interpretation of a positive correlation between energy intake and height. Since height is a measure of the size of the subject, this interpretation is not implausible. In order to rule it out, it would be necessary to show that in the large sample studied there is no significant correlation between BMI and height.

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¹ Womersley, J, Durnin, J V G A, *British Journal of Nutrition*, 1977, **38**, 271.

² Benn, R T, *British Journal of Preventive and Social Medicine*, 1971, **25**, 42.

³ Newens, E M, and Goldstein, M, *British Journal of Preventive and Social Medicine*, 1972, **26**, 33.

* *We sent copies of these letters to the authors, whose reply is printed below.—ED, BMJ.

SIR,—Miss Pamela Mumford and Dr Elizabeth Evans are right in pointing to the relative imprecision of the methods we used for collecting dietary information from the 3500 subjects in our studies. The difficulties and inaccuracies inherent in all methods of collecting such data probably account for the absence of relationships between food intake, body weight, and metabolic characteristics in some studies. However, even using these imprecise methods we *did* find relationships, the key ones in our argument being statistically most unlikely to have arisen by chance. The question, then, is whether the imprecision of the methods we used might have spuriously created the correlations we found or affected their order of magnitude. We could only consider both these aspects briefly in our condensed report.

However inaccurate a method of measurement may be, so long as those errors in estimate (be they erroneous overestimates, underestimates, or both) are randomly distributed, they will not, other than by chance, introduce a systematic trend and so generate artefactual correlations. There is no reason for supposing that the method used for the assessment of food intake systematically underestimated for the heavier or overestimated for the lighter subjects. We accept that obese people sometimes fail to recall the whole of their food consumption but so do non-obese subjects^{1 2}; lapse of memory is not necessarily proof of self-deception. A few obese subjects may consciously mislead their inquisitors but a "deception factor" is hardly likely to operate throughout the leanness-adiposity spectrum; the inverse relationship which we reported extended right across the range of BMI, not just between the obese and the remainder. While we think it very unlikely that the method of data collection actually created the relationship we agree that its imprecision may well have led to an underestimate of its true strength.

Other, apparently more accurate, methods of dietary evaluation might increase the value of the correlations we found; but they are not without their own quite serious problems of application. In theory, weighing and recording every item of food consumed is ideal but, in practice, it is so tedious, messy, socially embarrassing, and intellectually demanding that many subjects alter their eating habits or

make guesses to ease the task. Many "drop out" or decline to participate, introducing biases in the population sampled, particularly at the lower end of the social scale³; whereas our method is acceptable to most. We have been able to compare our assessment of food intake with actual food consumption in a sample of subjects and have found a high degree of correlation. Repeated estimates weeks or months apart on other samples of our populations show very comparable mean intakes and significant preservation of rank order within the groups. We shall be describing our methods and these validating studies elsewhere in detail.

Finally, we would enter a mild protest at the slightly distorted way in which our conclusions have been put. We did not really "claim that the less people eat the heavier they are," a statement which might be thought to carry cause-and-effect implications that we certainly did not draw. We recorded data which suggested that the amount of food people ate was inversely related to their degree of adiposity, leaving the interpretation of this correlation open, though we made a few suggestions.

The comments of Drs Cooper, Gurney, and Jutsum are well founded. Keys *et al*¹ noted that, of all the indices of obesity, the BMI was least correlated with height, but some residual relationship remained. We did find a small but statistically highly significant negative correlation between height and BMI in our populations and consequently there was a positive correlation between caloric intake and height, although this only achieved significance in the Beecham and Whitehall men.

Multiple regression analysis, however, showed that there was no significant independent relationship between caloric intake and height when the BMI was introduced into the regression equation other than in the Whitehall men, while the BMI remained significantly negatively correlated with food intake independently of height in all population groups. Thus part of the inverse correlations we reported may be due to the confounding variable of height, but even after making statistical allowance for this the effect remains.

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¹ Campbell, V A, and Dodds, M L, *Journal of the American Dietetic Association*, 1967, **51**, 29.

² Trulsson, M F, *Journal of the American Dietetic Association*, 1954, **30**, 991.

³ Thomson, A M, *British Journal of Nutrition*, 1958, **12**, 446.

⁴ Keys, A, *et al*, *Journal of Chronic Diseases*, 1972, **25**, 329.

Doctors and children's teeth

SIR,—Your recent leading article (12 May, p 1231) on children's teeth made many good points but unfortunately omitted to mention any means of ingesting fluoride other than in water. There are still many unenlightened parts of the country where this safe and simple procedure has not been instituted and the main alternative should be tablets; these ought to be stocked by all child health clinics and prescribed from infancy onwards in non-fluoridated areas. An earlier leading article¹ recommended a dose of 1 mg daily for children over 2 years, and advised the user to suck the tablet slowly rather than swallowing it to give a topical as well as a systemic effect.

