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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Twenty-fifth Meeting of the Nutrition Society was held at Guy's Hospital Medical School, St Thomas Street, London SE1 9RT on Friday, 8 *December* 1978, *when the following papers were read:*

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The minimal nitrogen metabolism of lambs. By E. R. ØRSKOV and D. A. GRUBB, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Metabolic faecal nitrogen (MFN) and endogenous urinary N (EUN) are defined as the amounts of N excreted in faeces and urine respectively when an N-free diet is given. They are thought to represent the obligatory losses of N from the gut and tissues of the body and their sum represents the requirement of the animal for N maintenance (Mitchell, 1964; Blaxter, 1964). Faecal N in ruminants is largely bacterial; both the N of metabolic products, urea entering the gut and dietary N contribute to the N required by bacteria.

When lambs were given solutions of steam-refined volatile acids and of casein by continuous intragastric infusions for periods of weeks, no faeces were produced (Ørskov & Grubb, 1977). Under these circumstances reabsorption of the N-containing materials of abraded cells and secretion can be inferred, and the net N requirement of the animal for maintenance will be the measured EUN.

Five lambs weighing 26–32 kg which had been nourished on protein containing solutions for the previous 4 weeks were given protein-free solutions by intragastric infusion of volatile fatty acids, supplemented by minerals and vitamins for 14 d. The solution provided 450 kJ/kg $W^{0.75}$ and the proportions of acids were: acetic 550, propionic 250 and butyric 100 mmol/mol.

Urinary excretions of N fell on changing to the N-free infusate but was virtually constant after the first day when it was $5{\cdot}34 \pm 0{\cdot}20$ g N/d or about 440 mg/kg $W^{0.73}$. This value is considerably greater than a value of 90 mg/kg $W^{0.73}$ obtained for EUN from studies in twelve laboratories (see ARC, 1965). In one of the lambs receiving the infusate methylcellulose and glucose was also infused into the terminal ileum and this resulted in the production of faeces containing 0·5 g N/d. Urinary N simultaneously fell by 0·5 g N/d.

These results, together with those of Ørskov *et al.* (1970) which showed that when an increase in faecal N was invoked by increasing caecal fermentation with normally fed sheep urinary N excretion fell, suggests that a clear separation of endogenous faecal and urinary N losses cannot be made. In the present experiment, it can be calculated (ARC, 1965) that the net N requirement for maintenance would have been EUN, 1·08 g N/d, MFN 4·0 g N/d had the 30 kg lambs consumed 0·8 kg/d dry diet giving a total of 5·08 g N/d. This value is very similar to the N excretion on an N-free diet in circumstances here where no faeces were produced. (397 compared to 417 mg/kg $W^{0.75}$).

The results strongly suggest that it would be most logical not to separate EUN and MFN since they, to a large extent, appear to have a common origin and the route of excretion can be altered by dietary manipulation.

Agricultural Research Council (1965). *The nutrient requirement of Farm Livestock. No. 2 Ruminants.* HMSO.
Blaxter, K. L. (1964). In *Mammalian Protein Metabolism,* [H. M. Munro and J. B. Allison, editors]. London: Academic Press.
Mitchell, H. H. (1964). *Comparative Nutrition of Domestic Animals.* London: Academic Press.
Ørskov, E. R., Fraser, C. Mason, V. C. & Mann, S. O. (1970). *Br. J. Nutr.* **24**, 671.
Ørskov, E. R. & Grubb, D. A. (1977). *Proc. Nutr. Soc.* **36**, 127A.

0029–6651/79/3820–325A $01.35

Carbohydrates and nitrogen in stools of East African children By R. VAN RENS, *Medical Research Centre, Nairobi, Kenya* and R. LUYKEN, *Sub Department of Nutrition, Royal Tropical Institute, Amsterdam, Netherlands*

Staple food of rural Kenyan children often consists for a considerable part of whole maize grains. Undigested maize grains are found in the faeces of these children. Consequently, only a part of the ingested carbohydrates is available as energy, energy intake being often already marginal in these children. The situation is aggravated when this type of food is ingested. However, it is not known whether the quantity of carbohydrates excreted in this way is a substantial fraction of the ingested carbohydrates or a negligible one.

A rough estimation of this problem was carried out by analysing random stool samples of Kenyan and Dutch schoolchildren. Carbohydrates were analysed according to Van de Kamer (1941), nitrogen by the Kjeldahl method.

Nineteen stool samples were obtained from inhabitants of an orphanage in Nairobi (age range: 6–12 years). The main component of one of the meals was cooked whole maize (average 80 g/d) and kidney beans (90 g/d).

The morning and evening meals consisted of maize flour or rice with meat and vegetables (Pieters & Van Rens, 1973). Ten stool samples were investigated after ingesting twice the normal quantity of whole maize. Also nineteen samples were obtained from rural schoolchildren. Ten Dutch children eating a Western type of food served as a control group.

The results of the analyses are given in the Table.

