix, remains the current basis for experimental work.

In 1966, Bar *et al.*⁶ repeated the Gorter-Grendel experiments and discovered two major errors. First, acetone treatment extracts membrane lipids, and cholesterol in particular, incompletely. Second, more refined measurements of the surface area of the human erythrocyte yield the value of $145 \pm 8\ \mu m^2$ instead of $99\ \mu m^2$, the value used in 1925. The Dutch investigators had in fact made two mutually compensating errors of about 40%.

We are proud that in 1935 Dr E. Gorter, at that time chairman of the pediatrics department at Leiden, accepted an invitation from Ghent State University to come and found the first university children's hospital in Flanders, Belgium. The strong interaction between pediatric observation and biochemical research, kindled by Professor Gorter, and promoted continually by his pupil and successor, Professor Hooft, who died in 1980, remedied effectively the developmental delay of pediatrics in Belgium. We owe much to Evert Gorter, who was once called: 'The best chemist among all pediatricians and the best pediatrician among all chemists.'

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Scientific Correspondence

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