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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Thirty-fourth Meeting of the Nutrition Society was held in the Royal Society of Medicine, 1 Wimpole Street, London on Monday, 16 *February* 1987, *when the following papers were read:*

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Evaluation of three commercially available vitamin-A-deficient diets. By M. PEDRICK, R. M. HICKS* and J. TURTON†, *Department of Cell Pathology, School of Pathology, Middlesex Hospital Medical School, London W1P 7LD*

Apparently no proven, commercially available vitamin-A-deficient rodent diet is manufactured in Europe. Accordingly, at our request, SDS Ltd (Witham, Essex) manufactured a low level vitamin A diet from natural ingredients (LVAD); this was calculated to contain 60–120 μg retinol equivalents (RE)/kg and should have produced vitamin A deficiency in 6–15 weeks. A semi-synthetic, low vitamin A diet (SSDi) containing cornflour, safflower oil and essentially vitamin-free casein, calculated to contain less than 15 μg RE/kg, was also manufactured by SDS Ltd as a positive control. Weanling female F344 rats (Olac 1976 Ltd) fed on the LVAD or SSDi diets showed no evidence of vitamin A deficiency assessed by reduced body-weight gain and clinical signs after 20 weeks, and the experiment was therefore terminated. Subsequent analyses demonstrated both the LVAD and the SSDi diets to contain less than 15 μg RE/kg, the threshold limit of detection.

A laboratory-prepared, semi-synthetic, vitamin-A-deficient diet ('Brompton' diet) was kindly provided by Drs P. Jeffery and P. Shields (Brompton Hospital). This diet was based on the formulation of Wise (1982) but modified to contain rice starch, safflower oil and essentially vitamin-free casein as sources of carbohydrate, oil and protein (A. Wise, personal communication). This diet routinely produced vitamin A deficiency in weanling male F344 rats in 6–7 weeks (P. Jeffery and P. Shields, personal communication). It produced evidence of vitamin A deficiency in our weanling female F344 rats in 5–7 weeks when, once again, the LVAD and SSDi diets gave negative results in parallel groups of animals.

Based on these findings, SDS Ltd manufactured a new diet (SSDii) with the same formulation as the Brompton diet. Analysis showed this new diet to contain less than the threshold of detection, i.e. 15 μg RE/kg. The SSDii diet was evaluated against the Brompton diet and produced signs of vitamin A deficiency in 7–8 weeks. Although it took slightly longer to be effective than the Brompton diet, this commercially produced SSDii diet has been satisfactory for our studies, consistently producing vitamin A deficiency in weanling female F344 rats in 7–8 weeks and maintaining them in a deficient state. Rats can be maintained on this diet for at least 30 weeks with low vitamin A supplementation in the drinking water; plasma retinol levels have been consistently less than 50% of control values. Rice starch, safflower oil and essentially vitamin-free casein as sources of carbohydrate, oil and protein are thus suitable starting ingredients for the production of vitamin-A-deficient diets.

We acknowledge the support of the MRC, Roche Products Ltd and SDS Ltd.

Wise, A. (1982). *Archives of Toxicology* **50**, 287–299.

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Studies on a bone-marrow-derived bio-immunomodulator in improving immune response in immunosuppressed, water-soluble-vitamin-deficient mice. By DEBASIS GHOSHAL[1], SAMIR ROY[2] and SUBIR BASU[1], *Departments of* [1]*Chemistry and* [2]*Microbiology, Bose Institute, Calcutta* 700 009, *India*

Inbred Swiss albino mice given a diet deficient in vitamins of the B complex and vitamin C for the first 72 h of weaning (weaning body-weight 10 (SE 2) g) exhibited marked suppression of the development of thymus, spleen and bone marrow. The deficient diet contained (g/kg): starch 700, vitamin-free casein 200, peanut oil 60, mineral mix 30, vitamins A, D, E and K and starch to 10 g. For the control diet, vitamin B complex and vitamin C replaced some of the starch added to the vitamins. The cell mediated immune response (CMIR) as studied by T cell rosettes, and humoral immune response (HIR) studied by the complement fixation test, agglutination reaction and B cell surface-staining with anti-antibody, showed marked suppression in the deficient mice. The phagocytic activity of the neutrophil population, as judged by the skin window test and NBT reduction test, was also hampered. However, there was an initial upsurge of eosinophil that gradually decreased. The reticulo endothelial system of the deficient mice was suppressed as studied by a bacterial clearance test with *Mycobacterium smegmatis*. As a consequence, death occurred due to persistent infections, chiefly of the lung.

	Mean antibody titre	Increase of titre
Control (*n* 6)		
SRBC + TCF	1 in 4	—
SRBC + BIM	1 in 8	1 fold
Deficient (*n* 6)		
SRBC + TCF	1 in 2	—
SRBC + BIM (injected 48 h after antigen)	1 in 16	3 fold
SRBC + BIM (injected 24 h after antigen)	1 in 128	6 fold

SRBC, sheep red blood cells, TCF, tissue culture fluid.

Immunization of these deficient animals failed to evoke antibody responses against the particulate antigen (SRBC). Reconstitution of these animals with the control diet did not appreciably improve the splenic and thymic architecture, although the blood white blood cell populations returned to normal. To improve the fatal immuno-deficient condition of vitamin-deficient animals, a bio-immuno-modulator (BIM) obtained from 18 h bone marrow culture was injected into immuno-deficient animals. A marked improvement in immune response occurred. The BIM did not enhance the immune response in control, healthy animals. The BIM is possibly different from lymphocyte-secreted factors and thymus-secreted factors that augmented HIR and CMIR respectively. BIM appeared to improve both CMIR and HIR.

Effect of clenbuterol on the growth and carcass composition of hypophysectomized rats in the presence or absence of growth hormone. By S. JAMES and HAIDEE D. BARKER* *Coopers Animal Health Ltd, Berkhamsted, Herts HP4 2QE*

Rats treated with β-agonists such as clenbuterol have been shown to gain significantly more weight than untreated controls due to an increase in lean body mass accompanied by reduced body fat. This effect and its applications has recently been reviewed by Stock & Rothwell (1986). The gross effects are reminiscent of some of the actions of growth hormone (GH) and it is possible that the hormone is involved in the repartitioning effect of the β-agonists. Hypophysectomized animals produce no GH and so provide a suitable model for preliminary experimentation.

Hypophysectomized male Wistar rats (Charles River), weighing between 120 and 130 g were stabilized on a ground diet (Expanded Rat Breeder diet; SDS Ltd, Witham, Essex) and were randomly allocated into six groups of seven animals per group. Treatments were 0, 1 and 10 mg clenbuterol (as the free base)/kg diet, with or without 0·1 mg pituitary bovine GH/rat, administered daily subcutaneously in 0·2 ml isotonic 0·1 M-phosphate-buffered saline (PBS). Individual body-weights and group food intakes were measured daily during the 17-d treatment period. The animals were then killed and the following weights recorded: dead body, eviscerated carcass, gastrocnemius muscles, epididymal fat pads.

The GH treatment produced a significant increase in growth rate of 2·5 g/d (0 clenbuterol, PBS-only controls, 0·15 g/d) as well as a small (6·5%), non-significant decrease in carcass:viscera ratio. This was accompanied by increased food intake, but group average clenbuterol dosage remained similar at 0·138–0·140 and 1·39–1·41 mg/kg per d (1 and 10 mg clenbuterol/kg diet respectively). Clenbuterol at 1 mg/kg diet increased growth rate by 0·3 g/d and a further 0·5 g/d in PBS-only and GH treated animals respectively. At 10 mg clenbuterol these increases were 0·65 and 0·98 g/d. All increases were significant (Student's t test) at least at $P<0·05$.

