patchy environment created by the leaky cow as it does to wild herbivores.

We have analysed[2] the contribution of the Maasai and their herds to ecosystem dynamics, including patch production, in relation to the part played by wild grazers. Wildebeest have come to dominate the herbivore community in the Serengeti Ecological Unit since the exclusion of the Maasai from the national park. The wildebeest seek out high nutrient concentration areas in which to feed but ruminate, defaecate and urinate in short-grass habitats different from the areas they graze[3]. However, the resulting processes of nutrient stripping and concentration are taken to far greater extremes by Maasai herds which graze in selected pastures, but ruminate, defaecate and urinate in night enclosures. The resulting high-nutrient patches revert to grazing land after a few weeks (for temporary wet season camps), years (in permanent arid zone settlements) or decades (in wetter highland zone permanent settlements)[2].

The overall structure and species diversity of these Serengeti mosaic grasslands are remarkably resilient while grazing is maintained. Belsky[4] has shown the rapid recovery of both species diversity and spatial structure following a variety of natural and experimental artificial disturbances. In contrast to other conservation areas, the Serengeti grasslands resist invasion by introduced species[2]. However, exclosure of grazers leads to loss of productivity and diversity.

Patch production by cattle can go too far. Tolsma *et al.*[5] have shown that nutrient stripping by livestock generates phosphate and other deficiencies in intensively ranched Botswana rangelands, while concentration of nutrients around livestock watering holes leads to the growth of toxic plants. However, in Maasailand, 20 years of attempts to intensify livestock production have failed to change standard measures of the ecology of livestock production[6]. It is just as well for the conservation status of one of the world's richest and most diverse grassland ecosystems that the leaky Maasai cow retains its traditional role.

Katherine Homewood
Department of Anthropology,
University College,
Gower Street,
London WC1E 6BT, UK

1. Moore, P. *Nature* **357**, 644 (1992).
2. Homewood, K. M. & Rodgers, W. A. *Maasailand Ecology* (Cambridge University Press, 1991).
3. McNaughton S. J. *Nature* **345**, 613–615 (1990).
4. Belsky A. J. *J. Ecol.* **74**, I. 419–437, II. 937–952 (1986).
5. Tolsma, D., Ernst, W. & Verwey, R. *J. appl. Ecol.* **24**, 991–1000 (1987).
6. Homewood, K. M. *Ecology of Food and Nutrition* (in the press).

"Surface tension" in the lungs

SIR — In a letter to *Nature* in 1955, Pattle[1] noted that the foam in cases of acute pulmonary oedema was peculiar in that the bubbles were extremely small and very stable. He used two phrases to explain the phenomenon that he had so accurately observed. First, "The surface tension of the lung bubbles is therefore zero"; and subsequently "It is thus evident that the alveoli are lined with an insoluble protein which can abolish the tension of the alveolar surface." Thus the word 'tension' carries two distinct meanings; the first (dynes per cm) as a manifestation of surface free energy (ergs per cm^2) giving rise to the spontaneous contraction of liquids, a consequence of the unequal pull experienced by molecules in the interface between two phases having unequal concentrations; and the second, where a solid surface is too rigid to be distorted by surface free energies.

Recent claims to have achieved zero[2,3] or near-zero[4] surface tension at air/water interfaces are reminders that a body of lung physiologists and neonatologists, following up Pattle's observations, failed to distinguish between the two, quite different, implications of the term 'tension', with the result that a custom has prevailed in which all measurements from whatever device are presumed to identify with the 'tension' of the surface free energy kind.

It is remarkable that these claims, persistent over the past 30 years and pertaining to lung surfactant(s) placed on water in a Langmuir trough or within tethered bubbles or, by implication, within the alveolar spaces of a lung, had not been challenged until recently[5]. The claims are absurd as zero surface tension is tantamount to zero surface free energy, which in turn implies absence of interface. Yet no author has ever described the spontaneous demise of (standing) water into vapour following the application of lung surfactant. The surface tension of a water/vapour interface approaches zero only at the critical temperature (374.1 °C), at which point the concentration of liquid water is equal to the concentration of water vapour and a single phase is established.

Correct interpretation of the measurements is essential if one is to draw valid conclusions relating to lung mechanics, lung microanatomy and to the mechanism of action of lung surfactant. For example, the most common method of assaying lung surfactant is to measure the weight of the meniscus of water supported by a unit length of high-energy solid (for example, a heated platinum plate). Any contamination of the plate by organic matter, whether phospholipid or protein, inevitably reduces the surface free energy of the plate to that below water and the weight of the meniscus falls. Such an event in no way reports upon the true surface tension of the sample solution and could reflect merely the affinity of a phospholipid or protein or both, whether as monomers or aggregated, to a clean metal surface[2].

Comprehension between physical and medical scientists would be resolved if the latter were to limit their reports to what they measure and not what they think they are measuring.

A. D. Bangham
17 High Green, Great Shelford,
Cambridge CB2 5EG, UK

1. Pattle, R. E. *Nature* **175**, 1125 (1955).
2. Cochrane, C. G. & Revak, S. D. *Science* **254**, 566–568 (1991).
3. Schurch, S., Bachoven, H., Goerke, J. & Green, F. *Biochim. biophys. Acta* **1103**, 127–136 (1992).
4. Cockschutt, A. M. & Possmeyer, F. *Biochim. biophys. Acta* **1086**, 63–71 (1991).
5. Bangham, A. D. *Med. Sci. Res.* **19**, 795–799 (1991).

Fatal attraction to cyanobacteria?

SIR — Scavenging of shoreline deposits of neurotoxic benthic cyanobacteria by dogs has recently resulted in deaths at freshwater lochs (lakes) in Scotland. The first report in the scientific literature on animal deaths due to toxic cyanobacteria in lakes, by George Francis, appeared in *Nature* more than a century ago[1], and since then there have been many reports of cyanobacterial animal intoxications and the toxins responsible[2,3].

Until recently, the perceived circumstances of animal poisonings due to cyanobacterial toxins conformed to a general pattern. The buoyant growth habit of the widely encountered toxigenic, planktonic genera such as *Microcystis, Anabaena, Aphanizomenon, Nodularia* and some *Oscillatoria* species can result in scum formation in lakes and ponds during calm weather, so that an acutely toxic dose of cyanobacterial toxins can be presented to animals in a smaller volume than their daily water requirement. The animal deaths due to cyanobacterial toxins in Australia described by Francis[1] and elsewhere by many subsequent investigators have typically occurred after drinking from or near toxic scums[2,3].

Such poisonings have arisen under the following circumstances: if access to clear water is barred and animals are obliged to drink from regions containing toxic scum or blooms; or, as in the case of the deaths of 15 dogs and 20 sheep at Rutland Water, Leicestershire, in 1989