Carbohydrate and nitrogen content in faeces (g/100 g wet stool)

Carbohydrate range	Orphanage (%)	Rural school (%)	Dutch school (%)
12–20		5	
10–11·9	10	5	
8– 9·9	16	10	
6– 7·9	16	16	
4– 5·9	37	5	
2– 4·9	21	26	17
0– 1·9		32	83
n	19	19	10
Carbohydrate: Mean	6·2	4·8	1·2
SD	2·55	3·61	0·67
Idem after extra maize	7·6		
Nitrogen	1·0	1·3	
SD	0·23	0·30	

It becomes clear that a considerable quantity of carbohydrate is lost by stools. The highest level found was 17·5 g/100 g of stools.

At an average production of 275 g stool/d (Burkitt *et al.* 1972), this means a loss of some 50 g carbohydrates, representing some 200 kcal (960 kJ).

Consequently, nutritionists must be aware of a possible extra loss of energy via the stools when calculating energy value of East African diets.

Burkitt, D. P., Walker, A. R. P. & Dainter, N. S. (1972). *Lancet* ii, 1408.
Pieters, J. J. L. & Van Rens, R. (1973). *Trop. geogr. Med.* **25**, 365.
Van de Kamer, J. (1941). *Chem. Weekblad* **38**, 286.

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The effects of antibodies to dietary protein on the development of atherosclerosis in cholesterol-fed rabbits. By CHITRA PATHIRANA, M. J. GIBNEY, P. J. GALLAGHER and T. G. TAYLOR, *Departments of Nutrition and Pathology, University of Southampton*

The cholesterol-fed rabbit is the most widely used model for the study of atherosclerosis. However, the resultant lesions are considerably different from those found in human atherosclerosis. This difference can be partly overcome by subjecting the animals to immunological stress with injections of foreign protein (Minick & Murphy, 1974). Alternatively, non-cholesterol-fed rabbits that develop serum antibodies to dietary protein produce a form of atherosclerosis more typical of human atherosclerosis (Gallagher *et al.* 1978). The purpose of the present study was to see whether the development of antibodies to food proteins would influence the nature of the lesions in cholesterol-fed rabbits.

Groups of five New Zealand White rabbits were given *ad lib.* for 90 d, one of four isonitrogenous (308 g crude protein/kg), high energy (17 MJ/kg) diets containing 177 g coconut oil/kg. The dietary variables were protein source and cholesterol supplementation (10 g/kg). Protein was supplied as methionine supplemented sodium isolates of casein (Casumen) or soya-bean protein (Promine D).

In non-cholesterol-fed animals, casein produced a significantly higher mean concentration of cholesterol in the serum (7·7 *v.* 4·8 mmol/l ($P<0·05$) while in cholesterol fed animals, soya-bean protein produced a higher mean value (70 *v.* 62 mmol/l). In both instances these changes were mainly associated with LDL-cholesterol. Serum antibodies to dietary protein were detected with all treatments. Casein fed animals showed low antibody titres (mean 21, range 0–32). Although soya-fed animals showed higher antibody titres (mean 42, range 0–320), they were considerably lower than previously observed (Gallagher *et al.* 1978). This may have been due to perinatal exposure to the soya antigen in breeding stock diet.

The aortae were scored from 8–40 with increasing degree of atherosclerosis. Marked atherosclerosis, typical of the cholesterol fed rabbit was not influenced significantly by protein source (18–33 for casein, 30–34 for soya protein). The feeding of casein in the absence of cholesterol produced small amounts of atherosclerosis more typical of the spontaneous than cholesterol-induced atherosclerosis.

It is concluded that the levels of antibodies to dietary protein obtained in this experiment did not constitute a sufficiently strong immunological injury to alter the nature of the cholesterol-induced atherosclerosis.

Gallagher, P. J., Muir, C. A. & Taylor, T. G. (1978). *Atherosclerosis* **30**, 361.
Minick, C. R. & Murphy, G. E. (1974). *Adv. Exp. biol. med. Sci.* **43**, 355.

0029–6651/79/3820–325A $01.35

Some effects, in baboons, of consuming a galactose:glucose mixture. By CELIA WILLIAMS (Introduced by I. MACDONALD), *Department of Physiology, Guy's Hospital Medical School London SE1 9RT*

Six male and six female adult baboons were given a liquid diet composed of acid hydrolysate of lactose (75 parts) calcium caseinate (18 parts) with dried yeast, salts and vitamins for 10 weeks with fasting blood samples taken every two weeks. At the beginning and end of the experiment 0·5 g/kg body-weight of galactose in 4 ml/kg body-weight water was given and as with fasting blood, estimations of galactose, glucose, insulin, triglycerides and cholesterol made at intervals up to 180 min. Identical experiments were also carried out using glucose instead of the lactose hydrolysate.

The results showed that the level of galactose increased significantly, albeit slightly, in fasting blood on the lactose hydrolysate diet, and on this diet the glucose levels after a galactose dose were higher than at the beginning. The insulin response to galactose after 10 weeks on the lactose hydrolysate diet was lower than at the start. The level of triglyceride in fasting serum rose on both types of dietary carbohydrate to a similar extent in the male animals but in the females the increase on the hydrolysate diet was much greater than on glucose. The cholesterol level decreased on both diets to a similar extent.