Paired gastrocnemius muscles or epididymal fat pad weights were added and expressed as a percentage of total dead weight. Changes in these values reflect changes in carcass composition (S. James and H. D. Barker, unpublished results). The GH treatment raised muscle weight by 8·6% (from 1·16% of dead weight) and this order of improvement was retained (and significant at $P<0·05$) in the 1 and 10 mg clenbuterol/kg diet treatments which themselves increased this muscle mass by 12·9 and 17·2% respectively. Significant reductions (26·8 and 42·5% from 0·635% of dead weight) in fat pad sizes, resulted from 1 and 10 mg clenbuterol/kg diet with administration PBS only. Only marginal and non-significant further reductions were produced by the GH treatment.

Stock, M. J. & Rothwell, N. J. (1986). In *Control and Manipulation of Animal Growth*, pp. 249–259 [P J. Buttery and D. B. Lindsay, editors]. London: Butterworths.

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Starvation enhances the secretory responses of stripped rat colon and ileum to acetyl choline in vitro. By R. J. LEVIN, A. YOUNG, SARAH SHINN and SARAH HILL, *Department of Physiology, Sheffield University, Sheffield S10 2TN*

Starvation for 72 h has been shown to hypersensitize the secretory mechanisms of rat jejuna to secretory stimulants both in vitro and in vivo (Levin & Young, 1985, 1986), but little is known concerning its effects on the colon and ileum.

Electrogenic secretion was measured across isolated sheets of proximal colon and terminal ileum stripped of their external muscle layers. Basal short circuit currents (Isc) and the maximal changes in Isc (ΔIsc = peak Isc − basal Isc) induced by serosally applied acetyl choline (1 mM, colon; 0·1 mM, ileum) were measured using standard techniques (Baldwin & Levin, 1985). Starvation for 72 h significantly reduced the basal Isc in the colon (−36%, $P<0.01$, n 15) but elevated that of the ileum (+55%, $P<0.01$, n 19) on a fixed area basis compared with fed controls. Despite decreases in the dry weights of the tissue (−25% colon; −21% ileum), cholinergic stimulation had a greater effect in the fasted tissues compared with those of fed controls (ΔIsc colon +116%, $P<0.001$; ΔIsc ileum +161%, $P<0.001$).

Amiloride (an inhibitor of electrogenic sodium ion transfer) was added to the mucosal buffer (1 mM) bathing the colonic sheets. The reduction in the basal Isc due to the decrease in electrogenic absorption of Na^+ was significantly less ($P<0.01$) in fasted colon (−2·3 $\mu A/cm^2$, SE 0·7, n 15) than the fed controls (−6·2 $\mu A/cm^2$, SE 1·2, n 10). The colon from fasted animals thus has less electrogenic Na^+ absorptive capacity than that from fed animals.

The increased basal secretion and augmented secretory response of the fasted ileum coupled with the decreased absorptive capacity but augmented secretory response of the fasted proximal colon may offer a possible explanation for the terminal diarrhoea observed in human famine victims (Helweg-Larssen *et al.* 1952).

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Baldwin, D. & Levin, R. J. (1985). *IRCS Medical Science* **13**, 269–270.
Helweg-Larssen, P., Haffmeyer, H., Kieler, S., Thaysen, E. H., Thaysen, J. H., Thygrsten, P. & Wolff, M. H. (1952). *Acta Medica Scandinavica* **274**, Suppl. 1–460.
Levin, R. J. & Young, A. (1985). *Journal of Physiology* **365**, 109P.
Levin, R. J. & Young, A. (1986). *Journal of Physiology* **378**, 23P.

Absorption of oral bovine lactoferrin in the neonatal and adult pig. By H. HAGEMEISTER, M. SCHMITZ, INA SCHOPPE and C. A. BARTH, *Institut für Physiologie und Biochemie der Ernährung der Bundesanstalt für Milchforschung, D-2300 Kiel 1, West Germany*

A low degradation and absorption of the iron-binding milk protein lactoferrin (LF) has been associated with its proposed nutritional role of enhancing absorption of its ligand and supporting host resistance of the newborn (Hambraeus *et al.* 1984). We have investigated how much LF resists absorption in the small intestine. Our previous experiments in rats (100 g body-weight) had shown an apparent digestibility of 90%.

In order to quantify the disappearance of the dietary protein, the homoarginine technique was used (Hagemeister & Erbersdobler, 1985). For this purpose, six adult minipigs (age 1–3 years) were kept on a semi-synthetic maintenance diet. They were given a test meal (% energy: carbohydrate 55, fat 30, casein 15) containing bovine LF (purity > 98%; 10 g/kg test meal) at 12·4 g dry weight/kg body-weight$^{0.75}$. Five sucking piglets (age 3 weeks) consumed 75 mg LF in 50 ml fresh sow's milk. In both cases LF had been labelled by transforming chemically 90% of its lysine to homoarginine (HA). The concentration of HA and of a non-absorbable marker (chromium (III) oxide) were determined 3 or 6 h postprandially in the ileal contents. The relation of HA to marker was used to calculate the absorption of LF.

			Molar ratio (HA:Cr_2O_3) × 10^3			Precaecal absorption $\frac{A-B}{A} \times 100$	
				Ileal contents (B)			
Animals	(*n*)	Period postprandially (h)	Diet (A)	Mean	SEM	Mean	SEM
Piglets	5	3	51·8	5·5	1·2	89·4*	2·3
Adult pigs	6	6	51·8	1·5	0·3	97·0	0·7

*$P<0.05$ (Student's *t* test).

Absorption was more than 97% in adult animals and comparable to findings with other highly digestible dietary proteins. In sucking piglets it was significantly less (89%).

It will be of interest to study the same indices in freshly newborn piglets (Britton & Koldovsky, 1986). The data, so far, however, are compatible with the above mentioned nutritional role of LF during early postnatal life.

Britton, J. R. & Koldovsky, O. (1986). *Pediatric Research* **20**, 406A.
Hagemeister, H. & Erbersdobler, H. (1985). *Proceedings of the Nutrition Society* **44**, 133A.
Hambraeus, L., Fransson, G.-B. & Lönnerdal, B. (1984). *Lancet* ii, 167–168.

Biotin supply by large bowel bacteria: evidence from intracaecal avidin.

By C. A. BARTH[1], JOHANNE SCHOLTISSEK[1], M. FRIGG[2] and H. HAGEMEISTER[1], [1]*Institut für Physiologie und Biochemie der Ernährung der Bundesanstalt für Milchforschung, D-2300 Kiel 1, West Germany* and [2]*Abt. Vitamin- und Ernährungsforschung, Hoffmann-La Roche, Basle, Switzerland*

In earlier experiments the intracaecal infusion of biotin was accompanied by considerable absorption of the vitamin (Barth *et al.* 1986). Now we report on whether biotin synthesized by the large-bowel microflora constitutes a physiologically significant source of this vitamin for the host.

For this purpose five adult minipigs (age 2–3 years) each with a permanent caecal T-cannula were fed on a semi-synthetic maintenance diet containing 60 g cellulose/kg. The diet provided 157 nmol biotin/d causing biotin deficiency as evidenced by low plasma biotin levels (Table). Following an 8 week adaptation period there were nine sequential 1 week infusion periods (see Table) with or without oral lactulose (26 g/d) or nebacetin (2·94 g/d). Avidin infusion during weeks 2, 5 and 8 amounted to 18 mg/d. On the last day of each period plasma was obtained by venipuncture and urine was collected for 24 h on ice during the last 2 d. Biotin analyses were performed according to Frigg & Brubacher (1976).

