Bone nitrogen isotope composition and climate

SIR—Heaton *et al.*[1] report that the $^{15}N/^{14}N$ ratios of bone collagen from prehistoric humans with little or no access to marine protein show a negative correlation with present rainfall levels in the areas of South Africa they inhabited. Such a correlation cannot, however, be accepted uncritically, because the influences of dietary differences among such populations have not been considered. For example, $^{15}N/^{14}N$ ratios of bone collagen of early historic Griqua pastoralists from the arid interior of Orange Free State are higher than those of late prehistoric Iron Age farmers of the Northern Transvaal[2]. This is expected given the well-known trophic level effect on nitrogen isotope ratios of bone collagen[3]. The data for these two populations, if plotted together against rainfall, would produce a regression similar to that obtained by Heaton *et al.*, but would not reflect only climatic influences. Thus the authors' interpretation of the nitrogen isotope ratios for human bone collagen based strictly on climate seems to be premature.

By contrast, their data for modern herbivores demonstrate a correlation between rainfall levels and the nitrogen isotope ratios of bone collagen that cannot be ascribed to dietary differences because no relationship between plant $^{15}N/^{14}N$ ratios and rainfall was observed[1]. The authors suggest that the higher ratios of "mammals in arid areas are therefore more likely to be linked with the nitrogen metabolism in the body of the animal itself". In the absence of more specific explanations we feel compelled to summarize our own model and correct a potential flaw in its original presentation[4].

The enrichment of ^{15}N in the bone collagen of East African water-conserving herbivorous mammals relative to water-dependent ones that we reported suggested a relationship between physiological mechanisms of water conservation and nitrogen isotopic mass balance[4], as follows. Urea, the major form in which nitrogen is excreted by mammals, is known to perform an essential function in urinary water conservation[5]. Herbivorous mammals on diets high in protein have the capacity to excrete a highly concentrated urine under conditions of heat and water stress. This is accompanied in most species by an absolute and sometimes spectacular increase in urea output, as in the dikdik[6] and two cattle breeds[7], though not in the camel[8] or in cattle on low-protein diets[7]. Urine in *Bos taurus* has been shown to be depleted in ^{15}N relative to the diet, with the depletion being greater during the day than at night[9] (when water stress is presumably lower). If this difference occurs in all mammal species, animals under more continuous water stress would respond by excreting a more concentrated urine with a quantitative increase in the excretion of ^{15}N-depleted urea, resulting in higher $^{15}N/^{14}N$ ratios in the unexcreted nitrogen. In order to satisfy mass balance constraints, the tissues synthesized from this remaining nitrogen pool in water-conserving, urea-excreting mammals should have higher $^{15}N/^{14}N$ ratios than those of mammals that do not conserve water and/or intensively recycle urea. Such a mechanism would explain the nearly one-to-one correlation in rank order we have found between maximal urinary osmolality[7] and mean bone collagen $^{15}N/^{14}N$ ratios for zebu cattle, donkey, impala, goat, sheep and dikdik[4], and most of the data for herbivores presented by Heaton *et al.*[1].

This model is only a hypothesis but it can be tested by controlled water-stress experiments and analysis of nitrogen isotopic mass balance in animals living in different climates and microhabitats. Until the contributions of ecological and physiological processes to variation in animal tissue nitrogen isotope ratios are more clearly understood, palaeodietary analysis based on $^{15}N/^{14}N$ ratios of bone collagen must be regarded as uncertain in all but the simplest of ecological contexts.

STANLEY H. AMBROSE
Department of Anthropology
University of Illinois,
Urbana, Illinois 61801, USA
MICHAEL J. DENIRO
Department of Earth and Space Sciences,
University of California,
Los Angeles, California 90024, USA

