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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Fortieth Meeting of the Nutrition Society was held at the Royal Society of Medicine, London on Tuesday, 18 *March* 1980, *when the following papers were read:*

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Dietarily obese rats become leaner than controls when palatable food is withdrawn. By D. N. STEPHENS (*Introduced by* J. DOBBING), *Department of Child Health, The Medical School, Manchester M*13 9*PT*

The finding that rats given a highly palatable diet become obese is well established (e.g. Sclafani & Springer, 1976). Less is known of the consequences of withdrawing the palatable diet and its replacement with standard laboratory food.

Male hooded rats were all weaned at 30 d, allocated to three dietary groups, and fed as follows: Group C, commercial laboratory diet (PMD); Group P100, an assortment of palatable foods (e.g. baked beans, breakfast cereals, banana, cat food, etc.) plus PMD for 100 d; Group P30, PMD alone until 100 d and then in addition the palatable diet for a further 30 d. From 130 d the palatable foods were withdrawn from all groups and PMD continued.

Rats were weighed every 10 d and body water was measured by tritium dilution at 130 and 230 d of age. Body fat was estimated from body-weight minus fat-free mass (Rothwell & Stock, 1979) using an empirically determined relationship to calculate fat-free mass from body water.

At the end of the period of feeding the palatable diet, both P groups were heavier ($P<0.001$) and contained more fat ($P<0.001$) than group C, but the increase in fat-free mass failed to reach significance, confirming previous reports.

When the palatable diet was withdrawn, both P groups lost weight for about 10 d, though food intake was significantly depressed relative to group C for only 2 d. Nevertheless, by 230 d, the three groups no longer differed significantly with respect to body-weight, though both P100 and P30 groups were now less fat than group C (see Table). Differences amongst the groups in fat-free mass failed to reach significance.

In spite of reduced fat levels in the rats previously fed the palatable diet, cervical brown adipose tissue (BAT) was significantly increased in group P30 relative to group C (120·0 vs. 76·4 mg, $P<0.001$). Interscapular BAT deposits were unchanged.

These findings suggest that physiological adaptations to dietary obesity may outlast the obesity and result in increased leanness.

(Mean values with their standard errors; no. of rats/treatment in parentheses)

		Weight (g)		Fat-free mass (g)		Fat (g)	
Group		Mean	SE	Mean	SE	Mean	SE
C	(10)	534	14·8	446·0	20·4	88·3	15·0
P100	(11)	541	16·4	490·0	14·6	50·4*	8·5
P30	(10)	531	16·5	493·4	21·5	37·2*	10·2

*Statistical significance of difference from Group C; $P<0.05$.

Rothwell, N. J. & Stock, M. J. (1979). *Br. J. Nutr.* **41**, 625.
Sclafani, A. & Springer, D. (1976). *Physiol. Behav.* **17**, 461.

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Dietary saponin and the hypercholesterolaemic action of milk protein in rabbits. By CHITRA PATHIRANA, M. J. GIBNEY and T. G. TAYLOR, *Department of Nutrition, University of Southampton, Southampton SO9 3TU*

The hypocholesterolaemic effect of soya-bean protein relative to casein has been demonstrated repeatedly in rabbits (Carroll *et al.* 1975). This action has been explained both by the amino acid composition *per se* (Kritchevsky, 1979) and by the saponins normally found in soya-bean preparations (Potter *et al.* 1979). This experiment was designed to test the latter hypothesis.

Twenty-four New Zealand White rabbits (sixteen male, eight female) were fed ad lib. for 2 months on semi-purified diets in which the dietary variables were protein source and saponin supplementation (10 g/kg). Protein supplied 30% of the dietary energy and was either cow's milk protein or isolated soya protein. Fat (coconut–safflower oil, 3:2) supplied 5% and carbohydrate (starch–sucrose–lactose, 7:6:1) supplied the remainder of the energy. All diets were supplemented with methionine, vitamins and minerals.

No significant differences were found between energy intake or the live-weight gain of the rabbits in the four dietary treatments. The serum analyses are shown in the Table.

(Mean values with their standard errors; no. of animals/treatment in parentheses)

	With saponin				Without saponin			
Dietary protein ...	Soya (6)		Milk (6)		Soya (6)		Milk (6)	
Serum cholesterol (mmol/l):	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Total	1·8	0·1 **	5·8	0·8	2·6	0·2 **	6·22	0·5
HDL	0·39	0·03 NS	0·40	0·02	0·38	0·03 NS	0·38	0·03
VLDL+LDL	1·4	0·1 **	5·3	0·6	2·2	0·2 **	5·8	0·5
HDL as % of total	22·3	2·2 **	7·3	0·8	15·3	1·5 **	6·2	0·2

NS, not significant.
**$P<0.01$.

Serum total cholesterol and the VLDL+LDL fraction of serum cholesterol were significantly lowered by soya feeding. Saponin did not significantly influence total serum cholesterol or its sub-fractions with either protein source.

These results show that the hypocholesterolaemic action of soya-bean protein is not dependent upon soya saponins. We suggest that animal proteins are hypercholesterolaemic rather than plant proteins being hypocholesterolaemic.

Carroll, K. K. & Hamilton, R. M. G. (1975). *J. Food Sci.* **40**, 18.
Kritchevsky, D. (1979). *Lancet* i, 610.
Potter, J. D., Topping, D. L. & Oakenfull, D. (1979). *Lancet* i, 223.

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Fat transplantation in mice as a model for the study of concurrent fat loss and fat gain. By MARGARET ASHWELL and C. J. MEADE*, *Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ*

Redistribution of human fat can take place with increasing age and obesity, i.e. a fat loss from one site with a corresponding fat gain at another (Garn & Young, 1956; Ashwell *et al.* 1978). We describe here an animal model for the study of concurrent fat loss and fat gain using the previously described technique of transplanting small pieces of adipose tissue under the kidney capsule of mice (Ashwell *et al.* 1977).