Week	Oral supplement	Intracaecal infusion	Plasma biotin (nmol/l) Mean	Plasma biotin (nmol/l) SE	Urinary biotin excretion (nmol/d) Mean	Urinary biotin excretion (nmol/d) SE
1	—	Solvent	1·70	0·10	$93·5^{b}$	17·6
2	—	Avidin	1·90	0·23	$85·3^{a}$	11·3
3	—	Solvent	1·84	0·36	$97·2^{b}$	9·1
4	Lactulose	Solvent	1·96	0·43	$106·4^{b}$	12·8
5	Lactulose	Avidin	1·78	0·24	$93·2^{a}$	3·9
6	Lactulose	Solvent	2·00	0·19	$102·4^{b}$	9·0
7	Nebacetin	Solvent	1·99	0·20	$125·3^{b}$	13·9
8	Nebacetin	Avidin	1·64	0·15	$77·9^{a}$	18·8
9	Nebacetin	Solvent	1·82	0·27	$125·5^{b}$	9·0

[a,b]Means within columns with different superscript letters were significantly different (Student's *t* test): $P<0·05$.

No significant change of plasma biotin was caused by lactulose or avidin (Table). There was a significant average 84% rise in faecal biotin excretion during all avidin periods. Moreover, there was a significant average 23% decline in urinary biotin output following avidin. It is concluded that some biotin synthesized by colonic bacteria is available for absorption and hence for host metabolism.

Barth, C. A., Frigg, M. & Hagemeister, H. (1986). *Journal of Animal Physiology and Animal Nutrition* **55**, 128–134.

Frigg, M. & Brubacher, G. (1976). *International Journal of Vitamin Nutrition Research* **46**, 314–321.

Effects of rumen butyrate infusion on blood ethylmalonic acid concentration in sheep. By M. T. CARSON[1], E. F. UNSWORTH[1,2] and J. PEARCE[1,2], [1]*Agricultural and Food Chemistry Department, The Queen's University of Belfast, Belfast BT9 5PX* and [2]*Department of Agriculture for Northern Ireland*

The presence of ethylmalonic acid in the urine of sheep was demonstrated by Lough & Calder (1976). Their observations recorded that the level of this metabolite in conventionally fed sheep was <20 mg/l urine but in sheep receiving a diet containing rolled barley (900 g/kg) the level increased to 150–550 mg/l urine. They concluded that the ethylmalonic acid did not apparently arise from butyrate since they assumed that, in barley-fed sheep, the proportion of butyrate in rumen contents was somewhat lower than that in sheep given conventional diets. However, the enzymic carboxylation of butyryl CoA to ethylmalonyl CoA by bovine liver mitochondrial extracts has been demonstrated in vitro (Hegre *et al.* 1959) and in addition the elevation of rumen butyrate content has been observed on silage–concentrate diets (A. R. G. Wylie and E. F. Unsworth, unpublished observations). It therefore seems possible that increased butyrate supply to the tissues leads to increased ethylmalonic acid formation. To test the hypothesis that butyrate is a precursor of blood ethylmalonic acid, six 4-year-old cross-bred wethers (mean body-weight 80 kg) received equal amounts of butyric acid solution or physiological saline (9 g sodium chloride/l) by ruminal infusion in a 3×2 complete cross-over design experiment. Animals were given isoenergetically a basal pelleted, ground, dried-grass diet supplying 12·8 MJ metabolizable energy (ME)/d. Where butyrate was infused it supplied 3·2 MJ ME/d and the amount of the dried-grass given was correspondingly reduced. After 10 d of butyrate or saline infusion, blood samples were obtained from the jugular vein. Six samples were obtained over a 7 h period, the first sample being taken immediately before feeding. The experimental treatments were then changed and the infusions carried out for a further 10 d, blood samples being obtained as before. Plasma samples were analysed for ethylmalonic acid essentially by the method of McMurray *et al.* (1986).

One animal was removed from the experiment because it went off its feed and the results for the remaining five animals showed that the plasma ethylmalonic acid concentration was significantly increased ($P<0·05$) when butyric acid was infused. The mean (SE) plasma levels found for animals on butyric acid infusion were 2·47 (0·310) μmol/l (range 1·04–6·92 μmol/l) compared with 1·07 (0·310) μmol/l (range 0·39–2·79 μmol/l) when saline was infused.

These results imply a relation between rumen butyrate and blood ethylmalonic acid and the role of butyrate as a metabolic precursor of ethylmalonic acid merits further direct investigation.

Hegre, C. S., Halenz, D. R. & Lane, M. D. (1959). *Journal of the American Chemical Society* **81**, 6526–6527.
Lough, A. K. & Calder, A. G. (1976). *Proceedings of the Nutrition Society* **35**, 90A.
McMurray, C. H., Blanchflower, W. J., Rice, D. A. & McLoughlin, M. (1986). *Journal of Chromatography* **378**, 201–207.

Tissue incorporation and excretion of ^{14}C in pigs after injection of [1-^{14}C]- or [2-^{14}C]propionic acid into the caecum. By EVA A. LATYMER and A. G. LOW, *AFRC Institute of Grassland and Animal Production, Shinfield, Reading, Berkshire RG2 9AQ*

We recently showed that [U-^{14}C]sodium acetate was absorbed from the caecum of two 70–78 kg pigs and 26–27% of the ^{14}C was retained in body tissues 96 h later (Latymer & Low, 1984). We have now investigated in two pigs the metabolism of the second most abundantly produced volatile fatty acid in the pig large intestine, propionic acid. Because [U-^{14}C]sodium propionate was not available, one pig received [1-^{14}C]sodium propionate (pig 1) and the other [2-^{14}C]sodium propionate (pig 2) in order to assess the metabolic fate of propionic acid and any differences in metabolism of the carbon in its carboxyl and first methyl groups.

The balance study was carried out in pigs of 73 and 82 kg, fed on a barley–soya-bean meal diet. The sodium propionate was injected in a single dose through a simple cannula placed in the caecum. The pigs were kept in metabolism cages for 4 d, during which they ate normally and all urine and faeces were collected. After 4 d the pigs were killed, the blood was collected and the rest of the body was retained for ^{14}C analysis. Representative samples of each tissue or organ were minced, mixed and homogenized as appropriate and then solubilized.

In both pigs the greatest loss of ^{14}C in urine occurred on day 1, faecal losses were highest on day 2, and on day 3 (pig 1 only). Combined urinary and faecal losses of ^{14}C were 5·9% of the dose for both pigs. The distribution of ^{14}C in the body after 4 d is shown in the Table (expressed as % of the dose) (intestine values include contents).

Pig no.	Bile and liver	Kidney	Small intestine	Large intestine	Blood	Carcass	Recovery
1	0·43	0·03	0·00	0·08	0·07	1·60	2·21
2	1·78	0·31	1·24	0·56	0·19	13·00	17·08

Although the highest concentration of radioactivity was found in the liver, the highest total radioactivity was found in the carcass lean and fat.

The results indicate that exogenous propionic acid served as a source of energy for the pigs, but the metabolic fate of carbon atoms 1 and 2 was different.

Latymer, E. A. & Low, A. G. (1984). *Proceedings of the Nutrition Society* **43**, 12A.

Reduction in the saturated fatty acid content of cow's milk fat through diet formulation. By P. A. MARTIN and P. C. THOMAS, *Hannah Research Institute, Ayr KA6 5HL*

An experiment was undertaken to investigate the possibility of reducing the saturated fatty acid content of cow's milk fat by using oats rather than barley as the dietary cereal source. Eight Friesian cows were used in a duplicated 4×4 Latin square experiment. The cows were given four isoenergetic diets consisting of hay, soya-bean meal and barley or oats (approximately 34:12:54 on a dry matter basis), with the cereals given either rolled or rolled and treated with an acidified-formalin reagent. This reagent cross-links the cereal starch and protein, reducing their rates of fermentation in the rumen (Kassem *et al.* 1987). Due to the higher lipid content of the oats the oats diets provided 537 g fatty acids/d, whilst the corresponding value for the barley diets was 211 g/d. The fatty acid composition (g/kg) of oats diets was: 16:0, 180; 18:0, 20; 18:1, 390; 18:2, 380; 18:3, 30. The corresponding values for the barley diets were 300, 20, 150, 470 and 60.