1. Heaton, T.H.E., Vogel, J.C., von la Chevallarie, G. & Collett, G. *Nature* **322**, 822–823 (1986).
2. Schoeninger, M.J. & DeNiro, M.J. *Geochim. cosmochim. Acta.* **48**, 625–639 (1984).
3. Ambrose, S.H. & DeNiro, M.J. *Nature* **319**, 321–324 (1986).
4. Ambrose, S.H. & DeNiro, M.J. *Oecologia* **69**, 395–406 (1986).
5. Beewkes, R. in *A Companion to Animal Physiology* (eds Taylor, C.R., Johansen, K. & Bolis, L.) 266–288 (Cambridge University Press, 1982).
6. Maloiy, G.M.O. *Phil. Trans. R. Soc.* **B184**, 167–178 (1973).
7. Livingstone, H.G., Payne, W.J.A. & Friend, M.T. *Nature* **194**, 1057–1058 (1962).
8. Maloiy, G.M.O. *Symp. zool. Soc. Lond.* **31**, 243–259 (1972).
9. Steele, K.W. & Daniel, R.M. *J. agric. Sci. Camb.* **90**, 7–9 (1978).

Signal-transduction

SIR—Wakelam *et al.*[1], as discussed in News and Views by Petersen and Bear[2], have demonstrated that although concentrations of glucagon above 10^{-8} stimulate the intracellular concentration of cAMP but not of inositol phosphates in hepatocytes, low concentrations (10^{-9}M) increase inositol phosphates but not cAMP. We would like to draw attention to the corresponding effects of ACTH on cells from the rat whole adrenal cortex; concentrations of ACTH above 10^{-10} M stimulate cAMP levels but neither ^{32}P incorporation into phosphatidic acid and phosphatidylinositol nor the production of inositol phosphates, whereas ~ 10^{-12} M ACTH produces the opposite results[3,4]. Moreover, we have obtained similar results using rat zona fasciculata–reticularis (ZF–ZR) adrenocortical cells[5,6] and have shown that high concentrations of ACTH inhibit the increase in production of inositol phosphates by angiotensin II in ZF–ZR cells[7].

Wakelam *et al.*[1] also report that TH-glucagon, an agonist for all the biological effects of glucagon, is effective in stimulating the production of inositol phosphates but not the intracellular concentrations of cAMP. There are also ACTH analogues that are effective in stimulating steroidogenesis in ZF–ZR cells but are only weakly active in activating adenyl cyclase[8]. Their activity in stimulating phospholipase C would now be of interest.

The steroid output of ZF–ZR cells is stimulated mainly by ACTH-like peptides but zona glomerulosa (ZG) cells of the adrenal cortex, which produce aldosterone, are stimulated by many different factors. These factors employ either the adenyl cyclase or phospholipase C system[6]. There are as yet no reports of ZG stimulators acting through both systems in the concentration-dependent manner of ACTH in ZF–ZR cells. This would be an appropriate area for further study.

GUY ST J. WHITLEY
Department of Immunology,
St George's Hospital Medical School,
London SW17 0RE, UK
JAMES F. TAIT
Moorlands, Main Road,
East Boldre, Hants SO42 7WT, UK

1. Wakelam, M.J.O., Murphy, G.J. Hruby, V.J. & Houslay, M.D. *Nature* **323**, 68–70 (1986).
2. Petersen, O.H. & Bear, C. *Nature* **323**, 18 (1986).
3. Farese, R.V. *Endocr. Rev.* **4**, 78–95 (1983).
4. Farese, R.V. *et al. Biochem. biophys. Res. Commun.* **135**, 742–748 (1986).
5. Whitley, G. St. J., Bell, J.B.C., Tait, J.F. & Tait, S.A.S. *Proc. R. Soc.* **B22**, 273–294 (1984).
6. Tait, J.F., Chu, F.W., Hyatt, P.J., Tait, S.A.S. & Whitley, G. St. *J. Serono Symp. Corticosteroids and Peptide Hormones in Hypertension* (in the press).
7. Whitley, G. St. J., Hyatt, P.J. & Tait, J.F. *Steroids* (submitted).
8. Schwyzer, R. *A. Rev Biochem.* **33**, 259–286 (1964).

Achieved spacetime infinity

SIR—In his review[1] of my book with John Barrow, *The Anthropic Cosmological Principle*, Press accuses us of indulging in "a distressing amount of mathematical flim-flan . . . For example, . . . Barrow and Tipler [claim] 'A Penrose diagram allows us to define rigorously "an achieved infinity", a concept whose logical consistency philosphers have been doubtful about for thousands of years'. This is a silly assertion, but it is put forth with the utmost gravity, in such a way that many readers will be taken in. And it is only one of many such cases." I cannot comment on the "many such cases" which

are not given in the review, but the "silly assertion" is, perhaps, a case of misunderstanding. Philosophers have debated whether the notion of 'an achieved infinity,' is a meaningful concept since Aristotle questioned the notion in his *Phyics*; see Sorabji[2] for a discussion and a history of the debate up to the Middle Ages. The debate is still current; Popper and Wittgenstein[3] had a sharp exchange on the question in 1946.