Female CBA mice (aged 6–7 weeks) were injected intraperitoneally with 0·1 ml gold-thioglucose (GTG) (80 mg/ml). Six weeks later, forty 'GTG-obese' mice received transplants of 'GTG-obese' fat under the right kidney capsule and 'lean' fat from non-injected CBA mice under the left kidney capsule. Dietary restriction was begun immediately and mice were killed three weeks later. Fat cell size was determined in the gonadal fat from the four pairs of donor mice, in host mice gonadal fat before and after dietary restriction and in the fat grafts (Ashwell *et al.* 1976).

Dietary restriction caused an average decrease in body-weight of 36% in the twenty-four surviving host mice and their average fat cell weight (±SE), originally 0·51 (±0·11) μg, decreased in size to 0·27 (±0·16) μg. This cell size was intermediate between that of the original 'lean' and 'GTG-obese' donors (0·13 (±0·03) μg and 0·52 (±0·19) μg respectively). The fat cells in 'lean' grafts showed an over-all increase in weight of 0·12 (±0·17) μg during the 3 weeks of transplantation into dietary restricted hosts whereas the fat cells in 'GTG-obese' grafts showed an over-all decrease in weight of 0·35 (±0·08) μg during this period. The changes in fat cell size in the two types of grafts were significantly different ($P<0·001$). In sixteen mice, there was a fat loss from the 'GTG-obese' graft at the same time as a fat gain in the corresponding 'lean' graft. The net effect was to produce an equalization of fat cell size in the transplanted mice such that there was no significant difference in the fat cell sizes of the host mice and the fat cell sizes in both types of graft ($P<0·001$).

The fat transplantation model has clearly demonstrated that fat loss and fat gain can take place concurrently at different sites in the same animal.

Ashwell, M., Chinn, S., Stalley, S. & Garrow, J. S. (1978). *Int. J. Obesity* **2**, 289.
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Garn, S. M. & Young, R. W. (1956). *Am. J. phys. Anthrop.* **14**, 497.

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Differences in the metabolic rate following ingestion of sucrose and glucose in man. By N. SHARIEF and I. MACDONALD, *Department of Physiology, Guy's Hospital Medical School, London SE1 9RT*

There is evidence from experiments in rats (Allen & Leahy, 1966; Macdonald & Grenby, 1979), baboons (Allen *et al.* 1966; Brook & Noel, 1969) and man (Macdonald & Taylor, 1973) that not all dietary carbohydrates have the same effect on body-weight when given in isoenergetic quantities. As these were not acute experiments, it was decided to study, in man, the immediate effects on the metabolic rate of glucose and sucrose, since previous findings in rats (Sharief & Macdonald, 1979) had shown that the metabolic rate after the ingestion of sucrose was significantly greater than that after glucose, fructose or a mixture of the two.

In this investigation the O_2 uptake (VO_2), CO_2 production (VCO_2), respiratory quotient (RQ), and metabolic rate (MR) after the ingestion of glucose, sucrose or water were measured in man.

Six normal-weight subjects were studied. Their mean body-weight (±SEM) was 69·4 (±4·5) kg, 0·9% below ideal body-weight (IBW; Metropolitan Life Insurance Company, 1960). Each subject was studied three times with glucose, sucrose or water given in random order after an overnight fast. The carbohydrate was given at a dose of 5 g/kg IBW, made up in a slurry with 4 ml distilled water/kg IBW. Resting VO_2, VCO_2, RQ and MR were measured before and continuously for 3 h after ingestion, using the ventilated hood technique. For purposes of calculation the four measurements were averaged at 15 min intervals. The MR was calculated from the VO_2 according to the RQ. The first 15 min after the test meal were ignored.

The results show that VO_2, VCO_2, RQ and MR are significantly increased after sucrose than after an equal weight of glucose, for the whole period of 3 h after the test meal.

These findings confirm earlier results in rats that sucrose, in the short term raises the metabolic rate to a greater extent than glucose when equal quantities are ingested.

Allen, R. J. L., Brook, M., Lister, R. E., Sim, A. K. & Warwick, M. H. (1966). *Nature, Lond.* **211**, 1104.
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Nutritional status and dietary habits of Surinam immigrants in the Netherlands: iron and B vitamins. By R. J. EGGER, U. H. RENQVIST, J. H. VAN EE, H. V. D. BERG, A. DE GEUS and R. LUYKEN, *Royal Tropical Institute/Central Institute for Nutrition and Food Research TNO, Amsterdam/Zeist, The Netherlands*

A survey of nutritional status and dietary habits among immigrants was carried out from January to June 1978. In this study 155 Surinam immigrants (from African and East-Indian origin) and 183 Dutch (Caucasian controls) 8–9 year old schoolchildren, representing 85% of the selected population of an Amsterdam suburb, participated. Results presented in the Table will be discussed.

Iron and B-vitamins: major results of biochemical analyses in blood/serum and dietary intake (24 hour recall)

	East-Indians				Caucasians				Creoles			
	Mean	SD	(*n*)	% Abnormal†	Mean	SD	(*n*)	% Abnormal†	Mean	SD	(*n*)	% Abnormal†
Blood/serum:												
Hb (mmol/l)	8·4	0·6	(51)	4*	8·6	0·5	(108)	1	8·0	0·8	(55)	20*
Ht (%)	40·0	2·4	(51)	4	40·5	2·2	(108)	1	38·9	3·0	(55)	9*
Fe sat. (%)	20·7	7·2	(36)	22*	29·2	10·2	(89)	7	23·7	11·6	(41)	20*
				% Abnormal‡								% Abnormal‡
ETK (ratio)	1·12	0·09	(51)	14*	1·10	0·07	(103)		1·16	0·2	(54)	20*
EGR (ratio)	1·32	0·3	(51)	41*	1·11	0·1	(102)		1·26	0·2	(54)	41*
Serum folate (nmol/l)	8·2	4·0	(47)	4	8·1	2·5	(71)		7·2	2·2	(48)	6*
B_{12} (pmol/l)	605	198	(44)	5	558	185	(76)		583	160	(45)	2
Dietary intake:				%RDA‖				%RDA‖				%RDA‖
Iron (mg)	7·11	2·61	(52)	71*	9·22	3·86	(181)	92	8·10	2·83	(68)	81*
Thiamine (mg)	0·64	0·34	(52)	71*	0·82	0·31	(181)	91	0·78	0·33	(68)	87
Riboflavin (mg)	0·92	0·60	(52)	74*	1·35	0·63	(181)	108	1·12	0·61	(68)	90*

Statistical significance of differences from caucasian controls *($P<0·05$).

