Data in graphs and tables

SIR—The ambiguities inherent in the presentation of data in graphs and tables in the ways discussed by Ferreira (*Nature* **324,** 215–216; 1986) may be eliminated by applying the multiplier term to the physical quantity, not to the unit. Thus, to use the example given by Ferreira, the axis would be labelled:

10^{-3} × NADH concentration (μM)

or

10^{-3} × NADH concentration/μM

with scale marks of 2, 4 and 6 to represent NADH concentrations of 2,000, 4,000 and 6,000 μM respectively.

In this presentation the axis labels (or table headings) imply:

10^{-3} × NADH concentration/μM = 2

which rearranges to:

NADH concentration = 2,000 μM

This method is the one recommended to authors by the *Biochemical Journal*.

ALAN S. BEEDLE
Editorial Office, The Biochemical Society, 7 Warwick Court, London WC1R 5DP, UK

Chromogranin A and pancreastatin

SIR—Tatemoto *et al.*[1] recently reported the sequence and biological activity of a pancreatic peptide, which they named pancreastatin because it was found to inhibit glucose-induced insulin release from the pancreas. The authors suggested that pancreastatin "may belong to a hitherto unknown peptide family". We have noticed a rather striking resemblance between the amino-acid sequence of pancreastatin and that of chromogranin A deduced from cDNA sequences reported independently by us[2] and Iacangelo *et al.*[3].

Of the bovine chromogranin A sequence between residues 251 and 294, 70 per cent of the amino acids are identical to those of the 49 amino acids of porcine pancreastatin. In addition, the presence of glycine at position 295 in chromogranin A is consistent with the C-terminal amide structure observed in pancreastatin. The most striking difference between the two sequences is the lack of amino acids 5–8 of pancreastatin between residues 254 and 255 of chromogranin A.

We believe that the sequence resemblance between the bovine chromogranin A segment and porcine pancreastatin is too great to be coincidental. In line with previous sequence data suggesting that chromogranin A may be a precursor of regulatory peptides[2,3], one possible explanation of the observation is that pancreastatin is produced from chromogranin A by limited proteolysis. Such a precursor–product relationship between chromogranin A and pancreastatin is attractive because the endocrine pancreas contains chromogranin A[4–6]. But as chromogranin A is a secretory protein ocurring in a wide variety of peptidergic endocrine cells[4–6] and neurons[7], we find it unlikely that its only function is that of a precursor to pancreastatin.

Lee Eiden, an author of ref. 2, independently reports the sequence similarity between chromogranin A and pancreastatin elsewhere in the News and Views section of this issue[8].

WIELAND B. HUTTNER
ULRICH M. BENEDUM
European Molecular Biology Laboratory, D-6900 Heidelberg, FRG

1. Tatemoto, K. *et al. Nature* **324**, 476–478 (1986).
2. Benedum, U.M. *et al. EMBO J.* **5**, 1495–1502 (1986).
3. Iacangelo, A. *et al. Nature* **323**, 82–86 (1986).
4. O'Connor, D.T. *et al. Life Sci.* **33**, 1657–1663 (1983).
5. Lloyd, R.V. & Wilson, B.S. *Science* **222**, 628–630 (1983).
6. Cohn, D.V. *et al. Endocrinology* **114**, 1963–1974 (1984).
7. Somogyi, P. *et al. Brain Res. Rev.* **8**, 193–230 (1984).
8. Eiden, L.E. *Nature* **325**, 301 (1986).

Origin of hominid bipedalism

SIR—Sinclair *et al.*[1] suggest that hominid bipedalism evolved as a response to selection pressure for long-distance travel to scavenge from migrating ungulate herds. I suggest that simple temporal considerations of hominid evolution render a causal relationship between scavenging and emergence of bipedalism as highly implausible.

An examination of functionally relevant traits of the lower limb, such as shortened distance between sacroiliac and hip joints, carrying angle of the femur, feet with convergent big toes, short toes and arches, indicate that habitual erect posture and bipedalism had evolved in *Australopithecus* by 3.5 million years ago[2]. Throughout the evolutionary history of *Australopithecus* the dental, gnathic and cranial material suggests a high degree of vegetarianism for these early hominids. There is neither morphological nor archaeological evidence that the total dietary repertoire of *Australopithecus* included a larger meat-eating component than that of chimpanzees and baboons. The earliest clear evidence for scavenging, butchering practices and meat-eating is associated with early *Homo* starting around 2 million years ago[3].

The observation that more or less regular meat-eating postdates the emergence of bipedalism by considerably more than one million years falsifies the hypothesis of bipedalism having evolved in response to long-distance travel in pursuit of migrating ungulate herds. In my view the most convincing idea on the origin of hominid erect posture and bipedalism rests on Jolly's feeding hypothesis, in its refined version[4]. This proposes that the hominid locomotor pattern evolved in early *Australopithecus* as an adaptation to gathering and eating the young leaves, seeds, and pods of the ubiquitous thorn scrub of the African plain. These foods grow upon bushes that are too spiny and limber to climb, and too high to pick from a pronograde position. In addition, the earliest hominid, *Australopithecus afarensis,* had relatively short hindlimbs[5], another argument against long-distance travel during that phase of hominid evolution.

WALTER LEUTENEGGER
Department of Anthropology, University of Wisconsin, Madison, Wisconsin 53706, USA

1. Sinclair, A.R.E. *et al. Nature* **324**, 307–308 (1986).
2. McHenry, H.M. *A. Rev. Anthrop.* **11**, 151–173 (1982).
3. Bunn, H.T. *Nature* **291** 574–577 (1981).
4. Jolly C.J. & Plog, F. *Physical Anthropology and Archeology* (Knopf, New York, 1987).
5. Jungers, W.L. *Nature* **297**, 676–678 (1982).

SIR—Sinclair *et al.*[1] believe that human bipedalism arose in scavenging hominid ancestors that had to carry their children while following migrating savanna ungulates but this seems highly improbable.

There was no empty niche of migrating scavengers to be occupied by hominid ancestors. Not only vultures, but also canid, felid and hyaenid carnivores were much better preadapted for such a niche. They possessed sharp beaks or long canine teeth and did not need to carry stones for cutting carcasses. Moreover, the bipedal way of locomotion — whether fast or slow — is inefficient and costly[2,3].

Another argument against the migrating hypothesis in particular and the savannah theory of human evolution in general is that it is highly unlikely that our hominid ancestors ever lived in the savannas. Man is the opposite of a savanna inhabitant. Humans lack sun-reflecting fur[4] but have thermo-insulative subcutaneous fat layers, which are never seen in savanna mammals. We have a water- and sodium-wasting cooling system of abundant sweat glands, totally unfit for a dry environment[5]. Our maximal urine concentration is much too low for a savanna-dwelling mammal[6]. We need much more water than other primates, and have to drink more often than savanna inhabitants, yet we cannot drink large quantities at a time[7,8]. The fossils of our hominid ancestors or relatives are always found in water-rich environments.

It is difficult to understand why most anthropologists keep believing in the savanna theory (possibly because it goes back to Charles Darwin), or why so many anthropologists keep trying to seek the most improbable reasons for bipedalism, while they should know that there are much better explanations[9–11].

MARC VERHAEGEN
Mechelbaan 338, 2870 Putte, Belgium

1. Sinclair, A.R.E., Leakey, M.D. & Norton, M. *Nature* **324**, 307 (1986).
2. Washburn, S.L. & Moore, R. *Ape Into Human*, 77–78 (Little, Brown and Company, Boston, 1980).