Unfortunately drops for infants are not readily available and the tablets need to be crushed and dissolved in water—a task which will limit their use to the most dedicated of parents. What about a new preparation: Fluorabidec?

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¹ *British Medical Journal*, 1975, **1**, 535.

Trauma and Paget's disease of bone

SIR,—Further to Dr L R Solomon's article (7 April, p 931) and Dr T M Gasper's letter (5 May, p 1217), it might be appropriate to mention that in the French medical literature several cases of localised^{1 2} and even generalised^{3 4} Paget's disease of the bone have been reported to develop after trauma.

In the cases described the latent period between trauma and diagnosis varies considerably.⁵ The relative frequency of involvement of the different bones—and particularly the fact that the right femur is twice as frequently affected as the left one—points to the possibility that trauma is at least a factor in the development of Paget's disease.⁶ Trauma may precipitate a breakdown in the normal mechanism of gradual continuous replacement to which bone is constantly subjected and result in the development of Paget's disease.⁶ The relative immunity of the bones of the foot and the mandible is difficult to explain.

Trauma, however, cannot be solely responsible for Paget's disease, as the microscopic ultrastructure, and especially the discovery of intranuclear inclusion bodies in the osteoclasts of bone specimens affected by Paget's disease and not in other normal or pathological bone specimens, provides indirect evidence that the condition may be due to a slow viral infection.^{7 8} This hypothesis would also explain the geographical distribution of the disease and its tendency to run in families; but the virus has not yet been identified.

At present the aetiology of Paget's disease seems to be multifactorial: it is probably due to a slow virus infection, with trauma acting—in some cases—as a localising factor and heredity as a predisposing factor.

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¹ Lievre, J A, *Journal Belge de Rhumatologie et de Médecine Physique-Acta Medica Belgica*, 1974, **29**, 322.

² Lievre, J A, *La Presse Médicale*, 1936, **3**, 45.

³ Louyot, P, and Gaucher, A, *Revue Médicale de Nancy*, 1961, **582**.

⁴ Froment, J, Bonnet, P, and Brun, J, *Lyon Médical*, 1939, **33**, 169.

⁵ Raulis, A, *Journal Belge de Rhumatologie et de Médecine Physique-Acta Medica Belgica*, 1974, **29**, 367.

⁶ Jaffe, H L, *Clinical Orthopaedics and Related Research*, 1977, **127**, 4.

⁷ Mills, B, and Singer, F, *Science*, 1976, **194**, 201.

⁸ Rebel, A, *et al*, *Calcified Tissue Research*, 1976, **20**, 187.

Differences between Leeds fractures and London fractures?

SIR,—We were surprised at the assertion of Dr M R Baker and others that their control data on plasma 25-hydroxy vitamin D (25-OHD) concentration are almost normally distributed (5 May, p 1218), and this led us to calculate the coefficients of skewness ($\sqrt{\beta_1/S}$) for their series. The coefficients were 1.9

(controls) and 1.2 (fractures)—very similar to those of our own series, 1.6 and 1.8 respectively. All these distributions are therefore significantly positively skewed ($P < 0.005$).

We are, however, reassured that the observed difference in mean 25-OHD concentration between the two groups is statistically significant when suitable steps are taken to allow for the non-normality of the data. The reason that we did not observe such a difference in our own study presumably reflects, as Dr Baker and his colleagues suggest, the choice of control group. Because we wished to allow for the influence of a recent operation on the variables measured, we chose a control group from patients admitted to the same wards for elective surgery. Our fracture and control populations received almost identical sunlight exposure, whereas in the Leeds study the control group had been exposed to nearly twice as much sun as the fracture patients.

Although we were unable to implicate vitamin D deficiency in the pathogenesis of this fracture in London, the results of our study are consistent with the idea that the reduction in 25-OHD concentration observed in the Leeds fracture patients is due to their reduced exposure to sunlight, rather than due to some intrinsic defect in vitamin D metabolism.

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*.*This correspondence is now closed.—Ed,
BMJ.

Whooping-cough vaccination

SIR,—Dr David Turner (31 March, p 892) has questioned the need for erythromycin prophylaxis in young infants exposed to whooping cough, firstly because it would be difficult to get parental co-operation in giving a pleasantly flavoured syrup for two weeks to prevent an unpleasant or even dangerous disease and, secondly, because we should rather "speak with one voice" in advocating whooping cough vaccination, which will protect the vulnerable infant by herd immunity.

I believe that it is important to answer the second point and also to try to define more closely the concept of herd immunity. In certain infectious diseases such as diphtheria or poliomyelitis, when three-quarters of the population are vaccinated the natural spread of infection becomes so disrupted that the disease tends to die out. This valuable bonus I accept is effective herd immunity. However, in other infectious diseases despite the vaccination of three-quarters of the population the infection continues to spread unchecked in the community, although the disease may be most evident in the unvaccinated quarter of the community. This pattern appears true of measles and whooping cough, and unfortunately in the latter the "unvaccinated quarter" always contains the entire infant age group, where the mortality is largely concentrated.