†Percentage below or beyond cut-off points: haemoglobin (Hb)<7·5 mmol/l; haematocrit (Ht)<36%; iron saturation (Fe sat)<15%. (WHO, 1972).

‡Percentage below or beyond 5th resp. 95th percentile caucasian controls: erythrocyte transketolase activation ratio (ETK)>1·20; erythrocyte glutathione reductase activation ratio (EGR)>1·29; serum folate<3·9 nmol/l; serum vitamin B_{12}<335 pmol/l.

‖Percentage of the Dutch Recommended Dietary Allowances: iron, 10 mg/d; thiamine, 0·9 mg/d; riboflavin, 1·3 (boys) resp. 1·2 (girls) mg/d.

Haemoglobin S and thallassaemia minor were absent; glucose-6-phosphate dehydrogenase deficiency was observed in eight immigrant children. However, none of these children had abnormal values for iron or B-vitamins (*n* 54).

World Health Organization (1972). *Tech. Rep. Ser. Wld Hlth Org.* no. 503.

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The pig as a model for studies on the mode of action of guar gum in normal and diabetic man. By A. R. LEEDS* and S. S. KANG, *Department of Nutrition, Queen Elizabeth College, London W8 7AH* and A. G. LOW and I. E. SAMBROOK, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Guar gum, a viscous unabsorbable polysaccharide, reduces postprandial hyperglycaemia and insulinaemia in normal and diabetic man. Studies on the mode of action of guar gum in man have limitations so we have investigated the suitability of the pig as a model for such studies.

Five Large White boars of approximately 30 kg liveweight were fitted with permanent catheters in the anterior vena cava. The pigs received either a practical-type diet based on barley, weatings and soya-bean meal (A) or a semi-purified diet based on maize starch, maize oil, cellulose and casein (B). The pigs were adapted to the diets for 7 d and then blood samples were taken twice, immediately before feeding at 09.00 hours, and then every hour until 16.00 hours. The pigs received 725 g diet at 09.00 hours, and at 16.00 hours. 2 d later the pigs received the same amounts of diet together with 51 g guar gum and blood samples were taken at the same times as before. Blood samples were analysed for glucose by a glucose-oxidase method and insulin by radioimmunoassay.

All five animals had smaller postprandial blood glucose and insulin peaks after the meals containing guar gum, than after the meals without the gum. In animals receiving diet A, at 1 hour the mean (±SEM) blood glucose concentration was 14·0 (±0·72) mM/l (without guar) compared with 11·5 (±0·99) mM/l (with guar) and mean insulin concentration was 123 (±48·5) mU/l (without guar) and 29 (±11·0) μU/ml (with guar) (n 3). Animals receiving diet B showed similar, but more pronounced, differences between guar containing and non-guar containing meals. Insulin:glucose values were reduced after the guar containing meals.

We conclude that pigs respond to guar gum in a similar way to that observed in humans (Jenkins *et al.* 1976) and that the pig appears to be a suitable model for studying the mode of action of guar gum.

Mr. K. W. Titchell of the Hercules Powder Company Limited kindly supplied the guar gum. The authors are also grateful to Mr N. R. Bolster for technical assistance and Professor A. E. Bender and Dr R. Braude for initiating this collaborative work.

Jenkins, D. J. A., Leeds, A. R., Gassull, M. A., Wolever, T. M. S., Goff, D. V., Alberti, K. G. M. M. & Hockaday, T. D. R. (1976). *Lancet.* ii, 172.

*Heinz Research Fellow.

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Thermogenesis induced by cafeteria feeding in young growing rats. By NANCY J. ROTHWELL and M. J. STOCK, *Department of Physiology, St. George's Hospital Medical School, London SW17 0RE*

We have previously established the cafeteria feeding system as a suitable method of inducing reversible obesity in the adult rat (Rothwell & Stock, 1978). When the animals are presented with this varied and highly palatable diet they overeat by up to 80% compared to stock fed controls and deposit excessive amounts of fat. However, recent studies have demonstrated that the weight gains associated with cafeteria feeding are variable and in some rats hyperphagia is often accompanied by a marked increase in dietary-induced thermogenesis (DIT), (Rothwell & Stock, 1979). The present study was undertaken to determine if young growing rats would become obese when overfed or resist excessive weight gain by increasing DIT.

Twelve male, Sprague-Dawley rats (age, 33 d) were divided into two groups, each with a mean body-weight of 104 g and maintained on either a conventional stock diet (PRD, Christopher Hill Group Ltd.) or the cafeteria diet for a period of 14 d. Body-weight and food intake were recorded daily and resting oxygen consumption ($\dot{V}O_2$) was measured on Day 10 before and after injection of noradrenaline (NA, 25 μg/100 g body-weight, s.c.).

