matters arising

Feeding analogues

THE recent letter[1] on the control of food intake recalls the old caveat that it is very easy to make an analogue; the problem is to know what it is an analogue of.

The analogue presented invokes operation of an on–off feeding switch by the level of 'energy flow'. It is not at all clear what flow of energy is meant. The values used for turn-on and turn-off of feeding roughly correspond with those estimated by Le Magnen and Devos[2] as 'food utilisation', meaning rate of absorption from the gut. The block diagram, however, shows the effective energy flow after the summing point with lipogenesis–lipolysis; that is, for practical purposes, the energy flow triggering the feeding switch of the analogue is total metabolic rate. Now, for rats, the mean increment in metabolic rate from the beginning to the end of a meal (or, with sign change, from the end of one meal to the beginning of the next) is not more than 10%, and is often much less or even negative[2,3]. It does not approach the 230% (18–60 calorie min^{-1}) proposed by Toates and Booth[1]. In the case where the beginning of a meal coincides with the change from rest to activity, and general motor activity continues beyond the end of the meal, then increase in metabolic rate from just before the onset of the meal to its termination can be about 75%, but that change is almost entirely a function of the change from rest to activity with only a small contribution from feeding as such[3].

We therefore seem to have the situation in which energy flow is interpreted as rate of energy absorption from the gut but is inserted into the analogue as if it were metabolic rate; or in which it is interpreted as metabolic rate but gut absorption values are mistakenly used. This is not just a quantitative discrepancy. Even if 'energy flow' denotes metabolic rate and even if realistic values for metabolic rate are used the analogue as presented would have a nonsense output. Since the major changes in metabolic rate are attributable to change between rest and activity and since feeding must be a part of activity[3], feeding by this analogue can only occur during rest, when, by definition, it cannot occur.

The approximate agreement shown between real rats and the analogue for some variables could indicate that these variables are too insensitive for assessment of the function of the model.

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[1] Toates, F. M., and Booth, D. A., *Nature*, **251**, 710–711 (1974).
[2] Le Magnen, J., and Devos, M., *Physiol. Behav.*, **5**, 805–814 (1970).
[3] Morrison, S. D., *J. Physiol. Lond.*, **197**, 305–323 (1968).

TOATES AND BOOTH REPLY—We stated in our second paragraph[1] that the energy flow controlling feeding in our model is that sampled by hypothetical receptors in neural tissue. As the text and figure specify, we make a crude estimate of this by summating the large and considerably variable flow of energy from the gut, with the circadian variations in net flow from or to triglyceride. We are concerned with some of the energy flows between tissues, not the energy flows between the organism and the environment. The dilemma Morrison[2] poses is therefore fallacious. In a later version of the model[3] we allow for circadian variations in heat production by non-receptor tissues; metabolic rate data assist us there, although (as Morrison says[2]) such variations are relatively small. The physiological variables we use are sufficiently sensitive to give unrealistic behavioural predictions when set at values outside the observed range.

We heartily concur with Morrison's point[2] that the empirical interpretation of mathematical models is a treacherous business. We have been able to discuss the relationships between this model and the facts in more detail elsewhere[3]. We welcome this opportunity to counteract misunderstanding that a necessarily brief report almost inevitably permits.

[1] Toates, F. M., and Booth, D. A., *Nature*, **251**, 710–711 (1974).
[2] Morrison, S. D., *Nature*, **255**, 169 (1975).
[3] Booth, D. A., Toates, F. M., and Platt, S. V., in *Hunger: Basic Mechanisms and Clinical Implications* (edit. by Novin, D., Wyrwicka, W., and Bray, G. A.), (Raven, New York, in the press).

Lead in children's teeth

DAY, HART AND ROBINSON[1] examined the geographical dependence of lead concentrations in urban street dust in several ways. When subdividing data according to city quadrant they noted that the four values ranged from 91 to 122% of the mean value of 970 p.p.m.

We divided our data geographically when studying lead concentrations in crowns of permanent premolar teeth collected from Bristol school dental clinics. The data, plotted according to the home location of the children, fell equally on both sides of a line passing through the centre of the urban area and the Avonmouth industrial complex, approximately 10 km to the north-west. This orientation, however, is approximately at right angles to lead and zinc concentration contours[2] based on extensive sampling of soil and of elm and hawthorn leaves in connection with the regional environmental survey (Sabrina Project).

Table 1 Lead concentrations in tooth crowns according to city zones

Zone	Mean concentrations (p.p.m.)			
	Pb	Zn	Cd	Cu
NE	71±19*	132±32*	4.3	6.6
SW	53±16*	143±36*	4.4	6.6

* Standard deviations (28 samples). Difference (north-east/south-west) significant for Pb ($t = 3.7$, $P < 0.001$).

Concentrations of lead in Bristol children's teeth are typically 60 p.p.m. A mean value for tooth samples from 28 children living in a zone to the south-west of the chosen demarcation line was only 75% of that for the same number representing the zone to the north-east (Table 1). There was a non-significant difference in the opposite direction for zinc analyses; the values for cadmium and copper were the same in the two zones.

Another aspect of this type of environmental observation—but in which no geographical distinction was evident—is provided by a study[3] of concentrations of lead and other metals in developing teeth in cases of stillbirth and neonatal death. There was significantly more lead in the dentitions of those born in 1972–73 than in those dating from 1957–63.

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[1] Day, J. P., Hart, M., and Robinson, M. S., *Nature*, **253**, 343–345 (1975).
[2] Little, P., and Martin, M. H., *Environ. Pollut.*, **3**, 241–254 (1972).
[3] Stack, M. V., Burkitt, A. J., and Nickless, G., *Postgrad. med. J.* (in the press).

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