These results suggest that galactose has metabolic effects in baboons that are different from those of glucose.

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Seasonal variations in energy intake, body-weight and skinfold thickness in pregnant and lactating women in rural Gambia. By ALISON A. PAUL, ELISABETH M. MÜLLER* and R. G. WHITEHEAD, *MRC Dunn Nutrition Unit, Milton Road, Cambridge CB4 1XJ, and Keneba, The Gambia*

In the village of Keneba, The Gambia, a subsistence farming community, pregnant and lactating women are engaged in heavy agricultural duties during the rainy season from June to November. The period of greatest energy expenditure thus coincides with the traditional hungry season, and the women lose weight at this time (Thomson *et al.* 1966). Breast milk output is also lower during the rainy season.

The food intake of thirty-three women who were between the 4th month of pregnancy and 7th month of lactation was measured by weighing all meals on 6 d each month. Foods eaten between meals were estimated by a carefully standardized recall procedure. Ten to fifty samples of each food eaten were analysed for gross energy (GE), nitrogen (N), fat, available and unavailable carbohydrates (UC): metabolizable energy (ME) was calculated using: ME=0·977GE–6·6N–4UC (Southgate, 1975).

Energy intakes were lower between July and October than the rest of the year with extremely low intakes of 5·0 MJ (1200 kcal) being recorded in August.

		March–June		Rains: July–October		November–December	
		Mean	SEM	Mean	SEM	Mean	SEM
Pregnant women	MJ	6·8	0·29*	5·6	0·38	5·9	0·71**
	(kcal)	(1620)	(70)	(1340)	(90)	(1420)	(170)
	n	17		14		6	
Lactating women	MJ	7·3	0·75	5·7	0·33	7·9	0·33***
	(kcal)	(1740)	(180)	(1370)	(80)	(1890)	(80)
	n	8		11		17	

Significantly different from July–October, *$P<0·02$, ***$P<0·001$.
Significantly different from lactating women in November–December, **$P<0·01$.

Seasonal changes in body-weight and skinfold thickness were closely related to changes in energy intake and during lactation, to changes in breast milk output. The regain in body-weight after the wet season loss occurred more rapidly than the rise in breast milk output, suggesting that extra food is first used for maternal needs. If this finding is substantiated by further results from this ongoing study, it could have important implications for the provision of maternal dietary supplements.

We are grateful to G. J. Hudson, P. M. V. John and K. C. Day for technical assistance. E.M.M. was supported by the 'Co-operation of Technique Suisse' and 'Stiftung zur Forderung der Ernahrungsforschung in der Schweiz'.

Southgate, D. A. T. (1975). *Proc. Western Hemisphere Nutrition Congress* IV p. 51 [P. L. White and N. Selvey, editors]. Acton, Mass: Sciences Group Inc.
Thomson, A. M., Billewicz, W. Z., Thompson, B. & McGregor, I. A. (1966). *J. Obstet. Gynaec. Brit. Cwlth.* **73**, 724.

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The accumulation of copper in the liver of the domestic laying hen. By MARY H. STEVENSON and N. JACKSON, *Department of Agricultural and Food Chemistry, The Queen's University of Belfast and Department of Agriculture, Northern Ireland*

Jackson (1977) gave graded levels of copper up to 1936 mg Cu/kg diet to laying hens for 5 weeks and found that liver concentrations of Cu and iron were significantly increased by high dietary Cu. In order to extend the information on the effect of Cu on the laying hen, the rate of accumulation of Cu in liver was examined. In addition, zinc and Fe levels were also measured because of the known interactions of these two elements with Cu.

One hundred and fifty-six birds were randomly allocated to one of five treatment groups each containing thirty birds. Six other birds were killed prior to the start of the experiment (pre-experimental control birds). The birds were fed a control diet and this diet supplemented with 250, 500, 1000 and 2000 mg Cu/kg. Six birds from each treatment were killed 3, 6, 12, 24 and 48 d after the start of the experiment. Livers were removed, weighed, oven-dried and Cu, Zn and Fe concentrations measured.

The concentrations of Cu in the livers of control birds and those receiving diets supplemented with 250 mg Cu/kg diet were significantly lower ($P<0.001$) than those of birds receiving diets supplemented with 500–2000 mg Cu/kg. These higher Cu concentrations in the livers were significant after only 3 d on Cu supplemented diets and continued to increase up to 12, 24 and 48 d for the 500, 1000 and 2000 mg Cu/kg supplemented diets respectively. The Cu concentrations of the livers of birds receiving diets supplemented with 2000 mg Cu/kg increased from 74·0 μg Cu/g DM at 3 d to 1790 μg Cu/g DM at 48 d. The liver Cu concentration of the pre-experimental control birds was 11·3 μg/g DM. The effects of Cu supplemented diets on the levels of liver Zn were variable. After 3 and 6 d on diets supplemented with 1000 and 2000 mg Cu/kg, the liver Zn concentrations were significantly higher ($P<0.001$) than for the birds receiving the control and two lower Cu supplemented diets. However, after 6 d the effect was no longer apparent. Liver Fe concentrations were unaffected by all Cu treatments for 6 d but after 12 d the highest Cu treatment had significantly ($P<0.05$) increased the liver Fe concentration. By 24 d, diets supplemented with both 1000 and 2000 mg Cu/kg had significantly ($P<0.01$ for Tr 4, $P<0.001$ for Tr 5) increased liver Fe concentrations and these levels were further increased by 48 d. After 48 d, the liver Fe concentrations had increased from 344 μg/g DM for the control diet to 986 and 2025 μg/g DM for 1000 and 2000 mg Cu/kg supplemented diets respectively.