As one will see in the context of the book, we meant by an 'achieved infinity' a spacetime singularity. The Penrose diagram, or more generally the c-boundary construction, allows us to define a spacetime singularity in a mathematically precise way[4] by attaching a boundary to spacetime. The c-boundary construction is very elegant and natural for globally hyperbolic spacetimes, the only class of spacetimes we considered. In the c-boundary construction, a regular spacetime point is identified with the past light cone $I^-(\gamma)$ of a future-directed time-like curve γ that terminates in that point, and the future c-boundary points are identified with the past light cones $I^-(\gamma)$ of future-directed time-like curves that have no future endpoints in the spacetime. (Past c-boundary points are defined analogously using future light cones.) If a time-like curve defining the c-boundary point is incomplete (that is, of finite length) the point is said to be a 'singular c-boundary point'. These future singular c-boundary points are the future singularities.

It is generally accepted among relativists that if in fact the Universe is closed and terminates in a final singularity in finite proper time, then the Universe will actually reach this singularity. Strictly speaking, this is not true, since the c-boundary points are not in the spacetime, but there seems to be a general consensus to stretch the meaning of the word 'reach' in this case. Thus the final singularity really exists if the model of gravitational collapse is accurate. From the point of view of the c-boundary construction, the ontological status of the regular spacetime points and the future c-boundary points is the same: they are both the past light cones of time-like curves. Thus if one set of points really exist, then so does the other, and we agree that spacetime really exists. Furthermore, if the initial c-boundary of the Universe is an initial singularity, we can receive information from this singularity just as we can receive information from any other regular spacetime point in our past light cone. Thus it seems reasonable to say it really exists, if such is the initial c-boundary.

The singular c-boundary points are reasonably thought of as infinite because they are (in general) points at which physical quantities are actually infinite. Thus spacetime singularities — if they are indeed on the c-boundary of the actual Universe — are an achieved infinity and are precisely defined by the Penrose construction. I would have thought that this assertion is the overwhelming consensus of the relativity community.

FRANK J. TIPLER
Departments of Mathematics and Physics, Tulane University, New Orleans, Louisiana 70118, USA

1. Press, W.H. *Nature* **320**, 315–316 (1986).
2. Sorabji, R. *Time, Creation, and the Continuum*Ch.14 (Duckworth, London 1983).
3. Popper, K. *Unended Quest: An Intellectual Autobiography*, 15–16; 123 (Fontana, London, 1976).
4. Hawking, S.W. & Ellis, G.F.R. *The Large Scale Structure of Space-Time* (Cambridge University Press, 1973).
5. Penrose, R. in *Theoretical Principles in Astrophysics and Relativity* (eds Lebovitz, N.R., Reid, W.H. & Vandervoort, P.O.) 217–243 (University of Chicago Press, 1978).

Estimating guild sizes

SIR—John Lawton[1] has drawn attention in News and Views to the important simulation model of Shorrocks and Rosewell[2] which assumes that aggregation of individual *Drosophila* species is reflected through the parameter k of the negative binomial distribution. The model is based on successive sums of such distributions for two species. The resulting summed distribution is assumed to be negative binomial also, with parameter k_s, equal to the sum k_1+k_2 of those of the component species.

Shorrocks and Rosewell correctly note that this is precisely true only if $\mu_1 k_2 = \mu_2 k_1$, but claim that with equal mean values of 10 or more and values of $k<2$, it is a close approximation. This claim is technically wrong. Assuming the summed distribution is approximately negative binomial, and that the component species have equal mean values, the method of moments estimator of k_s may easily be found to be

$$\hat{k}_s = \frac{4k_1k_2}{k_1+k_2}$$

The approximation does not depend upon the mean value, and the mean value does not need to be 10 or more.

