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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Twenty-sixth Meeting of the Nutrition Society was held in the Morris Lecture Theatre, Robin Brook Centre, St Bartholomew's Hospital, West Smithfield, London on Monday and Tuesday, 19/20 *May* 1986, *when the following papers were read:*

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The effect of dietary protein concentration on bone and muscle growth and immunoreactive somatomedin C in the rat. By Z. A. H. YAHYA[1], P. C. BATES[1], SHREEDEVI S. DALAL[1], D. MORELL[2], A. T. HOLDER[2], A. TAYLOR[2] and D. J. MILLWARD[1], [1]*Nutrition Research Unit, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE* and [2]*Department of Growth and Development, Institute of Child Health, 30 Guildford Street, London WC1N 1EH*

In view of the implication of dietary protein deficiency in both stunting (Lampl *et al.* 1978) and the regulation of somatomedin C (SMC; Prewitt *et al.* 1982), we have initiated a study of the interrelation between dietary protein, muscle and bone growth and SMC levels in the rat.

Muscle and bone growth were examined during an 18 d period on rats fed on a diet containing 5 g protein/kg followed by refeeding on diets of 30, 60, 90, 120 and 200 g protein/kg. Linear growth of the tibia was examined in vivo by repeated X-ray measurement, and muscle mass (gastrocnemius, soleus and plantaris) and serum immunoreactive SMC were measured in groups of animals killed at intervals throughout the experiment. The extent to which bone and muscle growth are coordinately regulated was examined by measurement of the muscle mass:bone length value (MBR).

Well-fed rats (130 g) exhibited an immediate cessation of body-weight and muscle growth when fed on the 5 g protein/kg diet but tibial growth continued for 5 d so that the MBR was significantly decreased. Refeeding was initiated, with diets provided *ad lib.*, and measurements made at 3 and 7 d. During the first 3 d in the 60 g protein/kg group there was no increase in muscle mass or tibial length although food intake was similar to that of other groups. In the 90 and 120 g protein/kg groups muscle growth occurred, partially restoring the MBR, whilst the 200 g protein/kg group exhibited both tibial growth and sufficient muscle growth to restore the MBR to that of the original well-fed level. The changes in tibial growth were to some extent correlated with serum SMC which was markedly depressed by the 5 g protein/kg diet and increased after refeeding the 90, 120 and 200 g protein/kg diets in proportion to the protein concentration. During days 4 to 7 muscle mass increased in all groups but with slower growth in the 60 g protein/kg group compared with any other group, even though food intake was highest in this group. Although some tibial growth occurred in all groups during this time, the MBR rose in all except the 30 and 60 g protein/kg group. SMC levels remained depressed in the 60 g protein/kg group but increased in all other groups to within the initial (well-fed) range of values, the highest values occurring in the 120 g protein/kg group. These results will be discussed in relation to a regulatory mechanism involving bone growth as a target for SMC and muscle growth exhibiting a partial dependency on bone growth.

Lampl, M., Johnson, F. E. & Malcolm, L. A. (1978). *Annals of Human Biology* **5**, 219–227.
Prewitt, T. E. A., D'Ercole, A. J., Switzer, B. R. & Van Wyk, J. J. (1982). *Journal of Nutrition* **112**, 145–150.

The effects of inhibiting corticosterone production with Trilostane on protein and energy metabolism in young rats given restricted amounts of dietary protein and energy. By PENNY COYER, P. A. DONACHIE, P. C. BATES, J. P. W. RIVERS and D. J. MILLWARD, *Nutrition Research Unit, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

Cortiscosteroids are generally thought to play an important role in the mobilization of muscle protein for gluconeogenesis during protein–energy deficiency. They also participate in thermoregulation (Deavers & Musacchia, 1979) and in the regulation of heat production. As part of our interest in these joint roles of cortiscosteroids we report here the effects of using the drug Trilostane to inhibit corticosterone production in young rats given restricted diets. Rats were housed at 24° and fed a high- (220 casein/kg; HP) or marginal- (90 casein/kg; MP) protein diet, either *ad lib.* (A) or at 25% of *ad lib.* intake (R). Trilostane (T) was administered twice daily at 50 mg/kg for 7 d.

The effects of Trilostane on body-weight and protein balance were surprisingly small. Both HP and MP energy-restricted rats lost body-weight and body protein, and exhibited reduced muscle protein synthesis. These changes were not modified by Trilostane in rats fed on the MPR diet. On the HPR diet, the rate of weight loss was slightly reduced in the treated rats, as was the rate of protein loss (although the difference between the two groups was small (g/7 d): −1·8 HPR, −0·7 HPRT, not significant). This reflected a partial inhibition of the fall in protein synthesis (15·7% HPA, 4·5% HPR, 6·5% HPRT). This response involved the maintenance of control rates of translation (g protein synthesized/g RNA), although plasma insulin concentrations were unaffected by Trilostane.

Trilostane exaccerbated the fall in rectal temperature which occurred in HPR and MPR rats (37·6° HPA, 36·4° HPR, 35·5° MPR, 35·8° HPRT, 30·8° MPRT). This primarily reflected changes in the rate of heat loss; i.e. heat production (metabolizable energy intake − energy gain) was unchanged by Trilostane when compared on a body-weight basis, although on the HPR diet it was depressed when related to body protein content.

Rats offered the MP diet to appetite ate the same amount of food as HP controls, and heat production was increased. Trilostane administration resulted in a 45% fall in food intake and a fall in heat production. This suggests that the level of energy intake and thermogenesis achieved on MP diets is sensitive to corticosteroids.

These findings are consistent with the suggestion that corticosteroids can act to stimulate heat production, and further imply their importance for thermoregulation in malnourished animals. The results are also in agreement with the previous report that corticosterone suppresses muscle protein synthesis by blocking the stimulatory effect of insulin on translation (Millward *et al.* 1983). The overall effect of Trilostane on protein balance and muscle protein mobilization was nevertheless small, and could not be detected in rats consuming restricted amounts of an MP diet.

We were grateful to Sterling Winthrop for the gift of Trilostane.

Deavers, D. R. & Musacchia, X. J. (1979). *Federation Proceedings* **38**, 2177–2181.
Millward, D. J., Odedra, B. & Bates, P. C. (1983). *Biochemical Journal* **216**, 583–587.

The effect of an anabolic steriod (Durabolin) on body composition of rats fed on protein- and energy-restricted diets. By J. J. CHOO and P. W. EMERY, *Department of Nutrition, King's College (KQC), Campden Hill Road, London W8 7AH*

Testosterone derivatives are known to increase the rate of protein deposition in female animals but not in intact males. However, their effects on fat metabolism are less clear. We have investigated the effect of Durabolin (nandrolone phenylpropionate, Organon Ltd) on body composition in male and female rats under two different dietary conditions: (1) a low-protein diet fed *ad lib.* to increase fat deposition, (2) a normal diet fed in restricted amounts to induce fat mobilization.

Groups of six male (initial weight 200 g) or female (150 g) Sprague-Dawley rats were fed on semi-purified diets containing either 65 g protein/kg fed *ad lib.* (LP) or 195 g protein/kg fed at 70% (female) or 55% (male) of the *ad lib.* intake (FR). These groups of rats received daily, subcutaneous injections of either saline (9 g sodium chloride/l; control) or Durabolin (males and LP females 4 mg/kg body-weight, FR females 2 mg/kg body-weight). The animals were killed after 7–10 d and their carcasses analysed for water (oven drying), fat (Soxhlet extraction with petroleum ether) and crude protein (nitrogen × 6·25; Kjeldahl method).