Jackson, N. (1977). *Br. J. Nutr.* **38**, 93.

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Some differences between dietary carbohydrates in their effects on weight loss and body-fat in rats. By I. MACDONALD and T. H. GRENBY, *Guy's Hospital Medical and Dental Schools, London SE1 9RT*

There are reports that in rats and baboons the rate of weight gain is influenced by the type of carbohydrate in the diet (Allen & Leahy, 1966; Brook & Noel, 1969), and one report in man indicated that the rate of weight loss on energy-restricted diets can vary depending on the nature of the carbohydrate in the diet (Macdonald & Taylor, 1973).

To investigate this variation in the metabolism of different carbohydrates, twenty-four rats of each sex weighing approximately 500 g (♂) and 300 g (♀) were given diets containing either sucrose or glucose as the only carbohydrate source. The energy value of the diets was determined by bomb calorimeter.

In the initial phase of the experiment the amount of diet presented to each animal each day (4·2 kJ/100 g body-weight) was less than its energy requirement. The rats were weighed daily, and the weight of food presented was adjusted as necessary. At the end of this initial phase (♂ rats 22 d; ♀ rats 13 d) the amount of food presented was doubled and then kept at this level for a further 44 d (♂ rats) and 57 d (♀ rats). At the end of this second phase they were killed and blood, liver and carcass lipids were determined.

The rate of weight loss was significantly greater on the glucose than on the sucrose diet in both sexes. Expressing body-fat deposits as a percentage of total body-weight, in the males there was significantly more body-fat on the sucrose than on the glucose regime.

Allen, R. J. L. & Leahy, J. S. (1966). *Br. J. Nutr.* **20**, 339.
Brook, M. & Noel, P. (1969). *Nature, Lond.* **222**, 562.
Macdonald, I. & Taylor, J. (1973). *Guy's Hosp. Rep.* **122**, 155.

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The effect of feeding cellulose to rats and rabbits on the fragility of the erythrocyte membrane. By N. J. GOULDING, D. R. HUSBANDS*, P. G. R. BURSTYN and T. G. TAYLOR, *School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 5NH*

In an investigation of the effect of feeding diets containing 200 g/kg of palm oil and safflower oil on the blood pressure of rabbits (Kennedy *et al.* 1978) the fragility of erythrocytes was also measured to assess changes in membrane properties. When cellulose was added to the palm oil diet the fragility of the erythrocytes was increased. This effect was also studied in growing rats. Male rats (100 g) were allocated to two groups, each of five animals. The basal diet contained (g/kg) soya flour (500), barley (213), palm oil (150), dried yeast (73) and a vitamin and mineral mix. One group of rats was fed on the basal and the other group was given the same diet diluted with cellulose (solkafloc) to give 140 g cellulose/kg diet.

The rats were fed these diets for 14 d at which time they weighed about 200 g each. No difference between groups in the rate of growth was noted. Blood samples were taken by cardiac puncture and the erythrocytes obtained by centrifugation. Erythrocyte fragility was measured by determining the amount of haemoglobin released into solution at various concentrations of sodium chloride and the molarity of NaCl giving 50% haemolysis was calculated. Feeding cellulose led to increased fragility as with the rabbits.

Sodium chloride concentrations (mM) *giving* 50% *haemolysis*

(Mean values with their standard errors)

Dietary treatment		No. of	Rabbits		Rats	
Oil	Cellulose (g/kg)	animals	Mean	SE	Mean	SE
Safflower	—	4	81·9	0·9	—	—
Palm	—	5	81·9	0·7	—	—
Palm	200	5	84·1	0·6*	—	—
Palm	—	5	—	—	66·1	0·5
Palm	140	5	—	—	69·8	1·1*

*Significantly higher than corresponding low-fibre group, $P<0.05$.

The fatty acid composition of the erythrocyte membrane lipids showed no statistically significant changes between the two groups of rats. Changes in blood chemistry in rabbits fed palm oil diets with differing levels of fibre have been reported (Kennedy *et al.* 1978) but it is unclear how these changes relate to the fragility of erythrocyte membranes.

Kennedy, M., Burstyn, P. G. R. & Husbands, D. R. (1978). *Proc. Nutr. Soc.* 37, 98A.