Cafeteria rats consumed 50% more energy (3630±140 kJ) than stock fed controls (2430±40 kJ), $P<0.001$) but gained slightly less weight than controls, although not significantly so (g gained: control 79±2; cafeteria 74±5, not statistically significant). Thus, the efficiency of weight gain (g gain/MJ eaten) was reduced by 39% in experimental animals (control 33·1±0·9; cafeteria 20·2±0·9, $P<0.001$) and resting $\dot{V}O_2$ (ml/min per $W^{0.75}$) was increased by 36% (control 11·43±0·30; cafeteria 15·56±0·37, $P<0.001$). These differences in energy expenditure were accompanied by an enhanced sensitivity to the thermogenic effect of NA ($\dot{V}O_2$ after NA: control 15·62±1·20; cafeteria 24·34±1·21, $P<0.001$) and is similar to the changes seen in older animals.

The hyperphagia of young rats fed the cafeteria diet is less marked than that seen in adult animals but it seems that the capacity for DIT is as great, if not greater. The cafeteria rats in this study were able to maintain almost identical body-weight gains to stock fed controls in spite of their excessive energy intake. These results indicate that DIT can play a major role in energy balance regulation in young growing rats as well as adults and that, like the adult rat, this DIT involves changes in metabolism mediated by the sympathetic nervous system.

Rothwell, N. J. & Stock, M. J. (1978). *J. Physiol., Lond.* **276,** 60.
Rothwell, N. J. & Stock, M. J. (1979). *Nature, Lond.* **281,** 31.

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Nutritional differences between stock diets for the breeding and maintenance of rats. By A. WISE, *Medical Research Council Laboratory Animals Centre, Carshalton, Surrey*

Both Clarke *et al.* (1977) and the National Research Council (1978) assume that the major difference between the nutrient requirements for maintenance and breeding diets is in protein:energy value. Because so little is known about maintenance, they consider that their recommendations for other nutrients, adequate for breeding, may also be applied to non-breeding adults. However, in a survey by the MRC Laboratory Animals Centre that included twenty-four stock diets produced by five British manufacturers there appear to be significant correlations between levels of several nutrients and the protein in the diet. The latter has been taken by the author as a measure of the diet manufacturer's intention about whether it is a breeding, or maintenance diet. The extent of such differences has been shown by grouping the diets as those above and below an arbitary cut-off point at 180 g protein/kg.

	r	*P*	Maintenance (*n* 9)	Breeding (*n* 15)
Protein (g/kg)	1	0	161	207
Calcium (g/kg)	0·58	<0·01	7·7	10·4
Copper (mg/kg)	0·50	<0·05	13·7	17·0
Iron (mg/kg)	0·44	<0·05	104	130
Phosphorus (g/kg)	0·64	<0·001	6·3	7·7
Potassium (g/kg)	0·47	<0·05	6·9	8·7
Thiamin (mg/kg)	0·46	<0·05	10·8	16·1
Retinol (mg/kg)	0·63	<0·01	2·9	5·1
Pyridoxine (mg/kg)	0·51	<0·05	5·2	10·5
Vitamin D (μg/kg)	0·45	<0·05	30	60
α-Tocopherol (mg/kg)	0·46	<0·05	55	76

No significant correlations are found for other nutrients; chlorine, magnesium, sodium, zinc, choline, folate, nicotinic acid, pantothenate, riboflavin, cyanocobalamin, and vitamin K. However, it should be noted that the levels of these nutrients are very variable, ranging severalfold in some cases. The figures are calculated for preprocessed diet, but the relative differences will remain after processing.

The possibilities of diet-induced variation in experimental results should be borne in mind by users of stock diets for research purposes. There is no standard diet, and it should not be assumed that diet manufacturers are following the recommendations based on our present knowledge of requirements.

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National Research Council (1978). *Nutrient requirements of laboratory animals.* Washington, DC: National Academy of Sciences.

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Dietary patterns among overseas students in London. By C. JEYAKUMAR HENRY and ERICA F. WHEELER, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

A survey was conducted among overseas students to study their food habits, any changes in their dietary patterns on coming to Britain, and the extent to which they accepted English food. Sixty-three male and thirty-seven female students residing in a postgraduate hall of residence in London were interviewed by discussion based on a set of questions.

There was a relationship between acceptance of English food by the students and the duration of time spent in Britain (see Table). 'Acceptance' is defined as their being willing in general to eat English food, enjoying it sometimes, and having developed distinct preferences for some English foods over others.

	Time spent in Britain (years)		
	<1–1	1–3	3–9
Students who accepted English food (%)	9	18	78
No. in group: Male	23	22	18
Female	22	10	5

Non-accepters of English food said that it was 'too bland (meaning that spices were not properly used)', 57%; 'too oily', 11%; 'mushy food', 11%.

Outstanding comments by accepters were; 'well balanced and nutritious food', 44%; 'clean and well presented', 30%; 'bland food, which does not upset the stomach', 15%. 63% of the subjects felt that English food was more nutritious and health giving than their national foods. This concept of 'health giving foods' incorporated the ideas that consumption of meat products and milk were conducive to health, and that 'cleanliness', (hygienic preparation and presentation of food) is beneficial. These ideas derive from the Western education which all postgraduate students are likely to have received in school.

All the students reported an increased consumption of potato and wheat products. Even among the newcomers there was increased consumption of milk, confectionery, breakfast cereals, fruit juices and cheese. These findings are in keeping with an American study (Lewis & Glaspy, 1975). Favourite English foods were beefsteak, lamb chops, roast beef; disliked foods were predominately mashed potatoes and boiled vegetables. Only 8% said that they ate a wholly national diet, 56% said they ate a mixture of English and national food, and 36% essentially ate only English food. Interestingly, 80% of the students enjoyed eating raw salads.

Despite foreign students' initially conservative attitudes towards English food, the survey shows that their food habits and preferences change, and that the changes are related to their perception of the preparation, 'health giving' attributes and texture of food. Among the students there were as many who preferred the 'blandness' of English food, as those who regretted the absence of spices.