Treatment	Experimental period (d)	Food intake (g)		Weight gain (g)		Body protein (g)		Body fat (g)	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Low Protein									
♂ Control	10	230	9	38·8	6·8	43·1	1·6	34·7	2·9
♂ Durabolin	10	209	7	26·7	3·9	45·1	1·4	23·4**	1·5
♀ Control	8	129	7	11·8	3·1	27·4	0·4	15·0	0·9
♀ Durabolin	8	101*	7	−1·8*	4·4	25·7	1·0	11·1*	1·3
Food restriction									
♂ Control	8	98	0	−4·1	4·2	42·6	1·4	16·7	0·9
♂ Durabolin	8	98	0	0·3	2·8	45·7	0·9	13·4*	0·9
♀ Control	7	91	0	16·7	1·6	29·3	0·5	14·7	0·6
♀ Durabolin	7	91	0	24·3***	1·3	30·8*	0·4	14·2	0·9

*$P<0.05$, **$P<0.01$, ***$P<0.001$.

In LP male rats Durabolin treatment had no significant effect on weight gain or food intake but it markedly supressed fat deposition without affecting protein deposition. A similar change in body composition was observed in LP females, although in this case Durabolin treatment caused a reduction in weight gain which was associated with reduced food intake. Food restriction completely suppressed growth of male rats: Durabolin had no effect on final body-weight or protein content but did cause a reduction in body fat content. Less severe food restriction allowed some growth of female rats but Durabolin treatment was associated with a significantly greater growth rate which was entirely due to increased lean tissue mass.

Improvement of the ratio, lean:fat tissue in animals of both sexes under adverse nutritional conditions suggests that anabolic steroids might be useful in preventing excessive fat deposition during the treatment of malnourished hospital patients.

Supported by ISFE and the Cancer Research Campaign. The authors are grateful to Organon Ltd for the gift of Durabolin.

Thermic responses to a standard meal in matched smoking and non-smoking female subjects. By S. M. ROBINSON and D. A. YORK, *Department of Nutrition, School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU*

Epidemiological evidence indicates an inverse relation between excess body-weight and cigarette smoking (Khosla & Lowe, 1971). The lower body-weights of smokers may be explained by lower food intakes, higher energy expenditures or both. Cigarette smoking has been shown to be thermogenic (Dallosso & James, 1984) and to increase 24-h energy expenditure (Hofstetter *et al.* 1986).

Resting metabolic rates (RMR), thermic responses over 4 h (TEF) to a standard meal (2·09 MJ, Ensure®) and food intakes were assessed in eight age–weight matched pairs of female subjects (each pair consisted of one smoker and one non-smoker). In six pairs, metabolic rate was measured on a second occasion when the liquid meal was replaced with an equivalent volume of water. Energy expenditure was measured using the ventilated hood method of indirect calorimetry. Food intakes were measured (in six pairs) by the 7 d weighed inventory method and (in two pairs) by 24 h recall. Smoking subjects were allowed to smoke before the RMR measurement and during the rest of the experimental day.

	Body-wt (kg)		Energy intake (MJ/d)		RMR (kJ/kg$^{0.75}$ per d)		TEF (kJ/4 h)		TEF (%RMR)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Smokers	64·0	3·3	7·8	0·3	261	12	209	19	19·5	2·8
Non-smokers	63·9	2·8	7·4NS	0·6	239NS	10	155*	12	17·3NS	1·6

NS, not significant; *$P<0·05$.

The resting metabolic rates of the smokers tended to be higher (9%) than those of the non-smokers. Although non-significant, this may result from the thermogenic effects of the cigarettes smoked before the RMR measurement, or may reflect the slightly greater food intakes of the smokers. The mean increase in metabolic rate (%RMR) in the subjects receiving water was 3·6% (range −0·7 to +8·5%) for the smokers and 1·4% (range −6·9 to +9·3%) for the non-smokers, equivalent to a mean difference of 51 kJ/4h. The smokers' thermic response to the standard meal was almost 35% greater (mean difference of 54 kJ/4 h) than that of the matched non-smokers. It appears likely that smoking and feeding have additive rather than synergistic effects on metabolic rate.

Dallosso, H. M. & James, W. P. T. (1984). *International Journal of Obesity* **8**, 365–375.
Hofstetter, A., Schutz, Y., Jequier, E. & Wahren, J. (1986). *New England Journal of Medicine* **314**, 79–82.
Khosla, T. & Lowe, C. R. (1971). *British Medical Journal* **4**, 10–13.

Possible involvement of prostaglandins in diet-induced thermogenesis of 'cafeteria'-fed rats. By NANCY J. ROTHWELL and MICHAEL J. STOCK, *Department of Physiology, St George's Hospital Medical School, Tooting, London SW17 0RE*

In view of the close similarities between non-shivering thermogenesis and diet-induced thermogenesis (DIT), and the involvement of prostaglandins in the development of febrile responses, we have investigated the effects of prostaglandin synthetase inhibitors on the thermogenic responses to overfeeding.

DIT was induced by presenting young male rats with a choice of highly palatable human food items ('cafeteria' diet) to produce 42–50% increases in metabolizable energy intake. In the first experiment, the acute effect of various drugs was tested on the resting oxygen consumption ($\dot{V}_{O_2}$) of control and 'cafeteria'-fed rats. Average preinjection values for $\dot{V}_{O_2}$ (ml/min per kg body-weight$^{0.75}$) were elevated by about 20–30% in 'cafeteria'-fed (18·42 (SE 0·31)) compared with control rats (14·74 (SE 0·40), $P<0.001$). Acute treatment with either Flurbiprofen (5 mg/kg, subcutaneous (s.c.)) or salicylic acid (400 mg/kg, by mouth) did not significantly affect $\dot{V}_{O_2}$ of controls (5% decrease) but reduced that of 'cafeteria'-fed rats (by 21%) ($P<0.001$). Indomethacin (2 mg/kg), however, had no significant effect on either group and paracetamol (100 mg/kg) caused a modest stimulation of $\dot{V}_{O_2}$ (14–16%).

Chronic treatment of rats with salicylic acid (200 mg/kg s.c. for 14 d) inhibited food intake in stock- and 'cafeteria'-fed rats. The level of hyperphagia was lower in treated 'cafeteria' rats compared with saline (9 g sodium chloride/l)-treated 'cafeteria' rats, but these animals gained 21 g more weight than their stock-fed controls ($P<0.01$), whereas 'cafeteria' feeding failed to affect weight gain in rats injected with saline.

In saline-treated rats, 'cafeteria' feeding enhanced the thermogenic response to noradrenaline (increase in $\dot{V}_{O_2}$: stock diet 8·8 (SE 0·7), 'cafeteria' diet 12·0 (SE 0·9) ml/min per kg body-weight$^{0.75}$, $P<0.05$) and the thermogenic activity of brown fat (mitochondrial purine nucleotide (GDP) binding: stock diet 52 (SE 4), 'cafeteria' diet 88 (SE 6) pmol/mg protein, $P<0.01$). However, these indices were unaffected by 'cafeteria' feeding in rats treated with salicylic acid (noradrenaline response: stock diet 8·2 (SE 0·7) 'cafeteria' diet 9·0 (SE 0·8), not significant; mitochondrial GDP-binding: stock diet 46 (SE 2), 'cafeteria' diet 50 (SE 6), not significant).

These results suggest that prostaglandins are involved in the development of DIT and activation of brown fat in 'cafeteria'-fed rats; the mechanism and site of action of these effects are unknown.

Chronic effects of Baclofen on energy balance and thermogenesis in the rat. By NANCY J. ROTHWELL and MICHAEL J. STOCK, *Department of Physiology, St George's Hospital School, Tooting, London SW17 0RE*

Baclofen is a $GABA_B$-receptor agonist that is used in the treatment of muscle spasm in spasticity. However, we have also observed potent activation of brown adipose tissue (BAT) thermogenesis after central or peripheral administration of Baclofen to rats (Rothwell *et al.* 1985). Injection of 0·5–5 μg Baclofen into the ventromedial hypothalamus of rats causes dramatic increases in BAT temperature (over 2°) and thermogenic activity, and subcutaneous (s.c.) injections (1–5 mg/kg) stimulate oxygen consumption by over 20% in both lean and genetically obese rats.