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The effect of the dietary addition of Monensin upon digestion in the stomachs of sheep. By J. D. ALLEN and D. G. HARRISON, *Department of Agricultural Biochemistry, University of Newcastle upon Tyne, NE1 7RU*

Monensin is a ruminant feed additive which is known to increase feed conversion efficiency in fattening steers (Perry *et al.* 1976). The additive also increases the molar proportion of propionate in the rumen liquor (Dinius *et al.* 1976), indicating a marked effect upon rumen fermentation, and thus it was appropriate to investigate the over-all effects of Monensin upon digestion in the rumen.

Four mature wether sheep, each fitted with a rumen fistula and a re-entrant cannula at the proximal duodenum were fed a diet of dried grass nuts (920 g DM/d) and ground maize (90 g DM/d). The diet provided an intake of 920 and 24·4 g/d of organic matter (OM) and nitrogen respectively. Rumen pH, dilution rate, molar proportions of volatile fatty acids (VFA) and VFA production were measured. Duodenal digesta was collected over 24 h, using lignin as a marker, and the relative proportions of feed and microbial protein in the duodenum were estimated with ^{35}S. Monensin was then added to the maize, giving a concentration in the whole diet of 22 mg/kg and the experimental observations were repeated.

	Control	Monensin	SEM†	
Rumen pH	6·53	6·46	0·026	NS
Rumen molar proportions of:				
Acetate	0·672	0·655	0·0053	NS
Propionate	0·225	0·276	0·0042	••
C_4 acids	0·087	0·052	0·0007	••
C_5 acids	0·016	0·017	0·0033	NS
Rumen dilution rate (/h)	0·071	0·041	0·0040	•
VFA production (M/d)	4·21	4·55	0·022	••
OM apparently digested in stomachs (g/d)	407	442	3·8	•
Microbial N synthesized/kg of OM truly digested in stomachs (g)	24·5	20·2	0·16	••
Y(ATP)‡	14·9	11·8	0·30	•

NS, non-significant; •$P<0·05$, ••$P<0·01$.
†Pooled SEM derived from the analysis of variance.
‡Microbial DM (g) synthesized/mole of ATP derived from the fermentation. Values estimated from VFA production (Harrison *et al.* 1975).

Monensin has no effect upon rumen pH, but significantly increased the molar proportions of propionate and significantly reduced both the molar proportions of butyrate and the dilution rate. The additive significantly increased VFA production and the quantity of OM apparently digested in the stomachs. These findings suggest that Monensin may increase the rumen retention time, thus increasing ruminal OM digestion and VFA production, but markedly reduces the efficiency of microbial growth, implying a partial uncoupling of the rumen fermentation. These observations are in good agreement with the in vitro results of Van Nevel & Demeyer (1977).

Dinius, D. A., Simpson, M. E. & Marsh, P. B. (1976). *J. Anim. Sci.* **42**, 229.
Harrison, D. G., Beever, D. E., Thomson, D. J. & Osbourn, D. F. (1975). *J. agric. Sci. Camb.* **85**, 93.
Perry, R. W., Beeson, W. M. & Mohler, M. T. (1976). *J. Anim. Sci.* **42**, 761.
Van Nevel, C. J. & Demeyer, D. I. (1977). *Appl. Env. Microbiol.* **34**, 251.

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The relationship between dietary state, thyroid hormones, oxygen consumption and muscle protein turnover. By D. J. MILLWARD, M. A. HOLLIDAY, P. C. BATES, S. DALAL, M. COX and C. R. C. HEARD, *Clinical Nutrition and Metabolism Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

Reduced muscle protein turnover is a feature of malnutrition (Millward & Waterlow, 1978). Furthermore a fall in metabolic rate accompanies prolonged fasting in adults. T_3 may mediate each of these changes since it falls in fasting man (Portnay *et al.* 1974) and appears to regulate lysosomal enzyme levels in rats (de Martino & Goldberg, 1978). We have measured therefore T_3 levels, oxygen consumption (Stock, 1975) and the rates of muscle protein synthesis in malnourished rats, and in hypophysectomized and thyroidectomized rats treated with T_3.

Protein synthesis (see Table) was reduced in the malnourished and thyroid deficient rats, while T_3 administration to the latter animals increased protein synthesis (and promoted growth in the thyroidectomized rats). Oxygen consumption (VO_2) was also reduced in the malnourished and thyroid deficient rats, while T_3 administration to the latter animals increased the VO_2. However the changes in protein turnover and VO_2 cannot be explained entirely in terms of T_3 levels. Although T_3 levels were reduced in the energy deficient rats it was elevated in the protein deficient animals. Thus it would appear that any relationship between T_3 and protein turnover is not a simple one.

