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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Thirty-ninth Meeting of the Nutrition Society was held in the Physiology Lecture Theatre, Guy's Hospital United Medical and Dental Schools, London, on Wednesday, 27 May 1987, when the following papers were read:

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The dental effects of lactitol replacing sucrose in confectionery examined in a clinical trial.

By T. H. GRENBY and T. DESAI, *Department of Oral Medicine and Pathology, Guy's Hospital UMDS, London Bridge, London SE1 9RT*

One of the bulk sweeteners put forward as a substitute for sucrose to benefit dental health is lactitol, a polyol derived from lactose, but very little is known of its dental properties in vivo.

After approval from the Hospital Ethical Committee, and with their informed consent, thirty pre-clinical student volunteers were divided at random into two groups and given boiled sweets (unidentified) made with either lactitol or sucrose, to eat in place of their normal confectionery and snacks, along with their ordinary diet, over a 3-d period (average sweet consumption 30–40 g/d). They were asked to refrain from any oral hygiene measures, including tooth-brushing.

Initially they were given a full dental examination and all deposits were removed from their teeth. Dental plaque formed during the trial was measured by three methods. A. After applying disclosing solution, stained surfaces were classified *in situ* on a 1–5 scale. B. The area of plaque was measured by superimposing a grid on standard photographs of the teeth. C. All the plaque was collected from each individual, then dried and weighed.

Substituting lactitol for sucrose in the sweets reduced the amount of plaque accumulating on the teeth by a statistically significant margin in methods B and C.

	Sucrose group		Lactitol group	
Method	Mean	SE	Mean	SE
A (score)	2·65	0·18	2·38NS	0·16
B (units)	2·31	0·39	1·04**	0·17
C (mg)	9·7	1·2	6·1*	0·9

NS, not significant; *$P = 0{\cdot}025$, **$P < 0{\cdot}005$.

Chemical micro-analysis showed that the plaque from the lactitol group contained less glucose and sucrose but more calcium, phosphorus and protein than that from the sucrose group. These changes in the extent and composition of the plaque may be taken as advantageous for dental health, in view of the correlations established in the past between individuals' plaque accumulation and their dental caries experience.

However, the volunteers were also given a questionnaire on their eating habits, opinions of the sweets and any digestive effects. This yielded observations on the rough texture and adverse gastric reaction to the lactitol sweets. If these problems can be overcome, replacing sucrose in the sweets by lactitol produces an improvement in some of the factors associated with dental disease.

We are grateful to Express Foods Group Ltd for their support.

Dental caries potential of children's rusks. By T. H. GRENBY and A. PHILLIPS, *Department of Oral Medicine and Pathology, Guy's Hospital UMDS, London Bridge, London SE1 9RT*

A large number of laboratory tests have been done to determine the cariogenic (dental decay-causing) effects of adult foods, but very little information is available on products specially formulated for young children, and disquiet has been expressed over the high proportion of sugar that some products contain. A programme of experiments was therefore set up to study the dental properties of six different kinds of children's rusks with a wide range of sugar contents. They were tested first in a strain of caries-active laboratory rats. The rusks were pulverized and incorporated at 660 g/kg in diets given *ad lib.* for 56 d from weaning. Food and water intakes and weight gains were recorded. After 56 d the mandibular molar teeth were examined for dental plaque and caries.

Rusk no.	Rusks' content (g/kg) of:		Caries scores	
	Sucrose	Total sugars	Mean	SE
1	310	310	27·7	3·0
2	150	255	21·0	2·6
3	125	220	18·3	4·4
4	60	153	9·4	1·6
5	135	177	8·5	1·9
6	0	165	6·3	1·3

In a second trial with the three highest-sugar rusks the results followed the same pattern. It can be seen that with only one anomaly, possibly due to a high phosphorus content, the caries incidence was directly related to the sucrose content of the rusks. The scores fell into two main groups, with significant differences between the three highest and the three lowest.

The cariogenicity of a food depends on (1) its suitability as a substrate for oral micro-organisms to produce acid which can attack the mineral matter of the dental enamel, (2) its adhesiveness to the tooth surface, and (3) frequency of intake. Properties (1) and (2) were examined in experiments with the rusks in vitro. Ranked in order of decreasing caries potential, the results showed good general agreement with the animal results.

A link was established between cariogenicity and sucrose content, rather than combinations of other sugars such as glucose, maltose and lactose, with possible implications for child dental health.

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The use of questionnaires to predict fat and fibre intakes. By J. R. KEMM and A. J. BRAY, *Department of Social Medicine, University of Birmingham, Edgbaston, Birmingham B15 2TJ*

There have been many attempts to use questionnaires to categorize subjects by nutrient intake but their validity is doubtful (James *et al.* 1981).

Twenty-three adult volunteers from the university community (eight male, fifteen female, mean age 35 (SD 9·7) years) compiled a 7 d weighed intake record and also completed a 108 item, closed-response questionnaire on food habits. Mean (and SD) intake of energy was 8·4 (2·2) MJ/d, fat 84·7 (23·3) g/d, fat 37·3 (2·9) % of energy intake, fibre 24·2 (8·2) g/d, fibre 2·94 (0·88) g/MJ energy intake, calculated from standard food tables (Paul & Southgate, 1978) and additional tables from the Dunn Nutrition Laboratory, Cambridge.

The main sources of dietary fibre in the UK diet are vegetables, bread and flour, other cereals and fruit (Wenlock *et al.* 1984). Questions on the type of bread and breakfast cereal consumed identified groups with significantly different fibre intakes, but questions on vegetables and fruit consumption did not (see Table). The main sources of fat in the UK diet are dairy products, meat and meat products and yellow fats (Ministry of Agriculture, Fisheries and Food, 1986). Questions on the consumption of red meats and type of milk identified groups with significantly different fat intakes (as % of energy) but questions on the consumption of meat products, cheese or other fat-rich foods did not. Some questions on the consumption of fat-rich, fibre-free foods identified groups with significantly different fibre intakes while some questions on fibre-rich, fat-free foods identified groups with significantly different fat intakes.