The table shows data from two peak whooping cough years, before and after the recent decline in vaccination rates. The increase in notifications in 1978 would appear to be related to the decline in vaccination, but the number of infant deaths from whooping cough remains fairly constant at about one death per 50 000 births in each of the years. I interpret these figures as tending to show that

Whooping cough before and after decline in vaccination

	1974	1978
No of notifications	16 372	65 892
Vaccination rate (%)	77	<40
No of children born	640 731	592 668
No of infant deaths from whooping cough	13	11

whooping cough vaccination confers individual protection but not herd immunity.

For this reason I still put forward the view that erythromycin prophylaxis is the best practical approach to the protection of the vulnerable infant when whooping cough affects a family.

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New packaging of insulins

SIR,—At this hospital we are very concerned about the recent change in policy towards the packaging of insulins by one manufacturing company—that is, Novo Industri A/S. For many years the convention of colour coding insulin packs according to type and strength, regardless of manufacturer, has been regarded as a fine example of intercompany co-operation and of great value to pharmacists and patients.

In the convention the colours, blue, green, and yellow are particularly important in denoting strength; if Novo is genuinely concerned that we are not reading the labels properly it would have been more convincing had it used plain white packs with bold black lettering on all six faces of the box, instead of juggling with the established colours.

Novo's pack changes appear to us to be totally unjustifiable. The other insulin manufacturers have told us that they intend to retain the colour code convention, and we hope that Novo will restore the old packs as soon as possible.

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*.*We sent a copy of this letter to Novo, whose reply is printed below.—Ed, *BMJ*.

SIR,—Thank you for allowing me through your columns to reply to the letter you have recently received regarding the new packaging of our MC insulins. I should like to explain briefly the background to the packaging changes, which were not undertaken without much thought, discussion, and planning.

For some time concern has been expressed about the use of various colour codes for identification of type and strength of insulins and, with the advent of the highly purified insulins, there were insufficient colours available to continue with the old colour coding system. Among our own preparations, for example, both Lentard MC and Monotard MC were marketed under the same colour code, which was obviously an unsatisfactory state of affairs. Consequently, in October 1978 the British Diabetic Association called a meeting, which was attended by members of the medical and scientific section and the insulin manu-

facturers to discuss future policy for the labelling of insulins. This meeting concluded that:

(1) Colour should no longer be used to indicate insulin strength on insulin packs or labels (though it may be retained for vial caps, if generally agreed).

(2) Strength should be indicated by figures of adequate size set boldly, prominently, and repeatedly on the pack and label.

(3) Insulin type should be indicated in words on pack and label. Individual manufacturers' designation should be followed by the approved pharmacopoeial name, which should indicate species of origin.

(4) The inclusion of colour on the pack or label becomes a matter for individual manufacturers' policy. It may be a guide to brand recognition, production, or distribution control but must no longer be regarded as a general indication of strength or type.

(5) The public and professions must be informed of these changes.

We have followed these conclusions and recommendations with the approval of the medical and scientific section, the DHSS, the Pharmaceutical Society, and other relevant groups, culminating in the introduction of the new packaging whereby our insulins are identified by bold black printing on a white background, clearly indicating the type and strength of insulin (the band of colour on the cartons and labels is for production purposes only and does not constitute a colour code). Finally, we are conducting a widespread, comprehensive educational programme to familiarise members of the medical and allied professions and patients with the new system.

I hope that this brief summary will emphasise that we have not embarked on this exercise casually or unethically. Our concern, as always, is for the safety and well-being of the patient and we believe that the new packaging of our insulins is entirely consistent with that aim.

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Is there a future for clinical cytology?

SIR,—In her article (14 April, p 1031) Dr Dulcie V Coleman has presented much-discussed and pertinent questions relating to the future of cytology in this country.

The British Society for Clinical Cytology (BSCC) has been active in the development of this specialty over the past 15 years, both in the promotion of new investigational and diagnostic techniques and in the improvement of training and qualifications of cytopathologists and cytotechnologists. Dr Coleman produces a rather slanted view of the practice of most cytology laboratories in Britain. The initial use of unqualified personnel in the early 1960s was necessary to deal with the rapid increase in the number of uterine cervical smears when the national screening programme was introduced, and such screeners persist in only a few laboratories in this country. As a society we do not advocate the continuance of such a practice, though we recognise that among such "screeners" there are some highly skilled and competent workers—for whom, incidentally, the certificate of competence examination has been created to maintain standards in cytology screening.

The protracted negotiations by the BSCC with the DHSS, the Royal College of Pathologists, and the Institute of Medical Laboratory Science from the late 1960s have resulted at