Relationship between thyroid status, oxygen consumption and protein synthesis in muscle of rats

Dietary or endocrinological state	Rate of protein synthesis (%/d) Mean	SD	VO_2 (L/D per $kg^{0.56}$) Mean	SD	T_3 (ng T_3/ml) Mean	SD
Hypophysectomized	3·0	0·5	10·76	0·67		
Hypophysectomized + T_3 (5 μg/2 d)	8·4	2·1	13·23	0·78		
Thyroidectomized	4·3	0·6	14·15	0·29		
Thyroidectomized + T_3 (5 μg/2 d)	9·6	2·8	17·7	0·9		
Well fed	11·7	2·2	20·5	2·5	2·0	0·1
Protein deficient (3·5% protein *ad lib.*)	5·8	1·05	12·7	0·4	2·21	0·3
Energy deficient (10% protein, restricted)	4·3	0·4	13·0	1·7	0·66	0·06

de Martino, G. & Goldberg, A. L. (1978). *Proc. natn. Acad. Sci.* USA **75**, 1369.
Millward, D. J. & Waterlow, J. C. (1978). *Fedn Proc. Fedn Am. Socs exp. Biol.* **37**, 2283.
Portnay, G. I., O'Brian, J. T., Bush, J., Vagenakis, A. G., Azizi, F., Arley, R. H., Ingbar, S. H. & Brauerman, L. E. (1974). *J. clin. Endocr. Metab.* **39**, 191.
Stock, M. J. (1975). *J. appl. Physiol.* **39**, 849.

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The effect of snack and beverage consumption on the pH of human dental plaque. By G. N. JENKINS, W. M. EDGAR and A. J. RUGG-GUNN, *Department of Oral Physiology, Dental School, University of Newcastle upon Tyne, Framlington Place, Newcastle upon Tyne NE2 4BW*

Dental caries are believed to result from dissolution of the crystallites of the hard dental tissues following the production of acid from dietary carbohydrates (principally sucrose) by bacteria on the tooth surface (the dental plaque). The factors accounting for the cariogenicity of a food are not however fully understood. Because human clinical studies are impracticable, little evidence has hitherto been available to form a basis for rational dietary advice to reduce caries, except to minimise sugar intake especially between meals.

Measurement of pH changes in dental plaque may provide such evidence, on the probable assumption that these pH changes are the most important factor related to the cariogenicity of a food. The existence of protective factors in some foods which reduce the effect of acids, and the possible formation of non-acid complexing substances cannot be dismissed, however.

Using a glass microelectrode system, we have monitored the pH of plaque samples removed from volunteers before and at 4 min intervals over 30 min after consuming foods (snacks, sweets, beverages) singly and in combination with other foods in sequence as in meals. Foods show a wide variation in their acid producing potential; those which contain low concentrations of sugars, or are consumed rapidly, or provoke a high rate of salivation, and/or themselves have a neutral or alkaline pH, give rise to a small drop in pH, while foods having some or all of the contrary characteristics are highly acid-forming. Examples of the first group include sugarless chewing gum, peanuts, cheese, bread and butter, milk and unsweetened tea and coffee, while the most acid-forming foods include boiled sweets, toffees, sugared coffee and tea, diluted orange drinks and biscuits. Cheese or peanuts eaten when the plaque pH is low after eating a sugary food cause the pH to rise, probably owing to the protective flow of neutralizing saliva.

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The relationship between nitrogen and sulphur excretion in the urine of patients receiving intravenous feeding. By M. H. JOURDAN, *Department of Surgery, Guy's Hospital, London SE*1

In a dietary study undertaken on obese women it was found that reducing the energy intake of the subjects to 50% of that required for weight maintenance resulted in an increase in total sulphur: total nitrogen in the urine. (Jourdan *et al.* submitted for publication). One possible explanation for this finding is that the breakdown of sulphur relative to non-sulphur containing amino acids is increased at times of energy deprivation. This led us to wonder if such a change might also occur during the altered metabolism of the post-operative surgical patient.

Fourteen patients undergoing major surgery and fed intravenously for at least 10 d post-operatively were studied. 24 h collections of urine were made starting pre-operatively, the samples being kept at 0–4°. The total nitrogen content was determined by the micro-Kjeldahl technique, and the total sulphur by an automated modification of the methods described by Garrido (1964).

In eleven of the patients the ratio of sulphur to nitrogen in the urine was as expected from the composition of the amino acid mixture used for the intravenous feeding. In three of the patients however, significantly more sulphur relative to nitrogen was excreted in the urine than expected, and it may be that in certain patients there is relative sparing of some non-sulphur containing amino acids as part of the metabolic response to trauma.

Garrido, M. J. (1964). *Analyst* **89**, 61.
Oser, B. L. (1954). In *Hawk's Physiological Chemistry*. New York: McGraw-Hill.

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Vitamin D in the nutrition of the cat. By J. P. W. RIVERS and T. L. FRANKEL, *Institute of Zoology, Zoological Society of London, Regent's Park, London NW1 4RY* and S. JUTTLA and A. W. M. HAY, *Department of Animal Physiology and Nutrition, University of Leeds LS5 3HL*

Little is known of the role of vitamin D in feline nutrition. Rickets occurs rarely and then only in kittens kept in the dark (Scott, 1965), and kittens made experimentally rachitic recover spontaneously after a year on a deficient diet (Gershoff *et al.* 1957). This paper is a report on preliminary studies on normal levels of vitamin D in the cat, and whether either a deficiency disease or a syndrome of hypervitaminosis D can be induced in the animal.