More importantly, however, unless k_1+k_2 is close to $\hat{k}_s$ the approximation will be poor. To see the scale of error involved take two examples: first, $k_1 = 7/4$, $k_2 = 1/4$ gives a claimed k_s of 2 compared with estimated $\hat{k}_s$ of 7/8; second, $k_1 = 2$, $k_2 = 1/2$ gives a claimed k_s of 5/2 compared with estimated $\hat{k}_s$ of 8/5. Both examples show the approximation is not at all close. The effect that this and the assumption of equal mean values will have on the model requires thorough investigation, although it appears the correction would generate larger estimates of guild size than those estimated by Shorrocks and Rosewell which, as the authors themselves noted, were not quite as large as values from their field studies.

JOE PERRY
Rothamsted Experimental Station Harpenden, Hertfordshire AL5 2JQ, UK

1. Lawton, J. *Nature* **323**, 398 (1986).
2. Shorrocks, B. & Rosewell, J. *J. anim. Ecol.* **55**, 527 (1986).

Aluminium leaching from cooking utensils

SIR—Aluminium is known to have neurotoxic effects[1–3]. With the discovery that abnormally high levels of aluminium are present in senile plaques in Alzhimer's dementia[3–5], the cumulative effects of aluminium poisoning and the question of how this metal enters the body become problems that need immediate attention. Recently, Coriat and Gillard have drawn attention to the high aluminium content of tea leaves and their infusions; aluminium compounds can also be found in water and can be released from utensils during cooking[3–7]. We have found that the leaching of aluminium from utensils is dramatically enhanced in the presence of trace quantities of fluoride ion.

In an experiment conducted to estimate the rate of leaching, we have found that the presence of only 1 p.p.m. of fluoride (the permitted level of fluoridation[8]) in water adjusted with citric acid or sodium bicarbonate to *p*H ~ 3 (a *p*H often realized in cooking conditions) and boiled in an aluminium vessel, liberates nearly 200 p.p.m. of aluminium in 10 min, compared with less than 0.2 p.p.m. in the absence of fluoride. Prolonged boiling produces a concentration of ~ 600 p.p.m., which is reached more quickly the larger the surface-to-volume ratio of the water. The rate of dissolution is *p*H-dependent with a minimum at neutral *p*H. Crushed tomatoes (50g in 250 ml) cooked in the same vessel with 1 p.p.m. of fluoride produced a concentration of ~ 150 p.p.m. of aluminium in 10 minutes. Water consumed in some localities contains 10 p.p.m. or more of fluoride. And cooking or prolonged storage in aluminium ware of foods that contain large amounts of fluoride[9] (~ 500 p.p.m. in tea, 100 – 700 p.p.m. in fish) could easily release more than 100 p.p.m. of aluminium.

The normal resistance of aluminium to corrosion in mildly acidic or alkaline solutions depends on the formation of an inert oxide film. The corrosion in the presence of fluoride perhaps results from a permeability of the oxide film to fluoride ions, which disrupt the protective film.

K. TENNAKONE
S. WICKRAMANAYAKE
Institute of Fundamental Studies, Hantana, Kandy, Sri Lanka and Department of Physics, University of Ruhuna, Matara, Sri Lanka

1. Wils, M.R. & Savory, J. *Lancet* **ii**, 27 – 35 (1983).
2. Brusewitz, S. *Aluminium, University of Stockholm Institute of Physics Report 18 – 11* (1984).
3. Wurtman, R.J. *Sci. Am.* **252**, 48 – 56 (1986).
4. Duckett, S. & Galle, P. *C.r. hebd. Séanc. Acad. Sci. Paris* **282**, 393 – 395 (1976).
5. Candy, J.M. *et al. Lancet* **i**, 354 – 357 (1986).
6. Coriat, A.M. & Gillard, R.D. *Nature* **321**, 570 (1986).
7. Batchelor, B. *et al. Envir. Sci. Technol.* **20**, 891 – 894 (1986).
8. Pike, R.L. & Brown, M.L. *Nutrition: An Integrated Approach* 3rd edn, 190 – 191 (Wiley, New York 1984).
9. Aswathnarayana, U. *et al. Proc. Int. Symp. Geochem. Health* (Royal Society, London, 1985).