In the present study, daily treatment with Baclofen for 14 d (3 mg/kg, s.c.) in young, male Sprague-Dawley rats did not significantly affect energy intake (control 1050 (SE 15), Baclofen 1045 (SE 15) kJ/kg body-weight$^{0.75}$ per d) but reduced body-weight gain (control 119 (SE 3), Baclofen 105 (SE 2) g, $P<0.01$) and energy gain (control 905 (SE 35), Baclofen 800 (SE 20) kJ, $P<0.01$). Energy expenditure did not differ significantly between groups, but gross energetic efficiency was lower in Baclofen-treated rats (control 21·2 (SE 0·7), Baclofen 19·1 (SE 0·4)%, $P<0.05$).

Resting O_2 consumption ($\dot{V}_{O_2}$) 24 h after the daily Baclofen injection treatment was similar for both groups, but noradrenaline injection (250 μg/kg body-weight) stimulated $\dot{V}_{O_2}$ to a greater extent in Baclofen-treated rats (% increase in $\dot{V}_{O_2}$: controls 42 (SE 3), Baclofen 55 (SE 4), $P<0.05$). Acute injection of Baclofen stimulated O_2 consumption by about 15–20% in both groups, but this was almost completely abolished by prior β-adrenergic blockade with propranolol (10 mg/kg, s.c.). Interscapular BAT mass was unaffected by chronic Baclofen treatment, but tissue protein content was increased (control 19 (SE 1), Baclofen 23 (SE 1) mg, $P<0.01$) and the thermogenic activity of brown fat, assessed from purine nucleotide (GDP) binding to isolated mitochondria, was elevated by 40% (control 54 (SE 2), Baclofen 76 (SE 1) pmol/mg protein).

These thermogenic responses to peripheral treatment with Baclofen presumably reflect the central actions of the drug observed previously, but other work suggests that these do not involve GABA receptors.

Rothwell, N. J., Addae, J. I., Stock, M. J. & Stone, T. W. (1985). *Journal of Pharmacy and Pharmacology* **37**, 926–927.

Effects of Propranolol on fever, hypothermia and other metabolic effects of *Escherichia coli* endotoxins. By JENNIFER WAN and R. F. GRIMBLE, *Department of Nutrition, School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU*

Sympathetic nervous activity and non-shivering thermogenesis (NST) are important in the development of fever from bacterial endotoxins, since Propranolol is antipyretic (Blatteis, 1976). We have demonstrated both hypothermic and hyperthermic properties of *E. coli* endotoxins in rats (Wan & Grimble, 1986). The present study examines the effect of Propranolol on these effects and other actions of endotoxin on tissue protein and zinc metabolism.

Male Wistar rats (169±1 g), fed on laboratory chow, received either non-pyrogenic saline (9 g sodium chloride/l), or hypothermic (ED) or hyperthermic (ES) varieties of endotoxin (400 μg/kg body-weight; TCA extract, strain 055:B5, Difco Laboratories or butanol extract, strain 0127:B8, Sigma Chemical Co., respectively). Half the rats on each treatment were dosed intraperitoneally with Propanalol, 20 min before saline or endotoxin injections and 2·5 and 5·5 h afterwards (doses 20, 10 and 20 mg/kg body-weight). The remaining animals received saline at these times. Rectal temperatures were measured hourly (*t*0–*t*7) for 7 h. Rats were decapitated 17 h later, when blood and tissues were collected.

Treatment (*n*5) ...	Saline control	Saline control	ES	ES	ED	ED
Propranolol ...	−	+	−	+	−	+
Body temperature (°):						
*t*0	39·4	39·3	39·5	39·7	39·3	39·4
*t*1	38·9	38·8	39·2*	38·8	39·2	38·4†
*t*2	39·1	39·3	40·0	39·5	38·2*	37·0*†
*t*3	—	39·4	39·7	38·7*†	38·4	37·3*†
*t*4	39·2	39·7†	40·0*	39·2†	38·3*	38·1*
*t*5	39·2	39·7†	40·2*	39·7	38·9	38·7*
*t*6	39·2	39·2	40·1*	37·9†	39·1	37·4*†
*t*7	39·1	39·2	39·7*	38·6†	39·1	37·9*†
Liver total protein (g)	0·89	0·92	1·13*	1·16*	1·25*	1·20*
Muscle protein (g/kg)	178	179	157*	162*	158*	162*
Tibialis muscle wt (mg)	362	384	370	348*	322*	346*
Serum Zn (μg/ml)	1·41	1·31	1·12*	1·09*	1·07*	0·92*

*Significantly different from corresponding saline control group: $P<0.05$.
†Significantly different from corresponding group without Propanalol treatment: $P<0.05$.

Propranolol did not influence the action of the endotoxins on tissue protein or serum Zn. NST appeared to be essential for rises in temperature during fever and recovery from hypothermia. Propranolol effects were dose-dependent. In ED rats the 10 mg/kg dose had little effect on recovery from hypothermia, whereas the second 20 mg dose delayed recovery. In ES rats, the fever was blunted by Propranolol, and hypothermia developed following the third dose. Kluger *et al.* (1981) have postulated that a cryogenic (hypothermic) factor appears in endotoxin-treated animals in addition to 'fever promoting' interleukin 1. The ultimate temperature achieved after endotoxin administration may therefore depend on the level of sympathetic activity, unlike changes in protein and Zn metabolism.

Blatteis, C. M. (1976). *Journal of Applied Physiology* **40**, 35–39.
Kluger, M. J., Turnbull, A. J., Cranston, W. I., Wing, A. J., Gross, M. P. & Rotherburg, B. A. (1981). *American Journal of Physiology* **241**, R271–R276.
Wan, J. & Grimble, R. F. (1986). *Proceedings of the Nutrition Society* **45**, 83A.

Response of the vitamin-E-deficient rat to severe protein deficiency and 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*

The oedema and other characteristic symptoms of kwashiorkor have been postulated to reflect unsuppressed free-radical production and subsequent oxidative damage (Golden, 1985). To explore this hypothesis we have initiated an investigation of the effect of impaired anti-oxidative capacity on the response of the rat to malnutrition and infection.

In the current experiments vitamin E deficiency was induced by feeding weanling rats on a vitamin-E-deficient diet (200 g protein/kg) for 4–6 weeks with confirmation of the deficiency by measurements of plasma tocopherol concentrations, erythrocyte haemolysis and lipid peroxidation in the liver. The vitamin-E-deficient and control rats were fed on a low-protein diet (5 g lactalbumin or casein/kg) for 4 weeks (120 g body-weight) or 12 weeks (160 g body-weight) in an attempt to induce oedema. No oedema was observed in either group: indeed the vitamin-E-deficient rats seemed better protected from the protein deficiency as judged by their increased survival compared with control rats.

We then examined the acute response of vitamin-E-deficient and control rats (200 g) to increasing doses of the *Escherichia coli* lipopolysaccharide endotoxin (strain 0127:B8, LPS: 1·5, 3 and 4 mg/kg body-weight). Each group exhibited a dose-related fall in food intake, loss of body-weight, inhibition of muscle growth and protein synthesis with increased liver size and protein synthesis as previously shown (Jepson *et al.* 1986). Although endotoxaemia involves an oxidative stress, there was little indication in the vitamin-E-deficient group of increased susceptibility to the LPS in terms of the catabolic response, in part because the depression of food intake was only half that of the control rats. Hepatic protein synthesis was less elevated in the vitamin-E-deficient rats and livers were larger, possibly reflecting a combination of a blunted acute-phase response (i.e. less export-protein synthesis), and the better-maintained food intake. Paradoxically the extent of hepatic lipid peroxidation, a feature of the vitamin-E-deficient rats, as estimated by the thiobarbituric acid method (Bieri & Anderson, 1960), was reduced by the LPS treatment. Clearly, in terms of the indices we have examined so far, the vitamin-E-deficient rat does not exhibit any increased sensitivity to nutritional insults or oxidative stress.