In cats fed a diet of proprietary canned cat foods and shielded from ultraviolet light the mean plasma 25-hydroxycholecalciferol ($25OHD_3$) level, determined by a modification of the method of Haddad and Chyu (1971), was 85·8 ng/ml (SD±23·8). Levels were higher in older animals.

In animals weaned onto a semi-purified vitamin D_3-free diet, and also shielded from ultraviolet light plasma $25OHD_3$ levels fell exponentially with time. The mean half life of plasma $25OHD_3$ was calculated to be 8 weeks and after 9 months on this diet levels were too low to be detected in most animals. However, in animals kept on the diet for longer periods $25OHD_3$ could be detected in plasma and it showed some tendency to rise with age. This phenomenon, which possibly reflects mobilization of body stores acquired before weaning, may well explain the spontaneous cure of rickets observed by Gershoff *et al.* (1957).

Despite long periods of vitamin D deprivation, however, no signs of bone disease were seen in our animals, except for a slight slowing in the rate of epiphyseal closure. This confirms the view of Scott (1965) that vitamin D requirements of the cats are low, at least on diets like ours with a balanced Ca:P.

In a separate experiment animals were fed the semi-purified diet but with vitamin D added at 2·5 mg D_3/kg diet (approximately 0·5 mg D_3/4200 kJ). After 8 months on this diet all the animals had excessive mineralization of bone, and most had extensive calcification of the soft tissues. These changes could be reversed by removing vitamin D from the diet, or by feeding the diet of canned cat foods.

We conclude that though the cat does require vitamin D its requirement is extremely low and is probably negligible in all except long-term experiments. Vitamin D_3 is toxic to the cat and in the normal nutrition of the pet cat the risk of toxic effects of vitamin D supplementation undoubtedly outweigh any putative benefits.

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Gershoff, S. N., Legg, M. A., O'Connor, F. J. & Hegsted, D. M. (1957). *J. Nutr.* **63**, 79.
Haddad, J. G. & Chyu, K. J. (1971). *J. Clin. endocr. Metab.* **33**, 992.
Scott, P. P. (1965). In *Canine and Feline Nutritional Requirements* [O. Graham-Jones, editor]. Oxford: Pergamon Press.

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The effect of palatability on energy intake in two obese women. By MERRIL DURRANT and R. WLOCH, *Medical Research Council, Clinical Research Centre, Watford Road, Harrow HA1 3UJ*

Siverstone & Fincham (1977, 1978) described a refrigerated food vending machine for studying human food intake and demonstrated the effects of daily doses of anorectic drugs. The machine has four stacks, each with 30 buckets, so 120 items of food can be presented. Food is obtained by inserting a coin and pressing a button below the stack; the button press is recorded as the eating event. A control unit has been added which can set mealtimes at intervals throughout the 24 hours. Three daily meals were set at 09.00–10.00, 13.00–14.00 and 18.00–03.00 hours. At the end of each mealtime the stacks cycled on any uneaten food and aligned the new food for the next meal. The machine was checked and reloaded daily.

Two obese female subjects who confessed to compulsive overeating were studied. The protocol was approved by the hospital Ethical Committee. A 16·7 MJ/d menu was devised for each patient. Each meal comprised four different foods; two types were of normal palatability (e.g. sandwich, cheese, egg, fruit, salad) one type of beverage (e.g. milk, juice, oxo) and one type of highly palatable 'luxury' food (e.g. chocolate, biscuit). There were eight foods of each type i.e. thirty-two foods/meal. Patients were given sufficient coins to empty the machine when each item cost 1p (96p). Two days were run as baseline then for the next two days 'luxury' food was priced at 5p and for a further two days at 10p each. Patients were given no extra coins, thus forcing them to choose how to spend their money. Finally they were offered only food they disliked for the last two days.

	Subject AT		Subject JC	
	Energy intake MJ/d	Luxury energy (%)	Energy intake MJ/d	Luxury energy (%)
1p all foods	7·71±1·32	35	11·87±0·88	52
5p 'luxury' foods	8·21±1·18	17	9·80±0·39	28
10p 'luxury' foods	7·71	7	8·75±0·42	12
Disliked foods	3·23±0·74	—	6·31±1·09	—

Subject JC ate more and obtained a greater percentage of energy from luxury food. The extra cost of the luxury food forced her to reduce her intake. AT's energy intake from luxury food declined but her total energy intake was maintained by eating more 'normal' food. Both patients intensely disapproved of the period of disliked food and reduced their intake. AT showed more finnicky behaviour, both subjects tended to maintain energy intake at the expense of palatability.

Silverstone, J. T. & Fincham, J. (1977). *VIth International Congress on the Physiology of Food and Fluid Intake.*

Silverstone, J. T. & Fincham, J. (1978). In *Central Mechanisms of Anorectic Drugs* [S. Garattini and R. Samanin, editors]. New York: Raven Press.