	Barley	Treated barley	Oats	Treated oats	SED
Milk yield (kg/d)	15·9	16·9	17·1	18·2	0·5*
Fat yield (g/d)	660	627	649	662	29
Fatty acids (g/kg)					
Saturated (6:0–18:0)	753	750	619	614	13†
Mono-unsaturated (16:1–18:1)	211(10)‡	212(9)	344(25)	350(22)	11†(3†)
Polyunsaturated (18:2–18:3)	35	38	37	38	2

*$P<0.01$, †$P<0.001$.
‡Values in parentheses are *trans* 18:1.

Milk yields were slightly greater with the oats diets than with the corresponding barley diets (see Table) and were higher with the formalin-treated grains. There were no significant differences in fat yield between treatments but oats diets led to a significant reduction in the saturated fatty acid content of milk fat and to an associated increase in the mono-unsaturated acids. Formalin treatment of the cereals was without effect on milk fatty acid composition.

The milk fatty acid composition with the oats diets is consistent with hydrogenation of dietary unsaturated C18 acids in the rumen, subsequent desaturation of 18:0 in the mammary gland and concurrent suppression of intramammary synthesis of 6:0 to 16:0 acids.

The results indicate that oats have a useful role in the formulation of diets designed to reduce the saturated fatty acid content of cow's milk fat.

Kassem, M. E., Thomas, P. C., Chamberlain, D. G. & Robertson, S. (1987). *Grass and Forage Science* (In the Press).

The effect of lead and phospholipids on serum cholesterol. By J. QUARTERMAN and KATY TAM, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

In lead-intoxicated rats, changes in serum lipids occur which depend on the type of dietary lipid (Szymczak *et al.* 1983), and among the sources of lipid, dietary lecithin has, in proportion to its concentration, strong effects on Pb absorption (Quarterman *et al.* 1977). We have therefore examined the effects of dietary supplements of Pb and lecithin alone and together on serum total lipids and cholesterol in rats.

Rats weighing initially 115 (SE 5) g in groups of six were given a semi-purified diet containing 50 g arachis oil/kg and supplemented, as indicated in Table 1, for 20 d and then killed under anaesthesia. Pb was given as Pb acetate and lecithin was obtained from egg yolk and contained about 600 g phosphatidyl choline/kg (Sigma Chemical Co. Ltd, Poole, Dorset). There was a mild degree of Pb toxicity indicated by decreased body-weight gain, haemoglobin concentration and increased tissue Pb content, but Pb alone had no effect on serum lipids or cholesterol content. Lecithin given with the Pb supplement did not change serum lipid content but increased serum total cholesterol by about 60%.

A second experiment conducted under similar conditions showed that a lecithin supplement without Pb produced no significant change in serum lipids (4500 (SE 270) *v.* 5470 (SE 450) mg/l) or cholesterol (1030 (SE 50) *v.* 1180 (SE 50) mg/l).

Table 1. *Effect of dietary Pb and lecithin supplements on serum total lipids and cholesterol concentrations*

Dietary supplement (g/kg control diet)	Final body-wt (g)		Blood haemoglobin (g/l)		Kidney Pb (μg/g wet wt)		Serum total lipids (mg/l)		Serum total cholesterol (mg/l)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Nil (control)	173 ••	3	152•	8	49•••	4	5430	660	890	50
Pb (0·4)	158	2	134	1	257	29	5510	560	940	30
Pb (1·0)	142•••	2	122•	5	286	16	4470	470	800	80
Pb (0·4) + lecithin (5·0)	160	4	128	3	185	20	5490	470	1440•••	80
Pb (0·4) + lecithin (5·0) + arachis oil (150)	153	4	137	5	213	14	5470	690	1410•••	60

Significantly different from the group supplemented with 0·4 g Pb/kg only: •$P<0.05$, ••$P<0.01$, •••$P<0.001$.

The Pb exposure of these rats was much greater than is usually encountered by human subjects but the effect on cholesterol levels was large and lower levels of Pb exposure may present a hazard in this way as in many others.

Quarterman, J., Morrison, J. N. & Humpries, W. R. (1977). *Proceedings of the Nutrition Society* **36**, 103A.

Szymczak, J., Zechalko, A. & Breinat, J. (1983). *Bromatologia i Chemia Toksykiologiczra* **1612**, 89–93.

Adaptation of a rapid, single test-tube, fluorometric method for the estimation of selenium in foods and food products. By K. JORDAN, H. S. SHORTT and P. M. MATHIAS, *Dublin Institute of Technology, Kevin Street, Dublin* 8, *Irish Republic*

Selenium is recognized as an essential trace element for man (Golden, 1982). This has led to an increased interest in the Se intake and Se status of human populations. One of the most sensitive methods for the estimation of Se in a variety of materials is the fluorometric method (Michie *et al.* 1978). It involves a long (24–48 h) Kjeldahl digestion of material, a pH adjustment to convert Se to its tetravalent form which is then incubated with 2,3-diaminonaphthalene to form a piazoselenol complex. This is extracted into cyclohexane and the fluorescence of the complex measured. The method was modified by Spallholz *et al.* (1979) who used a single test-tube for the whole procedure thereby improving precision and reducing the total time for the analysis of Se in blood to under 4 h. The purpose of the present study was to adapt the single test-tube method for the rapid estimation of Se in foods and food products. After a few minor changes, the assay was standardized with respect to pH, incubation time and temperature, and extraction time. Precision, using serum, bread and milk samples, and accuracy, using certified materials of grass and wholemeal flour (supplied by The Agricultural Institute, Johnstown Castle, Wexford), compared favourably with the official fluorometric method (Michie *et al.* 1978). With this modified assay, normal plasma Se values ranged between 50 and 90 ng/ml. Preliminary results on some foods showed a mean value for Se in pasteurized cow's milk of 53 ng/ml, whilst in modified infant formulae (normal bottle dilution), including those used by patients with phenylketonuria or maple syrup urine disease, values (ng/ml) were lower: Lofenelac, 17; MSUD Aid, 5; Minafen, 21; SMA Gold Cap, 14. An average value for white bread was 123 ng/g.

These results compare well with those found by other workers (McKenzie *et al.* 1978). The rapid single test-tube procedure appears, therefore, to be highly promising for further investigative work into the Se content of Irish foods and the Irish diet.

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Plasma phospholipid fatty acid composition in men of Asian and European descent. By SHEELA REDDY and T. A. B. SANDERS, *Department of Food and Nutritional Sciences, King's College, Campden Hill Road, London W8 7AH*

The incidence of coronary heart disease in males of Indian descent is greater than that in other ethnic groups (Tunstall Pedoe *et al.* 1975; Miller *et al.* 1982). As part of a community study into diet and its relation to plasma lipoproteins in Gujarati men and men of European descent, we have determined the fatty acids composition of plasma phospholipids as an indicator of polyunsaturated fatty acid intake.

Fasting blood samples were obtained from men aged 45–54 years of both groups living in the Wembley area and the fatty acid composition of the plasma phospholipids was determined by capillary gas–liquid chromatography. The results (weight % of total fatty acids) are shown in the Table.

	Europeans (*n* 19)		Asians (*n* 18)	
Fatty acid	Mean	SE	Mean	SE
14:0	0·28	0·034	0·27	0·028
14:1	0·1	0·011	0·14	0·017
16:0	23·12	0·899	22·97	0·873
16:1	1·54	0·154	0·99**	0·125
18:0	13·97	0·358	15·04*	0·314
18:1	16·1	0·452	13·13**	0·691
18:2*n*-6	20·51	0·628	24·41**	0·493
18:3*n*-3	0·61	0·029	0·64	0·075
20:0	0·12	0·025	0·15	0·042
20:1	0·44	0·053	0·2**	0·04
20:2*n*-6	0·33	0·021	0·33	0·042
20:3*n*-9	0·16	0·014	0·17	0·033
20:3*n*-6	2·61	0·117	3·03	0·209
20:4*n*-6	8·82	0·323	10·51*	0·387
24:1 + 22:2	1·14	0·142	0·98	0·108
20:5*n*-3	1·85	0·177	1·34**	0·113
26:1	0·41	0·032	0·45	0·04
22:4*n*-6	1·55	0·521	1·4	0·481
22:5*n*-3	1·36	0·053	1·33	0·066
22:6*n*-3	4·82	0·282	2·63**	0·238

Significantly different from values for Europeans: *$P<0.05$, **$P<0.01$.