Bieri, J. D. & Anderson, A. A. (1960). *Archives of Biochemistry and Biophysics* 19, 105–110.

Golden, M. (1985). In *Nutritional Adaptation in Man*, pp. 169–188 [K. Blaxter and J. C. Waterlow, editors]. London: John Libbey.

Jepson M. M., Pell, J. M., Bates, P. C. & Millward, D. J. (1986). *Biochemical Journal* 235, 329–336.

Copper deficiency and plasma glutathione concentration in rats. By K. G. D. ALLEN, J. R. ARTHUR, N. T. DAVIES, P. C. MORRICE, F. NICOL and C. F. MILLS, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Adequate dietary copper is necessary for antioxidant protection mediated by cytosolic superoxide dismutase (*EC* 1.15.1.1). Glutathione (GSH) protects against oxidative damage by scavenging reactive radical species and as a substrate of selenium-dependent GSH peroxidase (*EC* 1.11.1.9). Previous work has shown a decrease in GSH peroxidase in Cu deficiency (Jenkinson *et al.* 1982) and an increase in hepatic GSH release to plasma in Se-deficient rats (Hill & Burk, 1985).

We have investigated the relation between plasma GSH and dietary Cu deficiency. Rats were fed on a Cu-deficient diet (<0·2 mg Cu/kg) for 7 weeks from weaning. Control rats (both *ad lib.* and pair-fed) were given the same diet supplemented with Cu to 10·00 mg Cu/kg. Blood was obtained under diethyl ether anaesthesia from the left ventricle of the heart and from the renal veins, and anticoagulated with EDTA. Blood was centrifuged, and the plasma deproteinized with 5-sulphosalicylic acid. Plasma GSH was measured within 5 min of drawing blood by a modification of the Tietze recycling assay (Griffith, 1980).

Influence of Cu deficiency on total plasma GSH (μM)

	Cu deficient (*n* 6)		Pair-fed control (*n* 8)		*Ad lib.*-fed control (*n* 7)	
	Mean	SD	Mean	SD	Mean	SD
Renal veins	10·20**	2·21	6·78	2·30	7·19	1·62
Heart, left ventricle	24·40*	11·22	16·37	2·95	15·59	1·59

Significantly different from *ad lib.*-fed and pair-fed controls (analysis of variance): *$P<0·05$ (heart), **$P<0·01$ (renal).

The results suggest either a role for Cu in GSH metabolism, or an adaptation of GSH metabolism to the compromised antioxidant systems in Cu deficiency.

K.G.D.A. is grateful to Colorado State University and the Rowett Institute for sabbatical leave.

Griffith, O. W. (1980). *Analytical Biochemistry* **106**, 207–212.
Hill, K. E. & Burk, R. F. (1985). *Archives of Biochemistry and Biophysics* **240**, 166–171.
Jenkinson, S. G., Lawrence, R. A., Burk, R. F. & Williams, D. M. (1982). *Journal of Nutrition* **112**, 197–204.

The influence of carbohydrate availability on the regulation of lipogenesis in the lactating mammary gland of the rat. By S. W. MERCER and D. H. WILLIAMSON, *Metabolic Research Laboratory, Nuffield Department of Clinical Medicine, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE*

High rates of lipogenesis are observed in the mammary gland of the lactating rat which can be suppressed by short-term starvation and rapidly reactivated by refeeding chow diet (Bussman *et al.* 1984; Jones *et al.* 1984). However, the regulation of the rapid changes in metabolism in the gland during the starved-refed transitions is not well understood. In the present study we have investigated the importance of carbohydrate availability in signalling to the gland during refeeding chow diet, by use of a glucosidase inhibitor, acarbose, and by gastric intubations of graded amounts of glucose.

Primiparous lactating rats of the Wistar strain (11–16 d of lactation) were fasted for 18 h. Refeeding 5 g chow diet led to a twenty-fold increase in lipogenesis in the mammary gland within 90 min, measured in vivo with ^3H_2O. However, intubation of acarbose led to a 75% suppression of the reactivation in the gland of chow-refed rats. In vitro studies revealed higher rates of [1-^{14}C]glucose incorporation into lipid and carbon dioxide in mammary gland acini isolated from chow-refed rats compared with acini from starved animals, but acarbose administration during refeeding completely prevented this augmentation of glucose metabolism.

Gastric intubation of 6 mmol glucose 75 min before killing led to a large stimulation of lipogenesis in vivo in the mammary gland of starved lactating rats, (refed, 73·0 (SE 6·7) μmol ^3H_2O incorporated/h per g; 6 mmol oral glucose, 60·3 (SE 7·8), not significant). However, this response was dependent on both the amount of glucose given and on the time elapsed between glucose loading and injection of ^3H_2O.

This switch-on of lipogenesis in the mammary gland of starved lactating rats by refeeding chow diet, or by glucose intubation, was associated with a decrease in the ratio, glucose 6-phosphate (G6P): fructose 1, 6-bisphosphate (F1,6BP) in the gland, indicating an increase in the activity of phosphofructokinase (PFK). Acarbose treatment during refeeding prevented this change in the G6P:F1,6BP value. The concentration of 6-phosphogluconate (an activator of PFK) was decreased in the mammary gland by starvation, but 90 min refeeding did not increase the concentration. The concentration of citrate (an inhibitor of PFK) in the gland did decrease to fed levels during refeeding. However, acarbose-treated refed rats also showed a decrease in citrate in the gland and yet maintained a high G6P:F1,6BP value and a low rate of lipogenesis; thus citrate is unlikely to be the key regulator of PFK activity.

We conclude that continual carbohydrate availability, rather than natural food consumption *per se*, is the major factor in the reactivation of lipogenesis in the mammary gland during the starved-refed transition. An important site of regulation of lipogenic activity in the gland appears to be at the level of PFK, although the physiological regulator of this enzyme during refeeding remains to be elucidated.

Bussman, L. E., Ward, S. & Kuhn, N. J. (1984). *Biochemical Journal* **219**, 173–180.
Jones, R. G., Ilic, V. & Williamson, D. H. (1984). *Biochemical Journal* **223**, 345–351.

A study of the possible role of the intestinal microflora in phytate hydrolysis in chicks. By A. MOHAMMED, R. F. GRIMBLE and T. G. TAYLOR, *Department of Nutrition, School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU* and B. RATCLIFFE, *AFRC Institute of Food Research, Reading Laboratory, Shinfield, Reading RG2 9AT*

The (USA) National Research Council (1971) reported that approximately 30% of the phosphorus in plant materials is non-phytate and can be considered to be utilized by chickens. The remaining 70% is in the form of phytate and must be hydrolysed by phytase to become available. There are three sources of this enzyme (1) plant, (2) intestinal, (3) microbial. In the present study an attempt was made to determine the relative contribution of the three sources of enzyme to the hydrolysis of dietary phytate. Phytase-containing and phytase-free diets were given to germ-free (GF) chicks and conventional (CV) controls. Phytase activity was removed by autoclaving. Diets provided 12·6 MJ metabolizable energy/kg; 195 g crude protein (nitrogen × 6·25)/kg; 5·58 g total P/kg of which 3·42 g was phytate P; 5 g calcium/kg; 1·25 mg vitamin D_3/kg. Eight pairs of male GF chicks were hatched and reared for 4 weeks in stainless-steel isolators. A further four pairs were studied in identical conditions but with conventional gut flora. Digestibility of phytate and total P was determined from a collection of droppings over a 3 d period during the 4th week (see Table).