Lewis, J. S. & Glaspy, M. F. (1975). *J. Am. diet. Ass.* **67**, 122.

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Effect of 3-hydroxybutyrate in obese subjects on very low energy diets.

By G. L. S. PAWAN and S. J. G. SEMPLE, *Department of Medicine, The Middlesex Hospital, London W1N 8AA*

In treatment of obesity, diets of 4·2 MJ (1000 kcal) will generally reduce weight slowly with little net loss of body protein (Kekwick & Pawan, 1956). However, many obese patients admitted to hospital for urgent and rapid weight reduction are treated by starvation or semi-starvation therapy. This can have some undesirable side effects, e.g. net body protein loss, a high lean:fat value for tissue loss (Benoit *et al.* 1965; Ball *et al.* 1967*a*; Ball *et al.* 1967*b*), impairment of immunocompetence and increased susceptibility to infection. This negative nitrogen balance was formerly assumed to be caused by gluconeogenesis, mainly from alanine of muscle tissue, to provide glucose essential for brain metabolism. However, ketones can replace glucose as fuel in brain tissue (Owen *et al.* 1967; Garber *et al.* 1974) and infusion of ketones during prolonged starvation can reduce body N loss (Sherwin *et al.* 1975).

We investigated the effects of administering sodium-DL-3-hydroxybutyrate to eight obese women, more than 150% ideal body-weight, who received on alternate days a 2·5 MJ (600 kcal) diet containing 34 g protein, with a total fast every other day for 21 d. The experimental plan was as follows: 5 control d, then 3 d intravenous 3-hydroxybutyrate (18 g/d), followed by a control 5 d period, then 3 d of intravenous isoenergetic glucose (18 g/d), and a final 5 d control period. In eight other obese women, the same procedure was followed, but the hydroxybutyrate was administered orally. In four of these subjects the order of hydroxybutyrate and glucose administration was reversed. Both intravenous and oral hydroxybutyrate produced a significant reduction in net body protein loss as measured by N balance. The hydroxybutyrate did not significantly affect the rate of weight loss, but increased the fat:lean value for tissue loss. Our subjects experienced no untoward effects and none complained of hunger.

This investigation was approved by The Middlesex Hospital Ethical Committee.

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Assay of vitamin K_1 (phylloquinone) by high performance liquid chromatography: values for human and cow's milk. By Y. HAROON, M. J. SHEARER, G. MCENERY*, V. E. ALLAN and P. BARKHAN (*Introduced by* I. MACDONALD), *Haematology Department, Guy's Hospital and Paediatric Department*, Whipps Cross Hospital, London*

The generally low concentrations of the physiologically essential K vitamins present in foods has made its assay difficult and thus information on man's dietary supply of vitamin K is both meagre and imprecise. Dietary vitamin K may be an important factor in the hypoprothrombinaemia of the newborn; those fed solely on human milk are more likely to develop severe hypoprothrombinaemia and bleeding (Sutherland *et al.* 1967). We have developed an assay for K vitamins based on high performance liquid chromatography (HPLC) (Shearer *et al.* 1979). Using this assay we have measured the content of vitamin K_1 (the major nutritional form of vitamin K) in human and cow's milk and in commercial infant formula foods.

Fresh colostrum from five women gave a mean concentration of vitamin K_1 of 3·2 μg/l (range 1·9–4·2). This was not significantly different from the concentration in established milk from fifteen women which gave a mean value of 2·2 μg/l (range 1·1–5·6).

Samples of dairy milk were analysed. The concentration of vitamin K_1 in 'Friesian' milk was significantly higher ($P<0.001$) than human milk with a mean value of 5·6 μg/l (range 4·0–8·9; n 10). 'Channel Island' milk gave a mean value of 11·7 μg/l (range 7·0–17·8; n 4).

A commercial infant formula food which was based on cow's milk but not supplemented with vitamin K_1 contained 3·8 μg/l, whilst a similar but supplemented formula contained 33·2 μg/l.

K_2 vitamins (menaquinones) were not detected suggesting that the major form of vitamin K in milk is vitamin K_1 derived from plant sources.

As far as we are aware this is the first time that vitamin K has been assayed in both human and cow's milk using a physico-chemical method, the only previous comparison having been made using a curative chick bioassay (Dam *et al.* 1942). The range of values reported using the chick bioassay was very much greater than we found by HPLC assay although both methods suggest that the vitamin K_1 content of human milk is substantially lower than cow's milk. The advantages of our HPLC assay over the bioassay included its specificity for different molecular forms of K vitamins and a greater sensitivity and precision. The HPLC assay has also been applied to the measurement of vitamin K_1 in other food stuffs (M. J. Shearer, unpublished results).

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The effect of streptozotocin diabetes on liver protein metabolism. By E. C. ALBERTSE, P. J. GARLICK, and V. M. PAIN, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

Previous studies using constant infusion of [^{14}C]tyrosine have failed to detect an effect of diabetes on liver protein synthesis (Pain & Garlick, 1974). McNurlan & Garlick (1979) have now demonstrated that diabetes does in fact reduce total (cellular and secreted) protein synthesis by the liver. They used a more appropriate method of measuring incorporation of label 10 min after injecting a single large dose of [^{14}C]leucine. During the 10 min incorporation period, total liver proteins, i.e. secretory and cellular, can be measured. This contrasts to the constant infusion method which determines only non-secretory liver proteins.

In the present study rates of protein synthesis and breakdown in liver were measured in diabetic (insulin withdrawn) and control (insulin treated) rats by the method described previously (Albertse *et al.* 1980). Albumin synthesis was estimated as a proportion of total liver protein synthesis by specific immunoprecipitation of albumin from liver homogenates (Pain *et al.* 1978).