		Fibre intake (g/MJ)		Fat intake (% energy intake)	
Questionnaire response	*n*	Mean	SD	Mean	SD
Type of bread consumed					
Wholemeal	17	3·32***	0·61	37·3 NS	2·2
Other	6	1·84	0·52	37·1	4·7
Type of breakfast cereal consumed					
Wholewheat	13	3·32**	0·67	36·5 NS	2·6
Other	10	2·45	0·91	38·3	3·2
Frequency of eating red meat					
<3 times per week	14	3·22*	0·76	36·1**	2·8
>3 times per week	9	2·50	0·92	39·1	2·1

NS, not significant. *$P<0·05$, **$P<0·01$, ***$P<0·001$.

These results demonstrate that questions about foods rich in a particular nutrient do not necessarily predict an individual's intake of that nutrient.

James, W. P. T., Bingham, S. A. & Cole, T. J. (1981). *Nutrition and Cancer* **2**, 203–212.
Ministry of Agriculture, Fisheries and Food (1986). *Household Food Consumption and Expenditure, 1984*. London: H.M. Stationery Office.
Paul, A. A. & Southgate, D. A. T. (1978). *McCance and Widdowson's The Composition of Foods*, 4th edn. London: H.M. Stationery Office.
Wenlock, R. W., Buss, D. H. & Agato, I. B. (1984). *British Medical Journal* **288**, 1873.

How valid are hospital studies of nutritional balance and metabolism? By M. ELIA[1], N. J. FULLER[2] and K. FOTHERBY[1], [1]*Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL* and [2]*Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ*

Nutrient recommendations in normal subjects and in hospitalized patients have frequently been based on the results of balance studies. In such studies, it has been assumed that the collections (especially those of urine) are complete. However, since there is often no objective evidence for this assumption, major errors may have been overlooked. The present study aimed to assess the validity of such assumptions in hospitalized patients receiving artificial nutritional support. The completeness of urine collections was assessed by administering a continuous infusion of sodium *p*-amino-hippurate (PAH; 750 mg, 2 μCi [^{14}C]PAH/d) to eight patients receiving total parenteral nutrition (TPN) and measuring the recovery of this marker in urine (thirty-two daily collections). PAH recovery was determined by radioactive, colorimetric and high pressure liquid chromatography (HPLC) techniques. Both the between- and within-subject variations in PAH recoveries were determined.

Complete urinary recovery of intravenously administered PAH had been confirmed in normal subjects and TPN patients studied under strictly controlled conditions. Patients with normal renal function achieved a steady state 3 h after receiving the PAH infusion. During such a state, the total body pool of PAH was estimated to contain about 5% of the 24 h infusion dose. No PAH was detected in intestinal effluent.

The mean recovery of [^{14}C]PAH collected in urine, once the steady state had been achieved, was 73 (SEM 3)% of the infused dose (range 38–112%). Only two collections were over-collections, whereas 21 (SEM 65)% were under-collections containing less than 80% of the infused dose. Some patients assessed for up to 6 d continuously demonstrated systematic under-collections. The radioactive method proved to be the most simple and reproducible method correlating well with the colorimetric assay (r 0·996 with 95% limits of agreement ($d \pm 2$ SD) ranging from 2·6 to −4·0%). However, some drugs or their metabolites, especially those with primary aromatic amino groups, were found to interfere with the colorimetric assay. Naturally occurring urinary substances (but not drugs) interfere to a variable extent with the measurement of PAH by the HPLC method. Predicted creatinine excretion was variable (r 0·748) when compared with [^{14}C]PAH excretion. Calcium, nitrogen and phosphate balances and creatinine clearance were found to be considerably in error.

It is concluded that errors in urine collections in patients receiving TPN in general medical and surgical wards may be both frequent and large. It is therefore suggested that metabolic and nutritional balance studies should include a urine marker such as PAH.

The maternal fat component in the weight gain of pregnant women. By J. V. G. A. DURNIN and F. M. MCKILLOP, *Institute of Physiology, University of Glasgow, Glasgow G12 8QQ*

The 'normal' weight gain of women at the end of pregnancy is supposed to be about 12 kg, relative to their pre-pregnant body-weight, and of this 12 kg about 3·5 kg is thought to represent extra fat (Hytten & Leitch, 1971). This is assumed to be a physiological reaction to pregnancy to provide an extra store of energy for the needs of late pregnancy and of lactation. It also comprises almost 50% (about 160 MJ (38000 kcal)) of the total extra energy requirements of pregnancy.

As part of a longitudinal study of the energy requirements of pregnancy, the changes in body mass and in maternal fat mass were measured on eighty-eight pregnant women in Glasgow. Measurements were made initially on these women either in the pre-pregnant state (twenty-two women) or early in pregnancy (between 8 and 12 weeks gestation). Body mass was recorded daily on the women.

Body fat was assessed from skinfold thicknesses and body-weight changes. Owing to alterations in the composition of the fat-free mass in late pregnancy caused by the addition of approximately 7 litres of fluid, the estimates of fat gain were calculated from post-partum measurements. Initially, the mean fat gain was calculated from skinfold thicknesses and body-weight changes 4 weeks after delivery on the assumption that by this time all extra body fluids would have been lost and any extra weight remaining would be mainly body fat. The differences between these estimates and those done in early pregnancy at 10 weeks gestation showed a fat gain of 1·7 kg by skinfold thicknesses and 2·2 kg by body-weight changes. However, observation of daily post-partum weight losses on a sub-group of twenty-four women showed that by 2 weeks post-partum, the great majority of extra fluid and uterine mass appears to have been lost. This may therefore be a more appropriate time to make estimates of fat gain. Fat gain calculated from measurements at 2 weeks post-partum gave a mean value of about 2·2 kg.

These gains are considerably less than the theoretical fat gain of 3·5 kg, although the total weight gain of 12·1 kg (from an initial weight at 10 weeks of 57 kg), birth weight of 3·4 kg and placenta weight of 0·64 kg for the Glasgow group were almost identical to the theoretical values of Hytten & Leitch (1971).

We think it is possible that the theoretical value is perhaps not the physiological norm.

This study was part of a large-scale project on the energy requirements of pregnancy, financed by the Nestlé Foundation.

Hytten, F. E. & Leitch, I. (1971). *The Physiology of Human Pregnancy*. Oxford: Blackwell.

Urinary mercapturic acid outputs of severely malnourished children. By D. D. RAMDATH and M. H. N. GOLDEN, *Wellcome Trace Element Research Group, Tropical Metabolism Research Unit, University of the West Indies, Kingston, Jamaica*

Mercapturic acids (MA) are a group of compounds produced during detoxification processes in vivo. The extent to which a cell is exposed to toxic substances is therefore reflected by increased urinary MA output. Toxins are effectively neutralized by the formation of a conjugate with glutathione (GSH); this reaction is catalysed by glutathione S-transferase (*EC* 2.5.1.18; GST) (Jakoby, 1978).