Chick status ...	GF		CV		Pooled standard deviation (8 df)
Dietary phytase	−	+	−	+	
No. of pairs	4	4	2	2	
Final body-weight (g)	678	656	708	604	89·2
Total P digestibility	0·357	0·357	0·426	0·369	0·0448
Phytate P digestibility	0·802	0·936	0·796	0·911	0·0193

The intestinal microflora had little or no role in the hydrolysis of dietary phytate under these conditions and total P digestibility was unaltered by the treatments. The digestibility of phytate was higher when dietary (plant) phytase was present regardless of the bacteriological status of the gut. Intestinal phytase seems to hydrolyse about 80% of the phytate and this can be enhanced by the addition of dietary (plant) phytase which in this case improved digestibility by 10–13% ($P<0·001$ in both environments).

National Research Council (1971). *Nutrient Requirements of Domestic Animals. 1. Nutrient Requirements of Poultry*. Washington DC: National Academy of Sciences.

Effect of oral chlortetracycline on faecal DNA excretion and mucosal cell turnover in rats fed on legumes. By SANOJA S. SANDARADURA and A. E. BENDER, *Department of Nutrition, King's College (KQC), Campden Hill Road, London W8 7AH*

We have explained the low digestibility of legume protein as being largely due to an increased excretion of endogenous DNA resulting from an increased rate of turnover of the mucosal cells of the digestive tract (Sandaradura & Bender, 1985). Although we postulated that only a part of the faecal DNA was likely to come from intestinal bacteria the contribution from this source was not clear. This was examined by suppressing bacterial growth by administering chlortetracycline and measuring total DNA excretion.

Thirty-two male Lister Hooded weanling rats were divided into four groups of eight. Animals were fed *ad lib.* for 21 d on diets containing cooked whole white kidney beans (*Phaseolus vulgaris*) or casein at 200 g protein/kg diet, either with or without supplementation with chlortetracycline hydrochloride (30 mg/kg diet). Food intakes, faecal N and faecal DNA were measured.

On the 21st day the rats were injected intraperitoneally with colchicine (1 mg/kg body-weight). Pieces of intestine, each 20 mm in length, were cut 200 mm from the pylorus end, slit open and fixed in formal-saline before embedding, sectioned at 5μm thickness and stained with Feulgen's reagent. Metaphases in arrested cells from twenty crypts per animal were counted.

Diet ...	Bean				Casein			
	Control		Added chlortetracycline		Control		Added chlortetracycline	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Faecal N (mg/g food)	6·89**	0·31	6·10**	0·31	4·53	0·16	4·02	0·20
Faecal DNA (mg/g food)	1·19**	0·09	1·09**	0·08	0·53	0·03	0·51	0·03
No. of metaphases/ crypt	29**	0·5	28**	0·4	15	0·6	14	0·5

**Significantly different from control: $P<0·01$.

Supplementation with chlortetracycline did not markedly alter the faecal N, DNA excretion or the mucosal cell division in rats fed on the bean diet.

Supported by a grant from the I.S.F.E. Foundation, Switzerland.

Sandaradura, S. S. & Bender, A. E. (1985). *Proceedings of the Nutrition Society* 44, 30A.

Long-term results of treatment of severe obesity with jaw wiring and waist cord. By J. S. GARROW and JOAN D. WEBSTER, *Nutrition Research Group, Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ*

Jaw wiring for the treatment of obesity was first described by Garrow (1974) but unfortunately obese patients almost inevitably regain the weight they have lost unless fitted with a nylon cord around the waist when the jaws are unwired (Garrow & Gardiner, 1981). We therefore do not treat patients by jaw wiring unless they agree to have a waist cord fitted when the jaw wires are removed, and to wear this for an indefinite period.

We now report a follow-up in the thirty-eight obese patients who have been treated in this way since September 1979. Nine patients dropped out during the jaw-wiring phase, and another twelve did not tolerate the waist cord or preferred a gastric bypass operation to assist them in maintaining their reduced weight. We have followed the remaining fourteen patients for 1–6 years, and also another three patients who had lost 40 kg by dieting without jaw wiring and who opted to have a waist cord fitted to help them maintain weight loss.

Characteristics of thirty-eight obese patients (thirty-six female, two male) treated by jaw wiring and waist cord

Jaw wiring . . .	Dropped out		Completed		Completed		Not done	
Waist cord . . .	Not applied		Dropped out		Continuing		Continuing	
n. . .	9		12		14		3	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (years)	30·6	6·3	31·8	8·7	27·2	7·3	35·7	4·9
Wt (kg)	101·9	17·8	111·8	15·5	118·0	11·7	118·3	11·7
Wt/height^2 (kg/m^2)	40·0	4·1	43·4	5·6	46·4	5·2	43·5	4·1
Excess wt (kg)	38·4	12·9	47·3	13·8	54·1	11·2	50·3	11·2
Duration of wiring (months)	—	—	9·0	3·1	11·1	3·6	—	—
Wt lost (kg)	—	—	36·8	11·1	42·4	7·4	41·7	16·5
Follow-up (months)	—	—	—	—	35·6	22·3	20·7	3·1
Overall wt loss (kg)	—	—	—	—	32·8	12·7	33·0	12·1
Overall wt loss (as % excess)	—	—	—	—	61·9	23·4	64·3	11·0

At present about 78% of the maximum weight lost with jaw wiring (about 62% of the original excess weight) is maintained on follow-up. The waist cord enables about half the jaw-wired patients to maintain most of their weight loss without recourse to abdominal surgery.

Garrow, J. S. (1974). *Proceedings of the Nutrition Society* **33**, 29A.
Garrow, J. S. & Gardiner, G. T. (1981). *British Medical Journal* **282**, 858–860.

A nutritional evaluation of the tuber of the plant *Cyperus rotundus*. By J. C. BULMAN, D. J. NAISMITH, *Department of Nutrition, King's College (KQC), Campden Hill Road, London W8 7AH*, and G. HILLMAN, *Institute of Archaeology, 31–34 Gordon Square, London WC1H 0PY*

Famine, which is at present rife in Africa, gives an indication of the ability of undernourished man to survive on plants which have been hitherto regarded as having little or no nutritive value, i.e. those with a high content of dietary fibre and, in some cases, woody tissue and the presence of toxins. There are many such species growing in North Africa which have been used for millenia as food sources (Maurizio, 1972). Recent advances in archaeological knowledge give an indication of the types of plant eaten in these regions. One example is *Cyperus rotundus*, an extremely prolific plant, now regarded as a weed. Attempts, unsuccessful as yet, are being made to eradicate it. *Cyperus rotundus* (Purple Nut Sedge), a member of Cyperaceae, inhabits much of the moist and marshy tropics and sub-tropics. Reproduction is mainly by means of small, nutlike tubers, growing on thin rhizomes which spread rapidly under tillage.

There is merit in looking again at sources of human food which were formerly eaten but no longer form part of the diet, and examining their nutritional potential, either in their fresh state or after cooking.

We have examined the composition of the tubers of *Cyperus rotundus* in some detail. Composition (g/kg, all on fresh weight basis) was: moisture 593, protein 23, fat 16 (volatile oil 6 g/kg total tuber), dietary fibre 160, carbohydrate 200, ash 8, of the minerals analysed (/kg): calcium 340 mg, magnesium 390 mg, sodium 380 mg, potassium 1·76 g, iron 120 mg. Gross energy was 7·9 MJ/kg. A low-protein content, but a high dietary fibre: starch value, with abundant Fe is noteworthy, implying restriction in the tuber's use as a major energy source but its satisfactory incorporation into the diet as one-quarter to one-fifth of total daily dietary energy.