The results in the Table show a decrease in the over-all rate of protein synthesis in the liver of diabetic rats, in confirmation of the results of McNurlan & Garlick (1979). Albumin synthesis showed a disproportionate fall, but this was insufficient to account on its own for the over-all effect. In previous experiments we have found that the proportion of total liver protein synthesis devoted to the production of plasma proteins other than albumin (about 20%), is unaffected by diabetes (V. M. Pain & P. J. Garlick, unpublished results). From the rates of synthesis of total protein, albumin and other secreted proteins we have calculated the rate of synthesis of liver cellular proteins. Degradation was then calculated as the difference between the rates of synthesis and growth of cellular protein. As shown in the Table, protein breakdown rates were elevated after 1 d of insulin withdrawal. By 4 d, however, breakdown rates were reduced to levels below that of controls.

Protein synthesis and breakdown in liver of diabetic and insulin treated rats

(Mean values with their standard errors)

	Control		Diabetic		
	Day 0	Day 3	Day 1	Day 2	Day 4
Synthesis total protein (%/d)	96·4±2·7	82·5±2·7	75·7±0·8	71·5±2·2	65·3±3·2
Synthesis cellular protein (%/d)	59·7	49·5	46·7	47·2	42·8
Albumin (% of total synthesis)	18·1±0·6	20·0±0·6	18·3±1·0	14·0±0·6	14·5±1·5
Breakdown (%/d)	58·5	51·2	67·0	57·0	39·7

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Obesity and high energy diets reduce survival and growth rates of rat pups. By BARBARA J. ROLLS[1], EDWARD A. ROWE[1], SUSAN E. FAHRBACH[1], LORANNE AGIUS[2] and DERMOT H. WILLIAMSON[2] and *Department of Experimental Psychology, University of Oxford,*[1] *Metabolic Research Laboratory, Nuffield Department of Clinical Medicine, Radcliffe Infirmary*[2], *Oxford*

Obesity can be induced in rats by the consumption of palatable foods (Rolls & Rowe, 1977). This method has provided an animal model to examine the course of pregnancy and lactation in obesity. Female hooded Lister rats were given high energy palatable foods and laboratory chow (obese group) or just chow (control group) from 10 weeks of age to the end of the experiment (see Rolls *et al.* 1980 for diet composition). The rats were mated at 22 to 29 weeks of age, when the obese group weighed 104 g more than the control group. A third group of normal weight rats received only chow until parturition and were then also given the high energy foods during lactation (diet-supplemented group).

The obese rats did not develop the hyperphagia normally seen during lactation. In the control rats food intake increased by 61% over the first 6 d of lactation compared with premating levels; the increase was only 12·5% in the obese rats. The obese rats ingested significantly less energy than the control rats during lactation ($P<0·01$), whereas the diet-supplemented rats ingested significantly more than the control rats ($P<0·01$). Changes in maternal body-weight were consistent with differences in food intakes: the diet-supplemented mothers gained more weight than the control mothers during early lactation; the obese mothers lost weight during this period, and at weaning 21 d after parturition the obese mothers were not significantly heavier than the control mothers.

After parturition litter size was standardized at eight pups. In the obese group 44% of the litters died within 6 d of parturition, while all the litters in the other groups survived. The surviving pups of the obese mothers ($P<0·001$), and to a lesser extent the pups of the diet-supplemented mothers ($P<0·001$) had poor weight gains compared to the control pups. The poor performance of the pups in the groups with the high energy foods may relate to the depression of maternal mammary gland lipogenesis 6–10 d after parturition which was more severe ($P<0·005$) in the obese group, and to the high lipid and low protein intakes of the rats given the high energy diet relative to the control group. The more severe effects in the obese group may relate to the elevated body-weight and its metabolic and endocrine consequences which may be responsible for the failure to become hyperphagic.

Supported by the Medical Research Council and the United States Public Health Service.

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Inhibitory effect of guar gum on the intestinal absorption of glucose in vitro. By I. T. JOHNSON and JENNIFER M. GEE, *ARC Food Research Institute, Colney Lane, Norwich NR4 7UA*

It is known that in man, the addition of the gel-forming polysaccharide guar gum to test meals leads to a decrease in post-prandial glycaemia and urinary glucose loss and this has been shown to be of therapeutic value in the treatment of diabetics (Jenkins *et al.* 1977). Though guar gum is thought to act by slowing the intestinal absorption of glucose, it is not clear whether this results from a reduction in gastric emptying rates, or from an inhibition of transport at the surface of the small intestine (Holt *et al.* 1979). In an attempt to resolve this problem we have studied the influence of guar gum on glucose transport in vitro, using an isolated preparation of rat intestine.

Everted sacs were prepared from excized rat jejunum and alloted at random to a control incubation medium containing 28 mM-glucose and three treatment media containing 0·1%, 0·25% and 0·5% guar gum. The apparent viscosities of the incubation media were determined using a rotary viscometer. After a 30 min incubation, the glucose transported into the serosal solution of each sac was determined by colorimetry and expressed in terms of tissue dry weight.

(Mean values with their standard errors for seven animals)

Guar gum concentration (%)	Viscosity (centipoises)	Glucose transport (umol/gm per 30 min)
0	1	30·9±10·4
0·1	3	21·2± 6·7
0·25	16	10·3± 5·2
0·5	104	8·0± 2·4

$P<0·01$ (by analysis of variance).

These results demonstrate a significant reduction in glucose absorption with increasing concentrations of guar gum. In separate experiments a similar effect was obtained using high viscosity carboxymethyl cellulose in place of guar gum. In both cases there is a rapid decline in absorption as the apparent viscosity of the medium is increased from 1 to 10 centipoises.

We conclude that both guar gum and carboxymethyl cellulose inhibit glucose transport in this in vitro system. This effect is probably due to an increase in the thickness of the unstirred solvent layer as the mucosal surface brought about by the higher viscosity of media containing gel-forming polysaccharide gums.

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Clearance and excretion of N^{τ}-methyl histidine by lactating dairy cows.