Severely malnourished children have poorly functioning antioxidant systems and are often exposed to increased oxidant stress, thus their detoxification systems are probably being overworked (Golden & Ramdath, 1987). This is supported by our previous finding that erythrocyte GST activity is induced in malnourished children (Charley *et al.* 1986). As MA are the end-product of GST activity we reasoned that the urinary MA output should also be elevated. This may assist in explaining the low levels of GSH found in children with oedematous malnutrition (Ramdath & Golden, 1986), and provide a quantitative measure of the total body load of exogenous and endogenous carbonyl-like toxins.

Urinary outputs (24 h) of MA and creatinine were measured in nineteen severely malnourished children, on admission and at discharge from hospital. Three healthy children served as controls. The results on admission are shown in the Table.

	Control		Malnourished		Marasmus		Marasmus–Kwashiorkor		Kwashiorkor	
MA output	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
μmol/kg body-wt	1·2	0·1	4·2	0·1	3·4	0·8	5·1	1·2	4·4	1·1
μmol/mmol creatinine	5·8	0·9	37·3	5·7	26·0	7·1	57·3	13·0	32·6	7·3

The high MA levels were maintained at discharge. Admission creatinine levels were significantly lower than normal; they were increased at discharge.

We conclude that the urinary MA output of severely malnourished children is significantly higher than normal. This is probably indicative of their burden of toxins. The high MA output at discharge is possible as a result of oxidant stress imposed by the high polyunsaturated fat content of the recovery diet.

This work was supported by the Wellcome Trust.

Charley, L., Ramdath, D. D., Golden, M. H. N. & Foreman, J. (1986). *West Indian Medical Journal* **35, Suppl.**, 50.

Golden, M. H. N. & Ramdath, D. D. (1987). *Proceedings of the Nutrition Society* **46**, 53–68.

Jakoby, W. B. (1978). *Advances in Enzymology* **36**, 383–414.

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The involvement of chloride ions in the hypersecretory response to differing levels of cholinergic stimulation in the starved rat jejunum. By A. YOUNG and R. J. LEVIN, *Department of Physiology, The University, Sheffield S10 2TN*

Starvation for 72 h hypersensitizes rat jejunum to secretagogue challenge both in vivo and in vitro (Levin & Young, 1985; 1986) resulting in greater fluid and electrolyte secretion. The identity of the ionic species responsible for the elevated electrogenic secretion is, however, unknown. The rat jejunum primarily secretes chloride, and we investigated the role of chloride ions in the hypersecretory starved state using iso-osmolar gluconate as its replacement in the bicarbonate buffer used. Isolated jejuna from fed and 72 h fasted rats were incubated in normal bicarbonate buffer or chloride-free bicarbonate buffer. Bethanecol, a muscarinic cholinergic agonist, was used as a secretory stimulus at doses of 50, 100, and 500 μM and 1 mM.

We also investigated whether the hypersecretory response to cholinomimetics was present over a wide range of stimulatory levels. A dose–response curve was constructed using bethanecol at doses ranging from 1 μM to 20 mM for both fed and 72 h fasted rats. Electrogenic ion secretion measured as the short-circuit current (Isc) was obtained using standard techniques (Baldwin & Levin, 1985).

Replacing the chloride in the incubating buffer with gluconate reduced the increases induced by bethanecol, this reduction being approximately 85% at each of the doses used both for fed and fasted jejuna. Although basal Isc did not differ between fed and fasted jejuna when using normal bicarbonate buffer, chloride replacement reduced the basal Isc to a greater extent in the fed (−72%, n 16) than in the fasted jejuna (−60%, n 19), indicating that in the fed state more of the basal electrogenic secretion is chloride dependent ($P<0{\cdot}05$, Student's unpaired t test).

At each dose of bethanecol the starved intestine gave a markedly elevated response compared with fed controls. The ED_{50} (the dose required to give 50% of maximal response) did not radically differ between fed and fasted jejuna, being approximately 92 μM for fed and 64 μM for fasted. This suggests little change in receptor affinity but possibly changes in receptor population.

Thus the hypersensitivity to cholinomimetics seen following starvation occurs over a spectrum of stimulus levels, and results primarily in an elevation in electrogenic chloride secretion.

We gratefully acknowledge financial support from the British Digestive Foundation.

Baldwin, D. & Levin, R. J. (1985). *IRCS Medical Science* **13**, 269–270.
Levin, R. J. & Young, A. (1985). *Journal of Physiology* **365**, 109P.
Levin, R. J. & Young, A. (1986). *Journal of Physiology* **378**, 23P.

Jejunal water transport in man: effects of bicarbonate or tartrate. By J. B. LEIPER and R. J. MAUGHAN (introduced by R. F. GRIMBLE), *Department of Environmental and Occupational Medicine, University Medical School, Foresterhill, Aberdeen AB9 2ZD*

In the normal human small intestine, bicarbonate is as effective as glucose in stimulating sodium and water absorption from solutions which contain no other actively transported solute (Sladen & Dawson, 1968). The bicarbonate effect appears to be linked to an active Na–hydrogen exchange process and is independent of the pH of the luminal contents (Turnberg *et al.* 1970). The active co-transport of glucose and Na enhances water absorption, but there is little direct evidence that bicarbonate has an additive effect on absorption from glucose–electrolyte solutions in man. We have examined, using a steady-state perfusion technique in the normal human jejunum (n 8), net water and solute transport from two glucose–electrolyte solutions one of which (solution A) contained bicarbonate (23 mmol/l) and the other (solution B) tartrate (23 mmol/l). We have previously shown (Leiper & Maughan, 1986) that moderately hypotonic solutions (≃200 mosmol/kg) maximize water absorption, and both test solutions in the present study had osmolalities of 210 mosmol/kg. A multilumen tube, incorporating a 150 mm mixing segment and a 300 mm test segment was positioned with the perfusion port just distal to the ligament of Treitz. In the mixing segment the mean (SD) glucose concentration of solution A decreased from 94 (5) to 68 (16) mmol/l and pH dropped from 7·9 (0·2) to 5·9 (1·6). The Na concentration of solution B increased (42 (1) to 59 (15) mmol/l), glucose concentration decreased (92 (3) to 58 (16) mmol/l) and pH remained at 3·8 (0·9).