The tubers contain non-nutritional factors which have various pharmacological effects, i.e. bacteriocidal, antihelminthic (Täkholm & Drar, 1950; Watt & Breyer-Brandwijk, 1962), which may offer protection to man especially in adverse circumstances. At present, information on digestibility and availability of nutrients is not available. Further studies on the nutritive value and other properties of the tuber in animals and man would be most worthwhile.

Maurizio, A. (1972). *Die Geschichte Unserer Pflanzennahrung*, Part 1. Berlin: Paul Parey.

Täkholm, V. & Drar, M. (1950). *Flora of Egypt*, vol. 2, pp. 74–76. Cairo: Cairo University Press.

Watt, J. M. & Breyer-Brandwijk, M. G. B. (1962). *Medicinal and Poisonous Plants of Southern and Eastern Africa*, 2nd ed., p. 374. London: Livingstone.

Effect of a 10 week jogging programme on metabolic rate in six lean sedentary females. By SUSAN LAWSON, JOAN WEBSTER, P. J. PACY and J. S. GARROW, *Nutrition Research Group, Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ*

A 10 week programme of jogging which has been advocated for the treatment of mild obesity (Cannon & Einzig, 1983) was undertaken by six lean, formerly sedentary females aged 24–35 years, weight:height^2 20·3–24·5. The intensity of physical exercise was increased each week (7–77 min/week) as permitted by increasing physical fitness. Food intake was not restricted. The protocol was approved by the Northwick Park Hospital Ethical Committee.

Fitness increased significantly ($P<0.01$): the time to cover 1·6 km (1 mile) decreased from 10·2 (SD 0·6) to 8·5 (SD 0·4) min, and distance covered in 12 min increased from 1857 (SD 116) to 2217 (SD 117) m. The effect on weight, resting metabolic rate (RMR) and resting heart rate (RHR) is shown in the Table.

Week ...		0	2	4	6	8	10
Weight (kg)	Mean	60·3	60·2	60·7	60·7	60·8	60·9
	SD	8·8	9·2	9·3	9·6	9·7	9·8
RMR (ml O_2/min)	Mean	213·2	222·3	237·7*	222·5	219·0	241·5**
	SD	10·4	19·4	21·8	7·1	18·7	9·0
RHR (beats/min)	Mean	97·2	81·7	77·0*	86·9	86·7	79·1*
	SD	14·6	12·8	12·5	10·5	19·2	9·2

Significantly different from week 0 (paired t test): *$P<0.05$, **$P<0.01$.

There was no effect of the exercise on body-weight, nor did measurements of total body water, potassium and density show any significant change in body composition. At weeks 4 and 10 (but not at the other measurement times) RMR was increased and RHR decreased significantly compared with week 0. The decrease in RHR is probably associated with increased physical fitness, but the increased RMR is more difficult to interpret. It may be an effect of exercise, or of increased food intake. Although food intake was not measured, body-weight slightly increased over a period when energy expenditure significantly increased, so energy intake probably increased also.

The volunteers did not enjoy the exercise programme, and did not intend to continue after the end of the study, although they were pleased by their increased fitness. This form of exercise alone is unlikely to be a satisfactory treatment for mild obesity.

Cannon, G. & Einzig, H. (1983). *Dieting Makes You Fat*, pp. 170–185. London: Century Publishing.

An association of anaemia with poor vitamin D status in otherwise adequately nourished Asian toddlers: a case for combined prophylaxis. By H. GRINDULIS[1], P. H. SCOTT[1], N. R. BELTON[2] and B. A. WHARTON[1], [1]*Sorrento Maternity Hospital, Birmingham B13 9HE* and [2]*Department of Child Life and Health, Edinburgh EH9 1UW*

Many aspects of infant feeding in Britain have improved since the publication of *Present Day Practice in Infant Feeding* in 1974 (Oppé *et al.* 1974), but there are still some areas of concern and one of these, voiced in the subsequent report 6 years later, was the problem of weaning in Asian infants (Oppé *et al.* 1980).

145 Asian children born at the Sorrento Maternity Hospital were reviewed at the age of 22 months. A significant association of iron deficiency and poor vitamin D status was found which could have been due to poor socio economic status. 40% of the children were anaemic (haemoglobin (Hb) <110 g/l), 40% had a low plasma concentration of 25-hydroxy cholecalciferol (<10 ng/ml) and 20% had both features. This was more than simple overlap of the two deficiencies; the children with low plasma 25-hydroxy cholecalciferol concentrations had significantly lower concentrations (mean and SD) of Hb (109 (16) *v.* 115 (13) g/l, $P<0.05$) and serum Fe (12·9 (5·6) *v.* 15·4 (6·7) μmol/l, $P<0.05$). On the other hand, the deficiencies were not merely individual features of generally poor nutrition; growth and other measures of protein–energy nutrition were slightly better in these children (weight standard deviation score +0·2 *v.* +0·1, not significant; subscapular skinfold 7·0 (1) *v.* 6·3 (1·5) mm, $P<0.05$; plasma albumin 44 *v.* 43 g/l, not significant; plasma alkaline ribonuclease (*EC* 3.1.4.22) 480 (73) *v.* 520 (80) units/l, $P<0.05$) and their plasma zinc was no lower than that in the non-deficient children (10·0 *v.* 10·2 μmol/l).

It seems, therefore, that child-health surveillance as currently practised (e.g. growth monitoring, clinical signs, etc.) will not detect these problems unless a Hb determination is included. In view of the association of poor Fe and vitamin D status, combined prophylaxis is desirable. At present, strategies for preventing rickets in this country are not combined with attempts to detect or prevent Fe deficiency. In our opinion they should be.

The psychomotor development of the anaemic children was slightly, but significantly, delayed. This has been noted elsewhere but our investigation had not been designed to study psychomotor development in any detail and so no conclusions are drawn, but we are carrying out an intervention study to investigate this phenomenon further.

Oppé, T. E., Arneil, G. C., Creery, R. D. G., Lloyd, J. K., Stroud, C. E., Wharton, B. A. & Widdowson, E. M. (1974). *Present Day Practice in Infant Feeding. Report on Health and Social Subjects* no. 9. London: H.M. Stationery Office.

Oppé, T. E., Arneil, G. C., Davies, D. P., Laurance, B. M., Lloyd, J. K., Stroud, C. E., Wharton, B. A. & Widdowson, E. M. (1980). *Present Day Practice in Infant Feeding. Report on Health and Social Subjects* no. 20. London: H.M. Stationery Office.

Iron therapy (with vitamin C) increases the rate of weight gain and psychomotor development of anaemic toddlers: a double blind randomized controlled trial. By A. AUKETT, Y. A. PARKS, P. H. SCOTT and B. A. WHARTON, *Sorrento Maternity Hospital, Birmingham B13 9HE*

Previous work at this hospital (Grindulis *et al.* 1986) and elsewhere has shown that anaemia in toddlers is common and is associated with psychomotor delay. However, it seemed unclear whether this association was cause and effect or merely due to the same underprivileged environment. A double blind randomized intervention study was therefore performed.

470 children, aged 17–19 months and living in inner Birmingham were studied. 25% had a haemoglobin (Hb) concentration <110 g/l. Following an initial haematological, anthropometric and developmental assessment, ninety-seven of these anaemic children (Hb 80–110 g/l) received either iron (24 mg) and vitamin C (10 mg) daily, or vitamin C only (control group) for 2 months and were then reassessed.

The children were assessed with twenty-four items taken from the Denver Developmental Screening Test (DDST) appropriate for the age group studied and presented in the standard manner. The project interpreter was used as necessary. We recorded the number of skills which the child had achieved; a maximum of twenty-four. Children who are average in every respect would achieve thirteen items at 18 months and nineteen at 20 months, i.e. in the normal course of development they would be expected to achieve six more of the psychomotor skills tested during the 2 months of the study.