By C. I. HARRIS, G. MILNE and J. D. OLDHAM,* *Rowett Research Institute, Bucksburn, Aberdeen AB1 9SB and *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Previous work has shown that N^{τ}-methyl histidine (N^{τ}-MH) was cleared rapidly into urine in non-lactating cattle (Harris & Milne, 1979), thus validating the excretion of N^{τ}-MH as a measure of muscle protein breakdown in these animals. This report extends the observations to lactating cows.

Four mature Friesian cows, aged 4·5–5·6 years, in late lactation, were given 12 kg concentrates (barley–groundnut meal–mineral, vitamin supplement, 880:100:20) plus 4 kg hay per d. N^{τ}-[$^{14}CH_3$]methyl histidine (approximately 100 μCi) was injected subcutaneously and urine was collected, via bladder catheter, for the next 10 d. Milk samples were taken at each milking.

Cow	Live weight (kg)	Milk yield (kg/d)	Recovery of radioactivity (%) in 10 d in: urine	milk	total	Mean daily urinary N^{τ}-MH excretion (m mol; *n* 7)
1	476	17·8	98·6	1·6	100·2	1·05
2	553	14·9	93·8	1·9	95·7	1·07
3	627	17·8	92·2	2·3	94·5	1·61
4	649	4·9	99·1	0·2	99·3	1·59

Recovery of injected radioactivity was quantitative with no more than 2·3% recovered in milk. Of the injected dose 78·6% of the radioactivity was associated with N^{τ}-MH and the impurities cleared into urine more rapidly than N^{τ}-MH so that after 46 h at least 88% of radioactivity was associated with N^{τ}-MH. Total radioactivity was rapidly eliminated in urine with mean recoveries of 80·4% (range 69·0–90·3) and 93·2% (range 87·1–97·2) after 2 and 5 d respectively.

The mean rate of N^{τ}-MH excretion (2·29 μmol/kg per d) was lower than found in non-lactating cattle (Harris & Milne, 1980). Assuming 8% of body-weight was muscle protein containing 3·5 mmol N^{τ}-MH/kg muscle protein (Nishizawa *et al.* 1979), the rate of N^{τ}-MH excretion was equivalent to 0·65 kg muscle protein catabolized/d, or a fractional breakdown rate of 0·008/d.

We conclude that N^{τ}-MH excretion is a valid index for measuring muscle protein breakdown rates in lactating cows and that it is sufficient to measure excretion of N^{τ}-MH only in urine.

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High concentrations of copper and zinc in sheep nutrition. By W. H. PARRY and FARIS AL-MUKHTAR, *Department of Science, Bristol Polytechnic, Coldharbour Lane, Bristol BS16 1QY*

Several workers have previously investigated whether dietary intakes of zinc have any influence on copper metabolism in sheep. Bremner *et al.* (1976) reported the effect of supplementing the intake of 12 week old lambs with up to 420 mg Zn/kg diet on plasma and liver Cu and Zn concentrations. More recently Campbell & Mills (1979) have reported the effect of supplementing the feed intake of pregnant ewes with 750 mg Zn/kg diet. In the main, these results showed that Cu metabolism in ewes and lambs was affected by increasing dietary Zn concentration.

Our present results are from experiments conducted with 5–6 month old wether lambs fed 700 mg Zn/kg diet and increasing concentrations of Cu at regular intervals up to 200 mg Cu/kg diet over 7 weeks. The dietary intake of 200 mg Cu/kg diet and 700 mg Zn/kg diet was maintained for 3 weeks before decreasing the dietary Zn concentration from 700 mg Zn/kg at weekly intervals to 40 mg Zn/kg diet over a 5 week period. Four control sheep were fed 6 mg Cu/kg diet throughout the experiment whilst their dietary Zn level varied as already described. Live weight and haematological measurements were made at weekly intervals during the 15 d experimental period. Blood samples were taken each week and liver biopsy samples were obtained from all sheep on three occasions during the experimental period.

Plasma Zn concentrations rose to levels of 6–8 μg/ml in the sheep given 700 mg Zn/kg of diet; the corresponding Cu concentrations remained reasonably constant at 1–2 μg/ml. There was some change in the haematological measurements over the weeks and some changes were noticeable in Zn and Cu concentrations in the liver. The most interesting result was in the binding of Zn and Cu to plasma proteins. The evidence suggested that the proportion of Zn and Cu bound to these proteins could be influenced by the dietary levels of these metals fed to the sheep during the experimental period. There were no deaths and no definite symptoms of Cu toxicosis.

These results will be presented and discussed in relation to possible high dietary intake of Cu in sheep grazing land which has been treated with pig slurry.

The authors are grateful to Dr Brian Cooke and Dr Brian Hardy, Dalgety Crosfields Ltd. for advice during the experiment and help in formulating and supplying the diet.

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The effects of exercise and training on the utilization of dietary glucose.

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Depletion of the endogenous carbohydrate stores has been shown to be a limiting factor in the ability to endure long-term exercise (Bergström *et al.* 1967). While considerable attention has been given to the role of blood glucose during exercise, very limited information (Costell *et al.* 1973) is available to describe the contribution of oral (exogenous) glucose to carbohydrate metabolism, or its possible modification by exercise training.

This experiment was designed to compare the dynamics of dietary glucose utilization at two different levels of energy expenditure in trained and habitually sedentary rats.

Twenty-four adult male Wistar rats were assigned to three treatment groups: a sedentary group, an exercised trained group and a restricted intake group. The dietary intake of the latter group was restricted to allow a similar rate of weight gain to the exercise-trained animals which tended to be somewhat lower than that of the freely eating sedentary controls. Following a ten-week period of training by daily treadmill running, measurements of metabolic rate and [^{14}C]carbon dioxide production were made on the rats immediately after gastric intubation of a [^{14}C]glucose meal. 5·0 ml of a 30% glucose solution containing 10 μCi was given to each rat. The measurements were made in a closed-circuit respiration calorimeter either at rest or including a 45 min bout of treadmill exercise (30 min after intubation).