In the test segment, net water absorption from solution A (1410 (620) ml/m per h) was higher than that from solution B (970 (670) ml/m per h, $P<0·05$). There was greater absorption of glucose from solution A (116·6 (52·2) mmol/m per h) than from solution B (77·8 (38·2) mmol/m per h, $P<0·02$). During perfusion the pH of the luminal contents increased in the test segment, but the pH of solution A (6·6 (0·8)) was higher than that of solution B (4·4 (0·8), $P<0·001$).

These results suggest that addition of bicarbonate to glucose–electrolyte solutions does increase net water absorption from the normal human jejunum. Glucose uptake, but not Na uptake, also increased in the presence of bicarbonate; it is therefore unclear whether the increased water absorption was due directly to bicarbonate uptake or to a buffering effect enhancing the active transport of glucose.

This study was approved by the Local Ethical Committee and supported by Rorer Health Care (UK) Ltd.

Leiper, J. B. & Maughan, R. J. (1986). *Journal of Physiology* **378**, 95P.
Sladen, G. E. & Dawson, A. M. (1968). *Nature* **218**, 267–268.
Turnberg, L. A., Fordtran, J. S., Carter, N. W. & Rector, F. C. (1970). *Journal of Clinical Investigation* **49**, 548–556.

Effect of combined vitamin E and protein deficiency on the hepatic response of the rat to the *Escherichia coli* endotoxin. By ASMA B. OMER, P. C. BATES and D. J. MILLWARD, *Nutrition Research Unit, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

Although vitamin E deficiency reduces the capacity to deal with oxidative stress, we were unable to demonstrate any clear disadvantage of the deficiency in rats exposed to either protein deficiency or acute endotoxaemia (Omer *et al.* 1986). We report here further investigations of the response to the *Escherichia coli* endotoxin in rats which were both protein- and vitamin-E-deficient. Weanling rats (body-weight 50 (SE 3) g) were given the following diets with (+E) and without (−E) vitamin E: (A) 200 g protein/kg for 16 d; (B) as (A) plus 16 d on a diet of 30 g protein/kg; (C) as (A) plus 16 d on a diet of 5 g protein/kg. The dietary fat source was linoleic acid (50 g/kg). These groups were then treated with the *E. coli* endotoxin (LPS: strain 0127:B8) at 1, 2, 3 and 4 mg/kg body-weight as previously described (Jepson *et al.* 1986). Measurements were made of food intake, hepatic protein mass and protein synthesis, hepatic lipid peroxidation and the changes in plasma zinc and albumin.

Vitamin E deficiency had no effect on the suppression of food intake induced by the endotoxin in any of the dietary groups (50% reduction in (A) and 65% reduction in (B) and (C)). It also had no effect on the rates of hepatic protein synthesis which were reduced by the protein-deficient diets in the untreated rats (7·5 (SE 0·21), 3·34 (SE 24) and 3·12 (SE 20) g protein/kg body-weight in (A), (B) and (C) respectively). All groups exhibited an increase in hepatic protein content and protein synthesis in response to the endotoxin, although the response was greater in the three vitamin-E-deficient groups. Thus, total hepatic protein synthesis was increased by a maximum of 85, 77, and 74% in groups (A), (B) and (C) (−E), compared with 42, 52 and 58% in the corresponding +E groups, although the differences were not significant for group (C). These increases in hepatic protein synthesis in response to the LPS were accompanied by a similar fall in plasma albumin in both −E and +E groups, a fall in lipid peroxidation in the liver of the −E groups and reduced levels of plasma Zn which was most pronounced in the −E, (C) group.

Thus, in contrast to our previous preliminary studies, it is clear that vitamin E deficiency is associated with an increased stimulation of hepatic protein synthesis and decreased plasma Zn in response to endotoxin. This suggests that any hepatic damage induced by vitamin E deficiency does not reduce the ability to synthesize acute-phase proteins.

Jepson, M. M., Pell, J. M., Bates, P. C. & Millward, D. J. (1986). *Biochemical Journal* **235**, 329–336.
Omer, A. B., Bates, P. C. & Millward, D. J. (1986). *Proceedings of the Nutrition Society* **45**, 114A.

The influence of quantity and quality of protein on rodent malaria. By N. P. M. BAKKER, *University of Amsterdam,* W. M. C. ELING, *Catholic University of Nijmegen,* A. DE GROOT, *University of Utrecht/Leyden,* E. J. SINKELDAM*, *TNO CIVO-Institutes, Zeist,* and R. LUYKEN, *Royal Tropical Institute, Amsterdam, The Netherlands*

Low-protein diets (0, 40 or 80 g casein/kg) have been reported to inhibit the development of *Plasmodium berghei* infections in young rats (Ederisinghe *et al.* 1981). We obtained comparable results using potato protein. Groups of ten rats (mean body-weight 50 g, CPB Wistar) were placed on isoenergetic diets containing 40, 80, or 160 g potato protein (Protamyl PF, AVEBE, Foxhol, The Netherlands) and 0·6 g *p*-aminobenzoic acid (PABA)/kg diet. Mortality after infection with 30000 parasites (*P. berghei* KL73) was 24, 50 and 100% in the respective groups. Death was preceded by paralysis which was associated with a dramatic decrease in body temperature, and haemorrhages in the brain. The parasitaemia and reticulocyte count in blood correlated with the protein concentration in the diet.

A similar model was used to study simultaneously the effect of protein quantity and quality. Simulating the cereal–pulse mixture, often recommended for tropical areas, groups of rats received the following protein mixtures in their diets, supplemented with PABA.

Group . . .	A	B	C	D
Maize gluten protein (g/kg)	30	60	20	40
Soya-bean protein (g/kg)	10	20	10	20
Milk protein† (g/kg)	–	–	10	20
Mean body-weight (day 8 after infection) (g)	55·9	69·0	58·9	79·2

†Refit HPA, DMF, Veghel, The Netherlands.

Mortality after infection, preceded by paralysis, was observed in group D only (60% of animals). It is remarkable that death occurred only in the group with the largest gain of body-weight.

The mechanism of suppression of *P. berghei* malaria by low-protein or by a vegetarian protein mixture, or both, and the exacerbation by milk-protein supplementation need further study.

This study was supported by NIZO (Netherlands Institute for Dairy Research, Ede, The Netherlands). The valuable advice of H. J. v.d. Kaay and B. Mons is gratefully acknowledged.