The children who received Fe plus vitamin C had an increased rate of weight gain (mean and SD: 10·0 (6·0) *v.* 5·7 (6·0) g/d, $P<0·005$) and more of them achieved the expected rate of psychomotor development (31 *v.* 12%, $P<0·05$). This average rate of development was achieved by more of the children whose Hb rose by at least 20 g/l than by those whose Hb did not rise substantially (37 *v.* 16%, $P<0·05$).

It seems that if anaemic children are treated, more of them develop at a normal rate. We conclude that there is a causal link between Fe deficiency and psychomotor development, it is not just a matter of association. However, there were still a number of children who, despite treatment, did not improve greatly.

Whilst Fe deficiency anaemia is unlikely to be the only factor in the slower development of children living in underprivileged circumstances, it can at least be easily identified and treated. Routine child-health surveillance in such areas should include haemoglobin determination.

Grindulis, H., Scott, P. H., Belton, N. R. & Wharton, B. A. (1986). *Proceedings of the Nutrition Society* **45**, (this meeting).

The effect of hypoenergetic feeding for 2 weeks on the contractile properties of the adductor pollicis of obese patients. By D. J. NEWHAM[1], A. M. TOMKINS[2] and C. G. CLARK[1], [1]*Department of Surgery, University College Hospital, London WC1*, and [2]*Department of Human Nutrition, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

It has been suggested that the contractile properties of skeletal muscle accurately reflect small and rapid changes in nutritional status which are otherwise undetectable. Abnormalities of the adductor pollicis have been reported in malnourished patients (Lopes *et al.* 1982) and are claimed to be rapidly reversed by oral refeeding (Russell *et al.* 1983*b*). Similar abnormalities have been reported in six obese patients after 2 weeks on a diet of 1670 kJ (400 kcal)/d (Russell *et al.* 1983*a*). In view of the potential importance of these findings we have studied a larger group of obese patients on a comparable dietary regimen.

Twenty-one obese patients (age 25–42 years) were hospitalized for 2 weeks and maintained on 1880 kJ (450 kcal)/d. On days 1, 7 and 12 the contractile properties of the adductor pollicis were studied using supramaximal stimulation of the ulnar nerve. Twenty normal subjects and three malnourished patients (without systemic disease) were also studied.

There was a significant weight loss in the obese patients, but no change in the contractile properties. Compared with the normal subjects they were less fatiguable (Binke *et al.* 1974). The malnourished patients showed the abnormal force:frequency relation, slow relaxation rate and increased fatiguability as described in the literature.

	Body-wt (kg)		Force:frequency (10/100%)		Maximum relaxation rate (% force loss/ 10 ms)		Fatiguability (force loss % initial)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Obese								
Period on diet (d):								
1	117·2	19·1	33·0	9·5	10·9	1·1	32·7	9·8
7	112·5	19·1	34·6	9·4	10·6	2·3	38·6	11·2
12	111·5	18·8	35·7	10·7	10·7	2·0	35·7	9·8
Normal	—	—	28·7	9·5	10·1	1·1	49·3	9·6
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Malnourished	35·9	33·2–40·0	55·7	51·7–58·1	8·0	7·4–8·3	57·8	43·0–68·7

These results do not agree with those of a similar study (Russell *et al.* 1983*a*). This raises doubts about the use of skeletal muscle function tests to detect small changes in nutritional status in obesity, although abnormal results seem to be found in malnourished patients. The role of such tests to monitor small changes in the nutritional status of obese patients is uncertain and consequently they must be used with caution.

Binke, R. E., Levine, D. N., Saleman, M. & Tsaeiis, P. (1974). *Journal of Physiology* **238**, 503–514.

Lopes, M. D., Russell, D. R., Whitwell, J. & Jeejeebhoy, K. N. (1982). *American Journal of Clinical Nutrition* **36**, 602–610.

Russell, D. R., Leiter, L. A., Whitwell, J., Manuss, E. B. & Jeejeebhoy, K. N. (1983*a*). *American Journal of Clinical Nutrition* **37**, 133–138.

Russell, D. R., Prendergast, P. J., Darby, P. L., Garfinkel, P. E., Whitwell, J. & Jeejeebhoy, K. N. (1983*b*). *American Journal of Clinical Nutrition* **38**, 229–237.

The use of transferrin and ferritin as 'nutritional' indices. By A. W. JOHNSON[1], A. G. RADCLIFFE[2], A. GOODE[3] and H. A. F. DUDLEY[2], [1]*Department of Medicine, St James's Hospital, Leeds,* [2]*Surgical Unit, St Mary's Hospital, London W2,* [3]*Surgical Unit, The London Hospital, London E1*

Transferrin has often been used as an indicator of nutritional status because of its short biological half-life. However, little attempt has been made to correlate the physiological role of transferrin with that of a nutritional index. The aim of the present study was to investigate the role of transferrin as a nutritional index whilst taking into account other indices of iron metabolism.

Sixty-two consecutive patients, attending hospital for moderate to severe abdominal surgery, were assessed before surgery for body-weight (% ideal body-weight), and serum transferrin, serum ferritin and blood haemoglobin concentrations. Twenty-eight patients were less than 90% ideal body-weight and constituted the malnourished group, the other thirty-four patients were considered to be nutritionally normal.

When serum transferrin concentrations were ranked whilst not disclosing the identities of the malnourished patients, there was a significant difference in the distribution of the two patient groups. However, the data for the malnourished group were sufficiently skewed to make this variable unreliable as a nutritional index by itself. When serum ferritin and blood haemoglobin were taken into account the malnourished group could be delivered into three sub-groups: group 1, six gastric ulcers, one duodenal ulcer; group 2, three gastric ulcers, two duodenal ulcers, five large-bowel cancers, two Crohn's disease; group 3, one gastric ulcer, two duodenal ulcers, two gastric cancers, two large-bowel cancers, one pancreatitis, one carcinoid syndrome. The normal group comprised eleven cholecystectomies, seven highly selective vagotomies, six laparotomies, five large-bowel cancers, four appendectomies and one duodenal ulcer.

Group . . .	Normal (*n* 34)		1 (*n* 7)		2 (*n* 12)		3 (*n* 9)	
	Mean	2SD	Mean	2SD	Mean	2SD	Mean	2SD
% Ideal body-weight	104	8	84	9	86	4	86	10
Transferrin (mg/l)	247	75	311	60	163	50	156	80
Ferritin (ng/l)	74	52	33	30	115	90	380	110

The observations in malnourished group 1 are consistent with anaemia, making the use of transferrin as a nutritional marker inappropriate. Group 2 had no obvious disturbance in Fe metabolism, with transferrin being a good nutritional index. However, group 3, although having a nutritionally appropriate transferrin concentration, demonstrated abnormalities in the other indices of Fe metabolism; this group had a high mortality rate (8/9). Thus, given a normal Fe metabolism, transferrin can be considered as a nutritional index. Furthermore, consideration should be given to the use of measurements of Fe metabolism as prognostic indices.

The metabolic response to prolonged walking exercise in fed and fasted man. By P. L. GREENHAFF, K. MCCORMICK and R. J. MAUGHAN (Introduced by M. GLEESON), *Department of Environmental and Occupational Medicine, University Medical School, Aberdeen AB9 2ZD*

In studies of the metabolic response to very prolonged, low-intensity exercise, only information relating to low-energy intake has been reported (Carlson & Froberg, 1967; Marniemi *et al.* 1984). The present study reports patterns of substrate mobilization for subjects on a 'mixed' diet (g/kg diet: 480 carbohydrate, 370 fat, 150 protein) and in a fasted state during prolonged walking.