The proportions of linoleic acid (18:2*n*-6), which is derived from vegetable oils, were greater in the Asians as were the proportions of arachidonic (20:4*n*-6) and stearic (18:0) acids. The proportions of eicosapentaenoic acid (20:5*n*-3) and docosahexaenoic acid (22:6*n*-3), which are found in marine oils and organ meats, were significantly lower in the Asians. The higher proportion of 16:1 and 20:1 fatty acids in Europeans could be indicative of the consumption of hardened marine oils as in margarine.

Miller, G. J., Beckles, G. L. A., Alexis, S. D., Byam, N. T. A. & Price, S. D. L. (1982). *Lancet* **ii**, 200–203.

Tunstall Pedoe, H., Clayton, D., Morris, J. N., Brigden, W. & McDonald, L. (1975). *Lancet* **ii**, 833–838.

Overnight metabolic rate in men and women. By G. R. GOLDBERG, H. L. DAVIES, P. R. MURGATROYD and A. M. PRENTICE, *MRC Dunn Nutrition Unit, Cambridge CB4 1XJ*

The Food and Agriculture Organization/World Health Organization/United Nations University (1985) recommendations for energy requirements are based on factorial calculations in which the energy cost of sleep is assumed to be equal to the basal metabolic rate (BMR). However, the validity of this assumption has been questioned since metabolic rate (MR) during deep sleep is considerably lower than BMR. We have analysed data from a number of studies using 11-m^3, indirect, whole-body calorimeters (Table). BMR was measured over 1 h immediately after waking when subjects were post-absorptive (13 h), at complete rest and at thermoneutrality. Overnight MR was measured from 23.30 to 08.00 hours (commencing 30 min after lights out). Lowest sleeping MR (SMR) was the lowest continuous hour recorded.

		Overnight MR:BMR		Lowest SMR:BMR	
Subjects	*n*	Mean	SD	Mean	SD
Lean ♀	34	0·95	0·04	0·88	0·04
Lean ♂	6	0·96	0·03	0·88	0·04
Obese ♀	12	0·99[a]	0·04	0·89	0·04
Pregnant:					
non-pregnant	13	0·94	0·03	0·88	0·04
36 weeks	13	1·00[b]	0·04	0·92[c]	0·03
Monitored ♀ and ♂ •	23	1·00[d]	0·06	0·90	0·04
Exercising ♀ and ♂, grade †:					
1	8	0·97	0·03	0·90	0·03
2	7	0·96	0·04	0·89	0·04
3	8	0·98	0·04	0·90	0·04
4	9	0·96	0·03	0·89	0·03
5	5	0·96	0·03	0·88	0·03

•Electrocardiogram electrodes attached.

†Exercise grades; 1, 0 kpm for ♂ + ♀; 2, 18 000 kpm for ♂ + ♀; 3, 36 000 kpm for ♂ and 27 000 kpm for ♀; 4, 54 000 kpm for ♂ and 36 000 kpm for ♀; 5, 72 000 kpm for ♂ and 45 000 kpm for ♀.

Within a column, values were significantly different: [a]$P<0.01$ *v.* lean ♀. [b]$P<0.001$, [c]$P<0.05$ *v.* non-pregnant (paired *t* test). [d]$P<0.001$ *v.* lean ♀ and ♂.

The results confirm that MR during deep sleep is approximately 10% lower than BMR. The average ratio of lowest SMR:BMR for all subjects was 0·90 (range 0·81–0·97). Average overnight MR:BMR in normal, lean subjects was 0·95 (range 0·85–1·02). This ratio was not significantly affected by different levels of exercise on the preceeding day. The ratio was significantly higher in subjects who were obese, in late pregnancy or attached to electrocardiogram electrodes. Except in the late-pregnant subjects these groups had normal ratios for lowest SMR:BMR, indicating that the higher overnight MR was caused by disturbed sleep as opposed to any inherent differences in overnight energy metabolism.

The data indicate that for most well-nourished, healthy subjects the use of BMR to estimate overnight energy expenditure would introduce an average overestimate of approximately 5%. However, since this is only applied to an 8 h period the error over 24 h is negligible.

Food and Agriculture Organization/World Health Organization/United Nations University (1985). *Energy and Protein Requirements. Technical Report Series* no. 724. Geneva: WHO.

Influence of carbohydrate supplementation on running performance. By C. WILLIAMS, M. G NUTE and M. P. WALKER, *Department of Physical Education and Sports Science, University of Technology, Loughborough, Leics LE11 3TU*

A high-carbohydrate diet has been shown to have a beneficial influence on running performance (Karlsson & Saltin, 1971). However little attention has been paid to the influence of carbohydrate solutions, ingested during a race, on running performance. The purpose of the present study was to examine the influence of drinking a carbohydrate solution on running speed and distance covered during a simulated race lasting 2 h.

Fifteen runners (ten male and five female) took part in this study which employed a cross-over design and involved two treadmill races lasting 2 h, separated by 7 d. Immediately before the first run each subject drank 250 ml of either the carbohydrate solution (a maltodextrin and sucrose mixture; CHO trial) or a placebo (placebo trial) whereas during the run the subjects were encouraged to ingest a further 750 ml in 125 ml portions. After the first 15 min, which was run at a speed equivalent to 70% maximum oxygen consumption ($\dot{V}_{O_2max}$), the subjects were free to choose their own running speeds, using a computer-linked, hand-held micro-switch, in order to complete the greatest distance in the remaining time. Venous blood samples were obtained before and immediately after the run while during the run capillary blood samples were obtained every 15 min along with samples of expired air. A summary of results is shown in the Table.

	CHO trial		Placebo trial		
	Mean	SD	Mean	SD	$P<$
Distance (km)	28·6	3·62	28·3	3·4	NS
Speed (km/h)	14·3	0·2	14·1	0·3	NS
Blood glucose (mmol/l)	5·37	0·35	5·12	0·41	NS
Blood lactate (mmol/l)	2·29	0·41	2·37	0·30	NS
Plasma free fatty acids (mmol/l)	0·71	0·38	0·99	0·33	0·05
Plasma glycerol (mmol/l)	0·58	0·15	0·69	0·16	0·01

NS, not significant.

There was no significant difference between the distances covered during the CHO and placebo trials, nor were there differences in the mean overall running speeds chosen during these two trials. However the running speeds towards the end of the 2 h were faster for the CHO trial than for the placebo trial such that after 90 min of running the differences reached statistical significance ($P<0·05$). As expected, the increases in plasma free fatty acids and glycerol were significantly less during the CHO trial than during the placebo trial, reflecting a decrease in fat metabolism. Thus the results of this study suggest that ingestion of a carbohydrate solution during a 2 h race for distance has no significant influence on performance, however some benefit might be gained during races of longer duration.

This study was supported by Collett-Marwell Hauge a/s, Norway.

Karlsson, J. & Saltin, B. (1971). *Journal of Applied Physiology* 31, 203–206.

Fibre, dietitians and irritable bowel syndrome. By JANET P. LOWELL, *School of Nutritional Science, Robert Gordon's Institute of Technology, Aberdeen,* and VALERIE MORRISON, C. K. W. LAI, C. C. KHIN, N. A. G. MOWAT and P. W. BRUNT, *Gastrointestinal Unit, Woodend Hospital, Aberdeen AB9 2YS*

The efficacy of dietary fibre and professional dietetic advice in irritable bowel syndrome (IBS) in its various forms is not proven. A single-blind controlled study was conducted in seventy-three patients with established IBS, recruited over a period of 18 months and managed on a high-fibre diet. Patients were randomly divided into three groups in batches of nine at a time. Group A had regular dietetic advice, group B a single interview with the dietitian, group C a diet sheet alone without explanation. Fibre intake was assessed by questionnaire at the beginning and end of the 6 month study. Four other patients failed to complete the study (one left the area, two failed to attend follow-up visits despite frequent reminders and one was subsequently diagnosed as having ulcerative colitis). Symptomatic response was assessed blindly by a clinician using a standardized scoring system (Thompson & Heaton, 1980).