Ederisinghe, J. S., Fern, E. B. & Targett, G. A. T. (1981). *Transactions of the Royal Society of Tropical Medicine and Hygiene* **75**, 591–593.

*For correspondence.

Vitamin E deficiency in the rat and the response to malarial infection. By A. M. OMWEGA, P. C. BATES and D. J. MILLWARD, *Nutrition Research Unit, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

Deficiencies of nutrients which protect against oxidative stress can cause oxidant-induced haemolysis of erythrocytes and could protect against the development of malaria, if erythrocyte lysis inhibited the development of parasitaemia. Whilst this effect has been confirmed in malaria-infected vitamin-E-deficient mice (Eckman *et al.* 1976), the reduced parasitaemia in riboflavin-deficient rats does not reduce mortality (Thurnham, 1986). We report here a further examination of this hypothesis in the vitamin-E-deficient rat infected with *Plasmodium berghei.*

Male rats (mean body-weight 42 g, *n* 36) were fed on a purified diet (200 g casein/kg) with (+E) or without (−E) vitamin E for 2 weeks. At 110 g body-weight, rats in each dietary group were sub-divided into three groups for further treatment: parasitized (+E:P and −E:P), pair-fed (+E:PF and −E:PF) and *ad lib.*-fed (+E:AL and −E:AL). The :P groups were infected (intravenously) with 10^7 *P. berghei* parasites and monitored for the next 2 weeks, with measurements of body-weight gain, bone length growth, food intake, parasitaemia, anaemia, rectal temperature (at 10.00 hours), selected hormone concentrations, organ size and muscle protein synthesis (measured by the large dose L-[4-H^3]phenylalanine method).

Effect of vitamin E deficiency on the response of the rat to P. berghei

	Body-weight (g)		Food intake (g/kg $W^{0.75}$)		Haemoglobin (g/l)		Parasitaemia (% erythro-cytes)		Rectal temperature (°)		Muscle protein synthesis (%/d)	
Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
+E:AL	178	16	3·9	0·4	193	17			36·6	0·4	11·8	1·2
+E:PF	138	11	1·7	0·4	223	7			37·1	0·3	7·6	1·0
+E:P	127	16	1·7	0·5	41·2	12	60·4	6·7	35·0	1·8	3·5	1·5
−E:P	128	24	1·4	0·6	29·2	8	21·4	2·4	32·7	1·1	2·1	0·3
−E:PF	148	11	1·4	0·6	215	3			37·2	0·4	9·0	1·7
−E:AL	208	18	4·2	0·2	193	23			36·8	0·3	12·4	0·3

W, body-weight.

As shown the −E:P group did have a reduced parasitaemia, but this was not protective since this group exhibited similar anorexia, growth suppression and inhibition of muscle protein synthesis, and were more hypothermic and anaemic. Thus vitamin E deficiency is not protective in that the reduced parasitaemia is more than offset by the severity of the anaemia and hypoxia induced by the haemolysis and the other systemic responses to the infection.

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Thurnham, D. I. (1986). In *Proceedings of the XIII International Congress of Nutrition 1985*, pp. 129–131 [T. G. Taylor and N. K. Jenkins, editors]. London: John Libbey.

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The effect of β_2-agonists on the endogenous nitrogen loss of sheep. By F. D. DeB. HOVELL, D. J. KYLE and P. J. REEDS, *Rowett Research Institute, Aberdeen AB2 9SB* and D. H. BEERMAN, *Department of Animal Science, Cornell University, Ithaca, New York, USA*

The effect of β_2-agonists on increasing nitrogen retention in a wide range of animals is well documented. Work at the Rowett with rats showed that the effect with normal growing animals is mediated by a reduction of protein degradation, with little or no effect on protein synthesis (Reeds *et al.* 1986). We reasoned therefore that β_2-agonists might reduce the endogenous N loss of animals given energy but no protein.

Ten sheep of between 32 and 48 kg live-weight were used. They were adapted to alimentation by the intragastric infusion of all nutrients. Endogenous urinary N loss was measured for a 10 d period when volatile fatty acids (VFA) were infused into the rumen, and water but no protein was infused into the abomasum. Eight of the sheep were given 150 μg clenbuterol (3·6 (SE 0·11) μg/kg) and six sheep 1·5 mg (40 (SE 2·3) μg/kg) cimaterol daily, infused into the abomasum. Half the sheep in each treatment group received the β_2-agonist during the first 5 d, and half during the second 5 d of the 10 d N-free period (the other 5 d being the control period). The results presented in the Table are means of the last 4 d of these 5 d sub-periods.

	VFA infused (kJ/kg $W^{0.75}$ per d)	Live wt (kg $W^{0.75}$)	Urinary N (mg/kg $W^{0.75}$ per d)	Urinary creatinine (mg/kg $W^{0.75}$ per d)
Control	451	16·3	214	53·0
Clenbuterol	447	16·3	185	52·7
SEM	4	0·40	6·4	1·0
Control	458	15·3	234	54·7
Cimaterol	442	15·3	220	53·8
SEM	10	0·72	5·0	4·2

W, body-weight.

Clenbuterol significantly ($P<0·05$) reduced the urinary endogenous N loss by 14%. Cimaterol also reduced endogenous N loss (by 6%) although the effect was not significant statistically. These effects were apparent irrespective of whether the drug was given during the first or second 5 d sub-period. Neither drug had any effect on creatinine excretion. These results can be interpreted as the drugs acting by reducing body protein degradation with the effect that the amount of amino acid lost to the pool by oxidation is reduced.

The authors are grateful to Boehringer–Ingelheim for the gifts of clenbuterol and cimaterol.

Reeds, P. J., Hay, S. M., Dorward, P. M. & Palmer, R. M. (1986). *British Journal of Nutrition* **56**, 249–258.