Trained animals exhibited a significantly higher ($P<0·01$) resting metabolic rate than either of the sedentary groups (15·8±0·3; 14·9±0·1 and 14·4±0·3 kJ/$^{0·75}$ per h for trained, sedentary and restricted animals respectively).

Both the peak rise and culmulative oxidation of dietary glucose over a 6 h period were significantly greater ($P<0·01$) in trained rats, both at rest and during treadmill exercise. Exercise significantly ($P<0·01$) increased the rate of oxidation of dietary glucose, which contributed approximately 50% of the energy requirement for exercise in trained animals.

Clearly exercise training induces metabolic changes which significantly increase the rate of oxidation of dietary glucose.

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Effect of fat and sugar at breakfast on blood glucose rises after lunch. By R. H. TAYLOR[1], D. J. A. JENKINS[2], T. M. S. WOLEVER[3], C. J. GRIFFITHS[3], KATHLEEN KRZEMINSKA[3], J. A. LAWRIE[3] and CAROLYN BENNETT[3], *Department of Gastroenterology, Central Middlesex Hospital, London NW10*[1], *Department of the Regius Professor of Medicine, Radcliffe Infirmary, Oxford*[2] and *University Laboratory of Physiology, Oxford*[3]

In a previous communication we showed that considerable differences exist between different classes of foods in the extent to which equal carbohydrate portions raise the post-prandial blood glucose (Jenkins *et al.* 1980). We suggested that those foods which raise the blood glucose least may be of most benefit to diabetics. However, as many of these foods also contain appreciable quantities of fat, before making any such recommendation it appeared important to assess the effect of fat feeding on the subsequent meal.

Three healthy subjects took four breakfasts of different composition on separate days in random order. The breakfasts (see Table) were planned to test the effect of fat and sugar on the glycaemic response to a standard lunch (protein 32 g, fat 7 g, sugar 35 g and starch 65 g; total energy 2·3 MJ (565 kcal) eaten 4 h later.

The finger-prick blood glucose response to breakfast showed the highest rises with high sugar and high starch meals. However, the reverse effect was seen after lunch, with the higher rises in blood glucose occurring after lunches which followed high fat breakfasts.

Breakfast	Protein (%)	Fat (%)	Sugar (%)	Starch (%)	Energy (MJ (kcal))	Lunch blood glucose rise (mmol/l) mean peak	mean 15–60 min
Low fat, low sugar	18	12	12	58	2·52 (630)	3·1±0·2	1·2±0·2
Low fat, high sugar	10	5	56	29	2·67 (668)	2·5±0·8	1·6±0·6
High fat, low sugar	14	59	8	19	2·67 (669)	4·0±0·4	2·8±0·3
High fat, high sugar	8	37	42	13	2·65 (661)	3·5±0·4	2·4±0·4

There was no significant difference between the effects of starch or sugar on the subsequent lunch response. Combining the high fat results, both the peak glucose rise and the mean 15–60 min glucose rise were significantly higher after lunch ($P<0·02$).

We conclude that fat consumption may impair the glycaemic response to the subsequent meal and that foods with a low glycaemic index and high fat content may not improve over-all diabetic blood glucose control.

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The relationship of dietary induced thermogenesis to metabolic efficiency in man. By D. A. YORK, J. B. MORGAN and T. G. TAYLOR, *Department of Nutrition, School of Biochemical and Physiological Sciences, Southampton University, Southampton*

Dietary induced thermogenesis (DIT) has been suggested as one possible mechanism to explain the differences in metabolic efficiency that have been reported in man and animals (Rothwell & Stock, 1979). We have studied DIT in two groups of male volunteers of stable body-weight who habitually consume either very large or very small daily energy intakes as assessed by either a 7 d weighed survey or diet history.

After an overnight fast, subjects rested for 30 min before measuring the resting metabolic rate (RMR) by open-circuit calorimetry. After a further 45 min each subject was given, on three separate occasions, either 2·090 MJ (500 kcal) or 4·18 MJ (1000 kcal) of Complan or an equivalent volume of water after which metabolic rate (MR) was monitored for a further 270 min.

The Table shows the characteristics of the subjects in each group.

Group	High Intake (*n* 8)		Low Intake (*n* 8)	
Energy intake (MJ/d)	13·591–18·254		5·415–7·046	
	Mean	SE	Mean	SE
Age	25·1	1·5	30·6	3·3
Weight (kg)	71·2	2·8	81·4	2·7
Height (m)	1·83	0·02	1·76	0·03
Lean body mass (kg)	62·2	2·0	63·1	2·4
Body fat (%)	12·6	1·3	22·5	1·8

Test meal (MJ) . . .	4·18		2·09		4·18		2·09	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
RMR (kJ/kg per 24 h)	88·2	10·9	79·0	3·1	83·6	3·6	82·8	6·2
Peak response:								
Increase (%)	48·7	2·8	47·0	8·7	35·8	2·5	17·1	5·4
Time of peak (min)	87·5	16·4	122·5	14·2	91·3	15·5	52·5	7·5
Total response in 4 h (kJ)	296·7	42·4	210·7	40·3	226·9	31·4	93·6	29·3
Increase over RMR (%)	28·6	3·4	21·6	4·4	20·0	2·8	8·2	2·3

RMR was similar in both groups. The thermic effect of a meal was greater for the 'high intake' group than for the 'low intake' group over the measured period particularly after the 2·09 MJ meal. Increasing meal size produced a greater increment in the thermic effect in the 'low intake' group than in the 'high intake' group. These results suggest that differences in the thermic response to a meal may be important to the understanding of the differing metabolic efficiency of high- and low-energy intake groups of similar lean body mass.

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