The daily fibre intake in all groups at the start of the study was similar (mean 17·7 g). In all the dietary fibre intake had significantly increased by the end of the study (mean 25·3 g/d, $P<0.001$). Surprisingly, there was no significant difference in fibre intake of the three groups at the end of the study (Table). Furthermore, none of the groups achieved the nationally recommended intake of 30 g fibre/d (National Advisory Committee on Nutrition Education, 1983). The following symptoms did, however, improve significantly: constipation ($P<0.02$), mucus in the stool ($P<0.025$), abdominal pain ($P<0.005$). Simple diet sheets have limitations, but currently employed dietetic methods do not appear to confer any advantage and should be revised.

Dietary fibre intake of patients (g/d)

Group . . .	A (*n* 22)		B (*n* 26)		C (*n* 25)	
	Mean	SD	Mean	SD	Mean	SD
Beginning of study	19·5	13·8	16·8	7·7	17·0	7·6
End of study	27·1	12·0	25·2	8·0	23·7	6·3
Increase	7·7	8·0	8·4	7·9	6·7	8·8

National Advisory Committee on Nutrition Education (1983). *Proposals for Nutritional Guidelines for Health Education in Britain.* London: Health Education Council.
Thompson, W. G. & Heaton, K. W. (1980). *Gastroenterology* **79**, 283–288.

Evaluation of gastroplasty in the management of severe obesity. By R. A. HARRISON, S. POPE and C. G. CLARK, *Clinician Nutrition Unit, Department of Surgery, University College School of Medicine, University Street, London WC1E 6JJ* and A. M. TOMKINS, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 6HT*

Twenty-two adults (eighteen women and four men) with severe obesity (body mass index more than 39) were monitored for 12 months after vertical banded gastroplasty had been performed (Mason, 1982). Each had found that pre-operative dietary approaches had been unsuccessful in controlling body-weight and they had been referred to a special clinic for assessment of suitability for the operation.

	Initial body size		Wt loss (kg) during each 3 month period				
	Wt (kg)	Body mass index (wt/height^2)	First	Second	Third	Fourth	Total
Mean	129·3	47·1	20·2	9·2	4·7	4·6	40·8
SD	20·7	5·8	7·2	4·8	3·0	3·6	17·4
Range	100–179	39–59	9–26	0–19	1–11	4–15	20–86

The weight loss was greatest during the first 3 months following surgery (Table). Discomfort and a degree of psychological stress were experienced during this time. This was minimized by the use of liquidized food during the early post-operative period. Mild intolerance to certain dietary items was experienced following re-introduction of food. Weight loss during the second 6 months was maintained but four patients experienced a temporary period of weight gain during the last quarter of the study. This responded to renewed dietary efforts. Post-operative wound dehiscence, venous thrombosis or major infection were not experienced. However, two patients required revisional surgery because of stenosis of the stoma. We suggest that this procedure is a suitable form of management for selected patients with severe obesity.

Mason, E. E. (1982). *Archives of Surgery* **117**, 701–716.

The effects of pre-operative nutritional status on post-operative complications in head and neck surgery. By A. M. WEIR[1], R. RICHARDSON[2], O. J. GARDEN[3] and A. SHENKIN[4], *Departments of [1]Otolaryngology, [2]Dietetics, [3]Surgery* and *[4]Biochemistry, Glasgow Royal Infirmary, Glasgow G31 2ER*

Pre-operative nutritional status has been put forward as an important factor in the development of post-operative complications in major abdominal surgery (Mullen *et al.* 1979). However, doubt has been cast on its importance as regards the outcome of head and neck surgery.

Twenty-four patients undergoing major head and neck surgery had an assessment of pre-operative nutritional status. Of these, nine went on to develop major post-operative complications (five pharyngeal fistula, three flap necrosis, one bony malunion), causing considerable prolongation of their hospital stay. The group without complications (group 1) consisted of twelve men and three women, mean age 61 (SE 10) years, whereas the group with complications (group 2) consisted of seven men and two women, mean age 57 (SE 9) years. Pre-operative anthropometric measurements were made. There was no significant difference between group 1 and group 2 in percentage ideal body-weight (97 (SD 20)% and 94 (SD 24)% respectively), percentage normal triceps skinfold thickness (76 (SD 40)% and 72 (SD 36)% respectively), or percentage normal midarm muscle circumference (89 (SD 8)% and 94 (SD 10)% respectively). These values suggest that pre-operative weight and muscle protein were fairly satisfactory, but that there was some loss of body fat. Serum levels of albumin, transferrin, vitamin C and zinc were measured pre-operatively.

	Group 1		Group 2	
	Mean	SD	Mean	SD
Albumin (g/l)	37·4	4·4	37·9	5·0
Transferrin (g/l)	2·4	0·6	2·7	0·2
Vitamin C (μmol/l)	26·2	21·6	11·7	7·3
Zinc (μmol/l)	13·2	2·4	12·4	2·2

All mean levels were within the laboratory reference ranges, there being no significant difference between the two groups (Mann-Whitney test).

The 'Sheffield' Prognostic Index (Simms *et al.* 1982) was also applied and neither group fell into the high-risk category (⩾50): group 1, 38 (SD 17); group 2, 29 (SD 10).

Neither group of patients was, therefore, particularly malnourished pre-operatively and there was no significant tendency for the group which developed post-operative complications to be more poorly nourished. This would suggest that in these relatively well-nourished patients, minor nutritional variations have no bearing on the development of major post-operative complications.

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Simms, J. M., Smith, J. A. R. & Woods, H. F. (1982). *Clinical Nutrition* **1**, 71–79.

Somatomedin C, insulin, and protein synthesis in patients receiving biosynthetic human growth hormone and undergoing surgery or during total parenteral nutrition. By G. A. PONTING[1], H. A. WARD[1], J. D. TEALE[2], D. HALLIDAY[3] and A. J. W. SIM[1], [1]*Academic Surgical Unit, St Mary's Hospital Medical School, London W2*, [2]*Department of Clinical Biochemistry, St Luke's Hospital, Guildford, Surrey*, [3]*Clinical Research Centre, Northwick Park, Harrow, Middlesex*

Plasma somatomedin C (SM-C) levels fall after musculo-skeletal trauma (Frayn *et al.* 1984). Somatomedins are thought to have anabolic effects on protein synthesis in muscle (Uthne *et al.* 1974). The present study investigated the relation between serum insulin, SM-C, nitrogen balance (NBAL) and whole body protein synthesis (WBPS) in patients receiving either biosynthetic human growth hormone (BSHGH, 0·1 mg/kg per d) or placebo. Patients were studied 5 d after either operation (group 1) or total parenteral nutrition (group 2). Group 1 received 1670 kJ (400 kcal)/24 h as a glucose solution (50 g/l); group 2 received 4180 kJ (1000 kcal)/24 h as carbohydrate, 4180 kJ (1000 kcal)/24 h as fat and 14 g nitrogen/24 h. Insulin and SM-C were measured by radioimmunoassay. WBPS was calculated from N turnover measured with a primed continuous infusion of [^{15}N]glycine and urinary N excretion.