The use of [^{3}H]proline to determine rates of collagen and noncollagen protein synthesis in skeletal muscle of lambs: effects of growth hormone. By J. M. PELL, *The Animal and Grassland Research Institute, Reading RG2 9AQ,* P. C. BATES, *Nutrition Research Unit, London School of Hygiene and Tropical Medicine, London NW1 2PE* and R. J. MCANULTY and G. J. LAURENT, *Biochemistry Unit, Cardiothoracic Institute, London SW3 6HP*

The large dose method of measuring rates of collagen and noncollagen protein synthesis using 5-[^{3}H]proline (Laurent, 1982) was adapted for rapidly growing lambs. Preliminary experiments were performed to determine a dose and specific radioactivity of [^{3}H]proline which would flood all tissue pools while maintaining a constant precursor pool specific radioactivity and linear incorporation of radiotracer into protein: 5 mCi [^{3}H]proline with 0·288 mol unlabelled proline per lamb injected intravenously in saline (9 g sodium chloride/l). Slaughter was 3 h after injection of the radiolabel. Protein synthesis rates were determined in biceps femoris muscle (mainly red-type fibres) and semitendinosus muscle (mainly white-type fibres) in five sets of 21-week-old twin lambs; one twin acted as control (C) and the other (G) was treated with growth hormone (GH; 0·1 mg/kg per d, subcutaneously from 9 weeks of age).

Daily mean live-weight gain was increased by GH treatment (C 282 (SE 13), G 338 (SE 8) g/d; $P<0·05$). Calculated muscle growth was significantly increased in G (biceps femoris 0·55 (SE 0·05) to 0·65 (SE 0·07) %/d; semitendinosus 0·55 (SE 0·05) to 0·64 (SE 0·07) %/d; $P<0·05$).

		Non-collagen protein concentration (mg/g)		Collagen concentration (mg/g)		Non-collagen protein synthesis (%/d)		Collagen protein synthesis (%/d)	
Muscle type		C	G	C	G	C	G	C	G
Biceps femoris	Mean	116	114	5·7	6·2	4·1	5·4*	0·57	0·74*
	SE	2	2	0·7	1·0	0·6	0·7	0·05	0·06
Semitend-inosus	Mean	120	117	3·4	3·9	4·0	4·3	0·74	0·70
	SE	2	1	0·2	0·3	0·3	0·2	0·07	1·10

Significantly different from C: *$P<0·05$.

The mechanism of the anabolic action of GH appears to be via increased rates of protein synthesis. However, red- and white-type muscle fibres respond differently to GH.

Laurent, G. J. (1982). *Biochemical Journal* **206**, 535–544.

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The physical properties of butter produced from the milk of Friesian or Jersey cows given diets based on either barley or oats. By W. BANKS, J. L. CLAPPERTON, D. D. MUIR and ANNE K. GIRDLER, *The Hannah Research Institute, Ayr KA6 5HL*

When oats replace barley in the diet of dairy cows, because the concentration of oil is much higher in the oats than in the barley, the proportion of saturated fatty acids in the milkfat is reduced and that of the mono-unsaturated fatty acids (especially 18:1) is increased (Martin & Thomas, 1987). These changes may be of nutritional benefit and this work was carried out to investigate the effects of these changes on the physical properties of the butter.

Four Friesian cows and four Jersey cows in the middle of their lactation were given a diet based on hay and molassed sugar-beet pulp. A mixture of barley and soya-bean meal was added for 3 weeks and then 500 g soya-bean oil/d were added for a further 3 weeks. The barley was then replaced by oats and the same two experimental periods were carried out. Butter was prepared at the end of each period and the extrusion value of the butter and the melting behaviour of the anhydrous fat were measured (see Table).

	Friesian				Jersey			
	Barley		Oats		Barley		Oats	
	No oil	Oil	No oil	Oil	No oil	Oil	No oil	Oil
Fatty acids (mmol/mol)								
Saturated (4:0–18:0)	774	605	702	582	827	656	774	661
Mono-unsaturated (16:1+18:1)	205	354	280	382	155	308	210	307
Polyunsaturated (18:2+18:3)	21	42	19	36	18	36	16	32
Physical properties								
Extrusion value (kg)	4·70	1·00	1·22	1·04	6·80	2·96	5·40	2·68
Proportion solid at 5° (per 1000)	741	634	585	604	843	645	739	673

Compared with the barley-only diet, both the oat diet and the addition of oil reduced the proportion of the saturated and increased that of the mono-unsaturated fatty acids. The milkfat of the Jersey cows was less susceptible to the effects of dietary manipulation. Decreases in the extrusion value and in the proportion of fat solid at 5° show the enhanced spreadability of the butter.

Martin, P. A. & Thomas, P. C. (1987). *Proceedings of the Nutrition Society* **46**, 114A.

Influence of underfeeding for 7 d on the physiological response to food ingestion. By P. I. MANSELL and I. A. MACDONALD, *Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Nottingham NG7 2UH*

Underfeeding appears to be associated with a reduction in sympathetic nervous system (SNS) activity in a variety of situations (Landsberg & Young, 1985). Further, the physiological responses to food ingestion are mediated in part by an associated increase in SNS activity. We have performed the following study to determine whether these responses are modified following a period of underfeeding in normal subjects.

Six healthy female volunteers (age 21–44 years, body mass index 20·3–24·8 kg/m^2) underwent a 7 d period of underfeeding at 60 kJ/kg ideal body-weight per d. Subjects were studied in the post-absorptive state while resting supine in a thermoneutral room at 30°. Experiments were performed in the normally fed and underfed states. After a 30 min period for baseline measurements, subjects consumed 30 kJ/kg body-weight of a liquid formula diet (Fortisip plus®, Cow and Gate) and the study was continued for a further 80 min. Measurements were made of metabolic rate (MR), respiratory exchange ratio (RER), heart rate (HR), blood pressure (BP) and calf blood flow (CBF). 'Arterialized' venous blood was analysed for glucose (GLU), glycerol (GLY), lactate (LAC) and β-hydroxybutyrate (βOHB) and plasma concentrations of free thyroxine (T_4), free 3,5,3′-triiodothyronine (T_3), noradrenaline (NA) and adrenaline (ADR).

Changes in baseline values of these variables are shown in the Table; there were no significant changes in baseline BP, CBF, GLY, T_4 or ADR.

Baseline variables before and after underfeeding for 7 d

State	Body-wt (kg)	MR (kJ/min)	RER	HR (beats/min)	T_3 (pmol/l)	GLU (mmol/l)	LAC (mmol/l)	βOHB (mmol/l)	NA (mmol/l)
Fed	63·2	4·21	0·806	65·1	5·37	4·52	0·800	0·055	1·12
Underfed	61·2**	3·91***	0·732***	60·1*	4·38*	4·36*	0·528**	0·229**	0·88

Significantly different from fed state: *P<0·05; **P<0·01; ***P<0·001.