Six male subjects walked 37 km/d, at a workload requiring 17 (SE 1)% of maximal oxygen uptake, for four consecutive days over a flat, three-lap course. During this time an unrestricted mixed diet was consumed pre- and post-exercise and all dietary intake was weighed on electronic balances. Mean daily energy intake amounted to 14·5 (SE 0·8) MJ and daily energy expenditure was calculated to be approximately 12·0 MJ. At a later date, a 1 d walk took place over the same course with the subjects exercising in a fasted state. Blood glucose concentration fell significantly on the 1st, 3rd and 4th days of the fed walk ($P<0·05$) but no subject became hypoglycaemic. Glucose concentration did not fall during the fasted walk and was significantly higher pre-exercise and at the end of laps one and three when compared with day 1 of the fed walk. Blood alanine concentration fell significantly ($P<0·05$) from the end of the first lap to the end of each day's walking during the fed walk; such a fall was not seen during the fasted walk. Blood lactate levels did not change during the course of either walk. Plasma free fatty acids, glycerol and blood β-hydroxybutyrate concentrations were unchanged during the first lap on each day of the fed walk, but increased by the end of the first lap of the fasted walk ($P<0·01$); at the end of lap one of the fasted walk significant elevations ($P<0·01$) were observed when compared with the fed walk. The increases from rest to the end of each day's walking for each of these three metabolites were of similar magnitude when comparing each day of the fed walk and the fed and fasted walks.

The present experiment indicates, firstly, that the pattern of mobilization of fuel substrates is highly repeatable from one day to the next providing dietary intake is similar. Secondly, food intake before and between exercise bouts may reduce fat mobilization and possibly increase blood glucose utilization in a similar manner to that encountered during more strenuous exercise.

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Effects of pre-exercise feeding of glycerol or glucose on metabolism and endurance performance in man. By M. GLEESON, R. J. MAUGHAN and P. L. GREENHAFF, *Department of Environmental and Occupational Medicine, University Medical School, Aberdeen AB9 2ZD*

Intermittent carbohydrate feeding during prolonged, strenuous submaximal exercise can spare muscle glycogen and delay fatigue (Coyle *et al.* 1983). In contrast, glucose ingestion 30–60 min before the onset of exercise appears to have adverse effects on endurance performance (Foster *et al.* 1979; Keller & Schwarzkopf, 1984). Glycerol is a rapidly absorbed gluconeogenic substrate which has been shown to spare muscle glycogen, protect against the development of hypoglycaemia and prolong endurance in rats when given before a run to exhaustion (Terblanche *et al.* 1981). The present study was undertaken to compare the effect of pre-exercise glycerol, glucose and placebo feedings on endurance and metabolism during prolonged submaximal exercise in man.

Six men were studied during exercise to exhaustion on an electrically braked cycle ergometer at a workload requiring 73·2 (SE 1·3)% of maximal oxygen uptake. The ingestion of glucose (1 g/kg body-weight) 45 min before exercise produced a 50% rise in blood glucose concentration and a three-fold rise in plasma insulin concentration at 0 min of exercise. None of the subjects exhibited a fall in blood glucose concentration below 4 mmol/l during the exercise and total carbohydrate oxidation was increased by 26% compared with the placebo trial ($P<0.05$). The ingestion of glycerol (1 g/kg body-weight) 45 min before exercise produced a 340-fold increase in blood glycerol concentration at 0 min of exercise but did not affect resting blood glucose or plasma insulin levels; blood glucose levels were up to 14% higher ($P<0.05$) in the later stages of exercise and at exhaustion compared with the placebo or glucose trials. Both glycerol and glucose feedings lowered the magnitude of the rise in plasma free fatty acid concentration during exercise compared with placebo ($P<0.05$). The mean endurance times were 86·0 (SE 5·4), 108·6 (SE 4·2) and 95·9 (SE 5·1) min for the glycerol, glucose and placebo trials respectively. Five of the subjects exercised for longer on the glucose trial compared with the placebo trial. Exercise time to exhaustion on the glucose trial was significantly longer ($P<0.01$) than on the glycerol trial. No significant difference in performance was found between the glycerol and placebo trials.

These results contrast with previous reports (Foster *et al.* 1979; Keller & Schwarzkopf, 1984) that have indicated glucose feeding pre-exercise produces hypoglycaemia during strenuous submaximal exercise and reduces endurance performance. It appears that man cannot use glycerol as a gluconeogenic substrate rapidly enough to serve as a major energy source during this type of exercise.

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A simple exercise test for field use in studies of nutritional assessment. By SANDRA DRUMMOND and J. V. G. A. DURNIN, *Institute of Physiology, University of Glasgow, Glasgow G12 8QQ*

One of the most important functions of normal man is the ability to undertake physical activity. In developed societies this may no longer be a factor of large significance in most work situations but for many common pursuits in non-working time, the ability to walk or run, or dance or play at various sports without undue stress, is something of considerable significance for individuals of almost all ages. In developing countries, where many forms of work still require physical effort, the importance of being able to undertake physical activity becomes even more marked. A simple functional test of physical exercise capacity (or, in more common physiological nomenclature, physical working capacity) is a useful adjunct to other, more static, measurements of nutritional status, such as anthropometric assessments of muscle mass or fatness. We are employing such an exercise test in studies currently being carried out on rural women in South India.

The test is a step test in three different levels of intensity (Maritz *et al.* 1961). Heart rate and oxygen consumption are simultaneously measured and an extrapolation is made to give the maximal oxygen uptake of the individual ($\dot{V}_{O_2max}$). The $\dot{V}_{O_2max}$ is accepted by most exercise physiologists as an important indicator of cardiovascular fitness and thus of a general ability to do physical exercise.

The standard and recognized technique of estimating $\dot{V}_{O_2max}$ involves measuring O_2 consumption and heart rate at different levels of work intensity by walking and running on a treadmill. In the present investigation, which was carried out in the Physiology Department in Glasgow, forty women, aged 17–34 years, did three different levels of exercise on a treadmill, from which an extrapolation giving $\dot{V}_{O_2max}$ was made. The test was then repeated using a step-test. The $\dot{V}_{O_2max}$ from each type of fitness test were compared and they were very highly correlated (r +0·91). However, a small correction needs to be made by adding 10% (the exact figure is 8·6%) to the step-test value, since the step-test compared with the treadmill values gives a regular and very consistent underestimation of $\dot{V}_{O_2max}$.

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Comparison of weight-for-height and arm circumference in the assessment of acute malnutrition. By ELAINE CARTER (Introduced by R. J. NEALE), *Antsokia Nutrition and Health Project, Shewa Province, Ethiopia, and Leicester Royal Infirmary, Leicester LE1 5WW*

In famine relief situations, weight-for-height (W/H), and mid-upper-arm circumference (AC) are the two commonly used methods of assessing nutritional status of children under 5 years of age. It is usual to consider that values of less than 80% W/H or AC less than 135 mm represent moderate malnutrition, while values less that 70% W/H or AC less than 125 mm represent severe malnutrition (Jelliffe, 1966). However, a recent study (Lindtjorn, 1985) on 115 children showed a significant discrepancy between W/H and AC in the assessment of nutritional status. The following study, conducted on a much larger sample, confirms these findings.

1266 children aged 1–5 years, of villages of Alburko wereda in the province of Wollo, Northern Ethiopia, were measured. Results showed that 436 children (34·4%) had AC of less that 135 mm while only 159 (12·6%) were under 80% W/H. 172 children (13·6%) had AC less that 125 mm, compared with only 20 (1·6%) who were under 70% W/H. The proportions in each category were highly significant ($P<0·001$) in both cases using chi squared test.

The present study shows a discrepancy between the two commonly used methods of identifying moderate and severe malnutrition. It appears that AC exaggerates the degree of malnutrition, and leads to confusion when deciding whether or not a community requires nutritional assistance. It is suggested that cut-off levels for AC and W/H should be adjusted to make them correspond more closely to each other.

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