	Group 1				Group 2			
	BSHGH (*n* 6)		Placebo (*n* 7)		BSHGH (*n* 4)		Placebo (*n* 6)	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
WBPS (g/kg lean body mass per d)	5·31*	1·1	2·5	0·3	4·0	0·5	3·7	0·4
Insulin (μmol/ml)	81	21	30	9	210	42	146	26
SM-C (% day 1)	1·06*	0·2	0·34	0·1	1·4*	0·4	0·75	0·1
	201*	14	56·4	8	308*	6	115	13
NEBAL (g/d, average for days 1–7)	−5·2*	0·4	−7·1	0·5	6·0*	0·5	2·7	0·4

*$P<0·05$ (Mann-Whitney U test).

The administration of BSHGH resulted in increased levels of insulin and SM-C, and improved NBAL in both postoperative and parenterally fed patients. When both groups were considered there was a significant correlation between WBPS and SM-C (r 0·61, $P<0·01$), but not between WBPS and plasma insulin ($P>0·05$). These results suggest that SM-C is a mediator in increasing protein synthesis in postoperative patients given BSHGH.

Frayn, K. N., Price, D. A., Maycock, P. F. & Carroll, S. M. (1984). *Clinical Endocrinology* **20**, 179–187.

Uthne, K., Reagan, C. R., Gimpel, L. P. & Kostyo, J. L. (1974). *Journal of Clinical Endocrinology and Metabolism* **39**, 548–554.

Energy requirements of Indian men: overestimation by new international method. By G. MCNEILL, J. P. W. RIVERS and P. R. PAYNE, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*

Recent proposals for the estimation of energy requirements (Food and Agriculture Organization/World Health Organization/United Nations University (FAO/WHO/UNU), 1985) are based on two premises: that basal metabolic rate (BMR) can reliably be predicted from linear regression equations of BMR on weight in different age groups, and that the ratio of the energy cost of any activity:BMR (CA:BMR) is relatively constant in different populations. We have used measurements of energy expenditure of rural Indian men to test the validity of these assumptions in a population living at a low plane of nutrition.

The BMR of fifty-eight men aged 20–49 years was measured in the study village, using modified Oxylog portable oxygen consumption meters (McNeill *et al.* 1987). Mean body-weight was 51·6 (SD 7·2) kg, mean body mass index 19·3 (SD 2·1) kg/m^2 and mean BMR 5·40 (SD 0·69) MJ/d. Observed BMR values were on average 12·1 (SD 8·8)% below those predicted by the FAO/WHO/UNU (1985) equations ($P<0·001$ by paired t test). Similar over-prediction has been found in other studies of the BMR of Indians.

In forty-three of the men the energy cost of a standardized step-test was measured, and the ratio of the energy cost of the step-test:BMR (ST:BMR) calculated for each subject. The ratio showed a significant positive correlation with body-weight:

$$\text{ST:BMR} = 0·055\,(\text{wt}) + 1·182 \qquad r + 0·55;\ P<0·001,$$

illustrating that 50-kg men had ST:BMR ratios on average 17·3% less than 65-kg men.

Similar positive correlations between CA:BMR and weight seem likely in other activities involving considerable body movement. These activities tend to occupy more time in rural populations in less-developed countries than in most Western populations. Use of CA:BMR ratios derived in heavy Western subjects may therefore lead to considerable overestimation of the total energy expended in such activities by lighter, more active subjects.

The fact that the new FAO/WHO/UNU (1985) method can overestimate both the BMR and the energy cost of some activities in small Indian subjects suggests that the method will substantially overestimate the energy required for maintenance of prevailing levels of body-weight and physical activity. Estimation of optimum levels of energy intake requires new information on minimum desirable levels of weight and physical activity in rural populations of less-developed countries.

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McNeill, G., Cox, M. D. & Rivers, J. P. W. (1987). *American Journal of Clinical Nutrition* (In the Press).

The Keneba prenatal supplementation programme: updated analysis after 4 years of intervention. By A. M. PRENTICE, T. J. COLE, F. FOORD, W. H. LAMB and R. G. WHITEHEAD, *Dunn Nutrition Unit, Milton Road, Cambridge CB4 1XJ* and *Keneba, The Gambia*

Prentice *et al.* (1983) reported that a prenatal dietary supplement offered to women in Keneba, The Gambia, resulted in a significant improvement in birth weight. This report extends the analysis to include 104 additional supplemented births.

Pre-intervention birth weights (n 181) averaged 2944 (SE 43) g during the dry season (February–June) when women were in positive energy balance (mean pregnancy weight gain >1200 g/month). Birth weights decreased to 2808 (SE 41) g ($P<0.01$) during the wet season (July–January) when women were in marked negative energy balance caused by food shortages and a heavy agricultural workload (weight gain <500 g/month). A supplement of groundnut biscuits and a vitamin-fortified drink was provided 6 d/week under supervision. Average net intake, allowing for home diet replacement, was 1800 kJ (431 kcal)/d for 24 weeks. The Table summarizes multiple regression and χ^2 analysis.

		Dry season: Supplement effect on birth wt (g)			Wet season: Supplement effect on birth wt (g)		
		Mean	SE	P	Mean	SE	P
All births (n 379):	Unadjusted	+2	62	NS	+225	56	<0·001
Births with known GA	Adjusted*	+13	58	NS	+200	53	<0·001
(n 288):	Unadjusted	+59	69	NS	+254	68	<0·001
	Adjusted†	+11	58	NS	+189	62	<0·005
Within-mother analysis†		−19	61	NS	+231	65	<0·001
		Supplementation: Before	After		Supplementation: Before	After	
%<2501 g birth wt (n 379)		12·5	8·6	NS	23·7	7·5	<0·02
%<Aberdeen 10th percentile (n 288)		25·5	26·6	NS	43·8	26·4	<0·05

GA, gestational age; NS, not significant.
*Adjusted for sex, season and parity.
†Adjusted for sex, season, parity and GA.

Birth weight was significantly improved over the whole year ($P<0.05$, not tabulated), but disaggregation of the data revealed an important threshold effect. All methods of analysis showed that the supplement was ineffective during the dry season but highly effective during the wet season. The predicted impact of the supplement in the wet season would be to reduce neonatal mortality by 37%. These results emphasize the importance of efficient and selective targetting of dietary supplements to truly 'at risk' groups.

Prentice, A. M., Whitehead, R. G., Watkinson, M., Lamb, W. H. & Cole, T. J. (1983). *Lancet* **i**, 489–492.

The three-town diet study: food intake. By JANET CADE, D. J. P. BARKER, BARRIE MARGETTS and JULIE MORRIS, *MRC Environmental Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton SO9 4XY*

The present paper describes the food intake patterns in three English towns, (Ipswich, Stoke and Wakefield) with large differences in disease rates including ischaemic heart disease (IHD) mortality. We have previously described nutrient intakes in the towns (Cade *et al.* 1987).

Diet was assessed using a 24-h dietary record and food frequency questionnaire in a random sample of 35–54-year-olds. The proportion of subjects who recorded various food groups is shown in the Table. These are the main food groups which were statistically significantly different between the towns. In Wakefield, with the highest IHD mortality of the three towns, more people ate wholemeal bread and skimmed milk and fewer ate high fat cheese than in either Stoke or Ipswich. More subjects in Ipswich ate fruit. Foods were also grouped into 'good' and 'bad' food groups using various recent dietary guidelines; the average 'good' food score was highest in Ipswich, the town with the lowest IHD mortality. 'Bad' food scores were similar in the three towns.

Percentage eating each food group

	Men			Women		
	Ipswich	Stoke	Wakefield	Ipswich	Stoke	Wakefield
n ...	369	397	375	406	438	417
Food group						
Wholemeal bread and flour	15	↓13	↑22	27	24	28
White bread and flour	80	↑85	↓76	67	↑76	↓66
Skimmed milk	8	↓ 6	↑11	14	14	15
High-fat cheese	↑46	42	↓28	34	↑35	↓26
Low-fat spread	↑10	↓ 5	7	9	5	9
All margarine	56	↑64	↓54	51	52	51
White fish, fried	8	↓ 6	↑12	↓ 6	7	↑12
Potato (not fried)	↑64	↓44	52	↑59	↓47	49
Potato (fried)	↓21	↑29	24	↓16	↑24	19
All fruit	↑47	↓33	38	↑55	↓43	48
Beer	↓24	↑38	38	↓ 4	↑12	7

↑, Highest value; ↓, lowest value where differences are significant ($P<0.05$) x^2 test (2 df).