Following food ingestion, HR rose, BP showed little difference and CBF rose; these changes did not differ between the fed and underfed states. The thermic effect of food (TEF) in the fed state averaged 0·297 kJ/min over 80 min and was less than that in the underfed state, 0·331 kJ/min (P<0·05, ANOVA). Plasma NA rose with food ingestion but the increase was not altered by the state of nutrition.

Underfeeding normal subjects for 7 d produced a considerable alteration in basal metabolism with an unexpected slight increase in the TEF.

The study was approved by the Medical School Ethical Committee and was funded by the Wellcome Trust.

Landsberg, L. & Young, J. B. (1985). In *Neuroendocrine Perspectives* no. 4, pp. 191–218 [E. E. Muller, R. M. MacLeod and L. A. Frohman, editors]. Amsterdam: Elsevier Science Publishers.

The implications of creatine–creatinine interconversions in man. By N. J. FULLER *Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ* and M. ELIA, *Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL*

Creatinine excretion has been used to assess nutritional status, muscle mass and the completeness of urine collections. However, it can also be affected by meat ingestion, pyrexial illness, renal function and creatine–creatinine interconversions in urine. This study was concerned with (a) assessing the variability in the ratio creatine:creatinine in fresh samples of urine obtained in different clinical and nutritional circumstances; and (b) the rate of creatine–creatinine interconversions over a wide range of pH (1–10) and temperature (<0–40°), to cover possible storage conditions of urine.

Condition	Creatine:creatinine molar ratio (range)	Reference
Normal adults	0–0·41	Present study
Children	0·09 –2·04	Clark *et al.* (1951)
Premature infants	0·04 –1·33	Present study
Trauma plus elective surgery	0·31 –1·38	Threlfall *et al.* (1981)
Acute infective illness	0·001–0·37	Present study
Starvation	0·61 –0·77	Present study
Total parenteral nutrition	0·003–0·044	Present study
Thyrotoxicosis	0·10 –1·25	Tierney & Peters (1943)
Muscular disorders	0·29 –2·00	Van Pilsum & Wolin (1958)

The results shown in the Table, along with some results from other studies, emphasize the wide variability in the creatine:creatinine molar ratio (0–2·0) in both normal subjects and in those with disease. The rate of conversion of creatine to creatinine was greatest at pH 3·5–4·0 (about 22%/d at 40°) which may occur with the use of acid preservatives. The rate of conversion of creatinine to creatine was maximal at pH 5·0–6·0 (about 5%/d at 40°). A rise in body temperature from 37 to 40° may increase creatinine production from both creatine and creatine phosphate by about 18%, possibly changing the relation between creatinine production and muscle mass. Temperature was found to have little effect on the equilibrium position, which was increasingly displaced towards creatinine as solutions became more acid (>95% below pH 4), and associated with a creatine: creatinine molar ratio of about 1 above pH 6.

It is concluded that there may be major changes in the creatinine content of urine during inappropriate storage conditions.

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Threlfall, C. J., Stoner, H. B. & Galasko, C. S. B. (1981). *Journal of Trauma* **21**, 140–147.

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Van Pilsum, J. F. & Wolin, E. A. (1958). *Journal of Laboratory and Clinical Medicine* **51**, 219–223.

Fat metabolism in human skeletal muscle and in the whole body after ingestion of a mixed meal. By M. ELIA, *Dunn Clinical Nutrition Centre, Cambridge CB2 1QL,* A. SCHLATMANN and P. FOLMER, *Department of Human Nutrition, Agricultural University, Wageningen, The Netherlands* and A. GOREN, *Faculty of Medicine, University of Ankara, Turkey*

The present study aimed to obtain integrated information about fat metabolism in skeletal muscle and in the whole body using (a) indirect calorimetry and timed urine collections (plus estimates of changes in the size of the urea pool), (b) concentration of metabolites in arterialized and deep venous blood from the forearm, (c) forearm plethysmography.

Before ingestion of a meal containing 3275 kJ (47·3, 39·4 and 13·3% energy from carbohydrate, fat and protein respectively), energy expenditure (14 h after an overnight fast) was 4·92 (SEM 0·14) kJ/min and fat oxidation 3·18 (SEM 0·14) kJ/min (64·8 (SEM 3·1)% of total energy expenditure). After meal ingestion, energy expenditure (measured over 4 h) increased to a mean value of 5·64 (SEM 0·19) kJ/min ($P<0·01$) whilst fat oxidation decreased. Uptake of non-esterified fatty acids (NEFA) by muscle also decreased (see Table).

	Period before and after meal (h)						
	−1	0	Meal	+1	+2	+3	+4
NEFA (P):							
μmol/l	533	523		175**	171**	316	436
flux	1130	1170		−110***	−10**	1030	1290
Triglyceride (P):							
μmol/l	663	673		843**	1159***	1440***	1451***
flux	–	–		–	–	–	–
Glycerol (B):							
μmol/l	49	51		33***	37*	49	59
flux	−190	−250		−190	−160	−270	−80
Total ketone bodies (B):							
μmol/l	205	188		183	153	142	140
flux	1210	890		800	560	1070	940
Insulin (P):							
mU/l	6·8	6·4		58·2***	21·6**	20·7*	9·6

P, plasma; B, whole blood. Flux is in nmol/l muscle per min.

Significantly different from the mean of the two pre-prandial measurements: *$P<0·05$, **$P<0·01$, ***$P<0·001$.

The plasma triglyceride concentration increased after the meal in proportion to the basal triglyceride concentration. The rate of glycerol release by the forearm was inversely related to the post-prandial triglyceride concentration.

The results suggest that the rate of net fat oxidation in the whole body, and uptake of fat by muscle, are both decreased after ingestion of a mixed meal. They also suggest suppression of net lipolysis in the whole body but continued hydrolysis of triglyceride in the muscle bed. Whereas in the pre-prandial period circulating NEFA is a more important fuel for muscle than triglyceride, in the early post-prandial period triglyceride (probably mainly circulating triglyceride) becomes a more important fuel than NEFA. Insulin is likely to play an important role in mediating many of these effects.

The effect of acute ingestion of a carbohydrate-based meal on the maximum running performance of rats. By IONA SMEATON, *Physiology Department, Guy's Hospital UMDS, London Bridge, London SE1 9RT*

The present study was designed to test whether the type of carbohydrate in a pre-exercise meal affected maximum running performance in adult male Wistar rats.