Overall the food intake data reflected patterns seen previously for nutrient intake in the three towns. The differences would not contribute substantially to an explanation of the differences in IHD mortality between the three towns.

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The effect of diet on *N*-nitrosoproline excretion in man. By I. R. ROWLAND[1], A. K. MALLETT[1], F. WARD[2], R. WALKER[3] and M. E. COATES[2], [1]*BIBRA, Carshalton, Surrey,* [2]*Robens Institute, University of Surrey, Guildford, Surrey* and [3]*Biochemistry Department, University of Surrey, Guildford, Surrey*

Three healthy males aged 22–26 years consumed their normal free-choice diet or that diet with an additional 18 g high-methoxyl pectin, 30 g coarse wheat bran or with a 50% increase in fat (total intake approximately 150 g/d). Each consecutive dietary regimen lasted for 3 weeks and one 24-h urine sample was collected during the final week. Dietary analysis, by continuous recorded intake method, showed that the subjects routinely maintained a high-fat (approximately 40% of energy intake) and moderate plant-fibre intake (16–20 g/d). The samples were stored at −20° until analysed for nitrate (Bartholomew, 1984) and *N*-nitrosoproline (NPRO) (Oshima, 1983).

		Diet						
	Subject	C_1	P	C_2	B	C_3	F	C_4
Nitrate excretion (μmol/d)	A	2·20	3·42	3·79	8·80	3·08	1·82	1·36
	B	0·95	2·98	2·31	3·96	4·64	1·69	2·28
	C	1·92	4·00	3·72	5·50	5·24	2·01	1·51
NPRO excretion (μg/d)	A	5·1	ND	ND	8·0	4·8	ND	3·6
	B	2·6	ND	3·2	ND	ND	ND	3·1
	C	28·8	4·7	4·7	45·5	20·9	ND	9·5

C_1, C_2, C_3, control periods; P, pectin supplement (18 g/d); B, bran supplement (30 g/d); F, fat supplement (total intake 150 g/d); ND, not detected.

The urinary excretion of NPRO by the human volunteers (Table) showed considerable inter-individual variation with subject C excreting much greater quantities than subjects A and B. The most marked diet-related effect was that of high-fat consumption, which decreased NPRO output to undetectable levels in all subjects. Dietary pectin also appeared to reduce NPRO excretion. Interestingly, there was no apparent relation between excretion of NPRO and nitrate although nitrate excretion was slightly lower during the high-fat dietary period, suggesting that the inhibitory effect of fat on NPRO formation may be related to nitrate metabolism within the body.

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The effect of the menstrual cycle on patterns of nutrient intake. By E.-L. ORAM (introduced by A. E. BLACK), *Human Sciences Department, Loughborough University, Loughborough, Leics LE11 3TU*

Six women volunteers living in university halls of residence, aged 18–22 years, of normal weight, menstruating regularly, and not using oral contraceptives were studied. They kept weighed dietary records for thirty consecutive days. The volunteers did not know the purpose of the study, and the investigator did not know the timing of menstruation until the end of the data collection.

Premenstrual intakes (days 1 to 10)

Subject No.	Energy (MJ/d)		Protein (g/d)		Fat (g/d)		CHO (g/d)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	13·36	3·77	145	28	127	66	382	93
2	11·03	2·50	119	45	93	34	310	95
3	12·18	2·75	119	20	129	44	314	105
4	12·75	3·17	103	40	133	44	385	117
5	10·18	1·84	93	44	109	57	238	40
6	9·74	3·23	102	33	108	68	234	79
Mean	11·56	1·45	113	16	117	16	311	66
Postmenstrual intakes (days 1 to 10)								
1	10·92	1·76	97	13	85	29	354	84
2	8·31	2·00	74	29	76	27	233	64
3	9·52	1·94	70	34	107	32	281	40
4	11·06	1·83	75	28	115	37	325	75
5	6·83	1·20	84	23	67	24	182	43
6	5·84	1·52	54	21	62	19	176	54
Mean	8·74	2·14	76	14	86	22	259	74

Nutrient intakes were calculated for 10 d preceding menstruation and 10 postmenstrual days (cycle days 1–10). Mean energy intake was 2·82 MJ/d higher in the premenstrual days ($P<0·001$). If menstruation was excluded and comparison made with cycle days 5–14, then the difference was even greater, 3·36 MJ/d. Other authors have reported differences in the same direction (for cycle days 1–10) but smaller (Dalvit (1981): eight students, age 18–22 years, 2·10 MJ/d; Manocha *et al.* (1986): eleven post-graduate students, age 22–30 years, 1·31 MJ/d; Gong *et al.* (1985): nine adults, mean age 30·2 years, 0·90 MJ/d). The differences between studies could be either age-related or socially induced.

A further study of fifteen students (E.-L. Oram and A. E. Black, unpublished results) showed a difference of 0·9 MJ/d (not significant) but this difference was 1·8 MJ/d ($P<0·001$) when the five subjects who did not ovulate were excluded.

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Did Scott simply starve? Lessons from 'The footsteps of the Scott Antarctic expedition'. MICHAEL A. STROUD (introduced by MICHAEL J. STOCK), *Department of Physiology, St George's Hospital Medical School, Tooting, London SW17 0RE*

The winter of 1986 marks the seventy-fifth anniversary of Scott's epic walk to the South Pole, on the return from which he and his four companions perished through cold and poor nutrition. In 1985, three men repeated his route to the Pole, manhauling all their supplies on sledges and using no dogs, depots or transport of any kind. Some of the lessons learned from that journey are presented here.

The trip of 1400 km took 70 d and entailed pulling sledges, that weighed 155 kg, in temperatures down to −35°. The men consumed a daily ration with an uncooked weight of 970 g that provided an average energy intake of 21 MJ/d; 57% from fat, 34% from carbohydrate and 9% from protein. It is unlikely that any previous expedition's diet has provided such a high proportion of energy as fat, but it was well-tolerated nevertheless. Over the journey, the men lost between 6·7 and 10·5 kg, and from this weight loss and the average energy intake over the trip, energy expenditure was estimated to have been about 25 MJ/d. Energy expenditure was also estimated using a diary-card method, with literature values for energy expenditure in the different categories of activity. This method gave a mean expenditure of nearly 29 MJ/d.

Scott's diet has been calculated as providing 18 MJ/d initially, increasing to 19 MJ/d on the Polar plateau, before falling again on the disastrous return from the Pole; 36% of energy intake was from fat, 42% from carbohydrate and 22% from protein. The average daily energy expenditure of Scott's party has been estimated at about 21 MJ/d (Rogers, 1981). Although the discrepancy between his party's energy intake and expenditure has been noted in the past, it has not been felt to be as important a factor in their eventual demise as the concurrent vitamin deficiencies from which they suffered. If, however, their actual expenditure was nearer that estimated on the present expedition, in which similar loads were pulled over the same terrain, then the size of their shortfall has been severely underestimated (equivalent to anything from 6 to 10 MJ/d). Thus, sheer starvation with consequent emaciation might have been the more significant factor. For example, Evans was the biggest man in Scott's team, and could have lost over 15 kg by the time he reached the Pole, let alone what he lost on the return journey.

About 2 weeks after the men reached the Pole on the 1985 expedition, measurements of basal metabolic rate were made that showed a mean 60% increase over the rates measured before their walk. This exceptionally large increase in resting metabolism has not been noted previously, after either prolonged exercise or cold exposure. Its sustained nature and certain other symptoms (e.g. high resting pulse rate, restlessness, heat-intolerance) suggests that it was due to increased thyroid activity.

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