A group of twenty rats (group 1) was trained to run on a motorized treadmill. The time until fatigue, when the rats touched the electrical stimulus at the rear three times, was measured and the amount of work done calculated. During this time the rats were given a complete diet containing 620 g maize starch/kg. After this training period the rats were divided into four groups, matched for weight and given, double blind, a diet containing 620 g/kg of (A) maize starch (control), (B) glucose, (C) fructose or (D) sucrose on five overnight feeding sessions over a 2·5 week period. The work output (E_T) during the runs following these test feeds was determined. On the interval days between these five test runs all the rats were given the maize-starch diet overnight and run as before, and the work output (E_I) calculated.

In order to standardize the amount of each diet eaten and the time between ingestion and exercise, a second group of twenty rats (group 2) underwent the same training and selection procedures, but this time were fasted overnight before the five test runs. These rats were then given 10 g portions of diet A, B, C or D 4 h before exercise. E_T was determined for each rat. Work outputs during the interval runs while on the maize-starch diet (E_I) were determined for this group of rats also. The results for both groups are seen in the Table.

		Work output (J)							
Test diet group		Group 1, overnight feed				Group 2, single meal feed			
		E_I		E_T		E_I		E_T	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
A	Maize starch	807	102·2	818	114·3	587	314·8	588	322·1
B	Glucose	795	75·7	869	72·1	667	251·5	698	291·5
C	Fructose	765	137·0	892	55·7	597	292·4	618	332·0
D	Sucrose	777	177·7	881	132·6	575	294·8	568	284·2

No significant difference was found between the work done during exercise to fatigue on the control maize-starch diet and on any of the test carbohydrate diets, given either as an overnight feed or as a single meal 4 h before exercise. An additional observation within these two populations of rats was that 27% did not significantly increase their run time after the initial training week.

I am grateful to the Health Promotion Research Trust for providing a research grant.

Nutrient intake of six elite women athletes. By IONA SMEATON, *Physiology Department, Guy's Hospital UMDS, London Bridge, London SE1 9RT*

Six elite female athletes, aged 17–28 years, completed 7-d weighed food intakes. The intake data were checked at intervals by a dietician, and the nutrient composition of the diet determined using values from Paul & Southgate (1978).

The subjects had a mean daily intake of 9·9 MJ (2360 kcal; range 1909–2621 kcal) and their body-weights ranged from 49·6 to 70·0 kg. Within their self-selected diets the intake of a number of nutrients was found to be below the UK recommended daily amounts (RDA) (Department of Health and Social Security, 1979).

Nutrient	RDA for very active ♀	Athletes' intake		
		Mean	Range	No. below RDA
Vitamin A (retinol equivalents)	750	1213	571–3102	2
Vitamin E (α-tocopherol equivalents)	10*	5·8	4·2–8·8	6
Vitamin C (mg)	30	88·7	22·4–157·0	1
Pyridoxine (mg)	2*	1·2	0·9–1·7	6
Vitamin B_{12} (μg)	3*	4·6	1·6–9·0	2
Total folate (μg)	300	190·4	114–362	5
Biotin (μg)	300†	23·4	17·2–35·1	6
Iron (mg)	12	13·5	11·0–16·4	2
Magnesium (mg)	300*	320·7	202–391	2
Zinc (mg)	15*	11·2	8·2–15·0	5
Copper (mg)	2†	1·64	1·5–2·1	5

*Food and Agriculture Organization (1985).
†Office of the Federal Register (1984).

From this small sample of athletes it would appear that in the absence of professional advice it is difficult to consume a diet which provides all vitamins and minerals at RDA levels. Whether altering their diet to provide vitamins and minerals at a level to meet RDAs would improve their performance is not known.

I am very grateful to the volunteers in this study and to the Health Promotion Research Trust for a grant.

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The effect of dietary fats on *N*-nitrosamine formation in man. By F. W. WARD and M. E. COATES, *Robens Institute, University of Surrey, Guildford GU2 5XH* and I. MACDONALD and A. SIMS, *Guy's Hospital UMDS, London Bridge, London SE1 9RT*

Experiments in rats have shown that inclusion of 100 g fat/kg diet depresses formation of *N*-nitrosamines from orally ingested nitrate. Butterfat was consistently more effective than vegetable oils (Ward & Coates, 1987). To investigate whether or not the same is true for man, two groups of young adult volunteers consumed a low-fat, high-nitrate diet for 1 week, followed by a similar diet supplemented with about 4000 kJ fat/d for a further week. They were allowed to eat their normal diet within the limitations of the experiment and were provided with a daily allowance of high-nitrate foods and a capsule containing 300 mg $NaNO_3$. During the 2nd week, one group was given butter, cream, and cheese. The other received margarine, salad cream, vegetarian cheese, peanut butter and peanuts. Fatty foods other than those issued were forbidden, as were high-fibre products which might inhibit nitrosamine formation.

On the last 4 d of each experimental period, 24 h urine collections were made. Portions of each day's sample from each participant were pooled and their content of *N*-nitrosoproline (NPRO), an indicator of nitrosamine formation, was determined by thermal energy analysis. The results (μg NPRO excreted/d) are given in the Table.

Dairy fats			Vegetable oils		
Subject	Low-fat	High-fat	Subject	Low-fat	High-fat
DM	1·9	0·6	FW	2·9	1·3
BB	2·5	0·8	JS	5·7	1·6
AS	3·3	0·9	GC	5·5	2·2
PD	1·4	1·7	KC	4·6	1·9
JM	8·0	1·4	One did not finish the course		
Mean	3·4	1·1		4·7	1·7
SE	1·2	0·2		0·6	0·1

Combining the results of both groups, excretion of NPRO was significantly lower ($P<0·01$) in the second period when the participants were eating the high-fat diets. Taking each group separately the vegetable oils significantly ($P<0·01$) lowered NPRO excretion. Although the effect of butterfat was of a similar magnitude the variance in this group was high and the difference was not statistically significant.

The results have demonstrated in human subjects the inhibiting effect of dietary fats on *N*-nitrosamine formation although, unlike the observations in rats, butterfat did not prove to be more effective in this respect than vegetable oils.

The authors are grateful to the Joint Committee of the Milk Marketing Board and Dairy Trade Federation for financial support for this project, and to the volunteers who made the work possible.

Ward, F. W. & Coates, M. E. (1987). *British Journal of Nutrition* **58**, 221–223.