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Serum carbohydrate levels following galactose and galactose plus glucose given to rats. By G. C. NEWSTEAD, *Department of Physiology, Guy's Hospital Medical School, London SE1 9RT*

Oral galactose tolerance tests (0·5 g/kg body-weight) were performed on ten fasting male Tuck Wistar rats weighing approximately 400 g. The galactose was delivered via an oro-gastric tube while the animals were lightly anaesthetised with diethyl ether. In another series of experiments in addition to the galactose, glucose was administered (0·5 g/kg body-weight) as an isotonic injection into the peritoneal cavity. In a third group sham experiments, using saline, were carried out.

The results show that the mean fasting serum galactose level in these animals is 5·0 mg/100 ml, a value approximately seven times larger than that in fasting man (Jolley *et al.* 1970). The fasting glucose level unlike the galactose concentration, is sensitive to experimental manipulations such as anaesthesia and intubation.

Following oral galactose loading, the serum galactose concentration rises rapidly and is still high 2·5 h later, much higher than values reported from experiments on man (Stenstam, 1946), dogs (Bollman *et al.* 1935) or rabbits (Roe & Schwartzman, 1932). It seems that the rat's rate of clearance of galactose is very slow compared to other adult animals that have been investigated.

The blood glucose levels, however, show little change, but the tendency is for the concentration of glucose to decrease, this is opposite to that which is observed in rabbits, dogs and man under similar circumstances.

Several workers have reported that if glucose is consumed by man at the same time as galactose, the galactosaemia is much less than after the consumption of galactose alone. However, in the experiments reported here the serum galactose levels are higher with simultaneous intraperitoneal glucose than when galactose is given alone. This suggests that in the rat glucose does not reduce the level of serum galactose.

It therefore appears that the metabolism of galactose by the rat is dissimilar to that reported in dogs, rabbits and man.

Bollman, J. L., Mann, F. C. & Power, M. H. (1935). *Am. J. Physiol.* **111**, 483.
Jolley, R. L., Warren, K. S. & Scott, C. D. (1970). *Am. J. Clin. Pathol.* **53**, 793.
Roe, J. H. & Schwartzman, A. S. (1932). *J. biol. Chem.* **96**, 717.
Stenstam, T. (1946). *Acta. med. scand. Suppl.* **177**, 125.

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The quantitative analysis of sugars in commercially-available yoghurts.

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The carbohydrate content of fermented milk products such as yoghurt has not been widely documented. The few studies that have been carried out have been on either American (Goodenough & Kleyn, 1976) or Australian (Shanley, 1973) products and have used tedious paper chromatographic separations. Consequently, we have developed a rapid assay method based on thin-layer chromatography for the separation and quantitative analysis of lactose, galactose, glucose and any other sugars which may be added to the fermented milk preparation, e.g. sucrose and fructose.

Commercially-available yoghurts from Great Britain and Iran were analysed for their carbohydrate contents. In all cases a high concentration of lactose (1·05–5·99%, w/v) was detected as was a significant quantity of galactose (0·05–1·7%, w/v). Glucose could be found in only two British and one Iranian yoghurt, and then only at very low concentrations (0·04–0·38%, w/v). Neither sucrose nor fructose could be detected in any of the yoghurts.

In order to mimic the production of yoghurt, boiled, pasteurised milk was inoculated with yoghurt from Iran, incubated at 37° for several days and the sugar content analysed each day. The lactose concentration diminished slowly with an obvious decrease after day 4, while the level of galactose reached a maximum on day 3 (1·6%, w/v), then decreased until disappearing completely on day 5. No glucose was detected at any stage of the experiment. Home-produced yoghurt of this type is normally consumed after the second day i.e. when it contains both lactose and galactose.

A similar experiment was carried out using strains of the two bacteria present in British yoghurts; *Lactobacillus bulgaricus* 2075 and *Streptococcus thermophilus* 2142. These were grown separately at 42° on defined media containing 10mM-lactose (3·4 mg/ml) as the sole carbohydrate source. Hourly measurements of growth, pH and sugar content of the medium revealed that 6 hours after inoculation both organisms had reached the stationary phase of growth and that the lactose concentration had fallen to approximately 0·5 mg/ml, in each case. Significant quantities of galactose were also found, reaching a maximum concentration of 0·8 mg/ml after 7 h. Only small quantities of glucose ($\leqslant$0·04 mg/ml) were detected at any stage and this sugar had disappeared completely from the medium 4 h after inoculation, thus explaining the absence of glucose in the commercially-prepared and laboratory-made yoghurts.

In conclusion, our studies show that fermented milk products, which are favoured by the hypolactasic communities in the Middle East and India, have a surprisingly high lactose content. In addition, yoghurt provides one of the few dietary situations where free galactose is available for absorption. This may have some significance to the management of patients with defects related to galactose metabolism.

Goodenough, E. R. & Kleyn, D. H. (1976). *J. Dairy Sci.* **59**, 45.
Shanley, R. M. (1973). *Aust. J. Dairy Technol.* **28**, 58.

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Early postoperative feeding with elemental diet. By S. SAGAR and R. SHIELDS, *Department of Surgery, University of Liverpool, Liverpool L69 3BX*

It is generally accepted that patients on hospital diet lose weight during a prolonged stay. This is of significance after operations with regard to postoperative recovery. We have studied several metabolic and clinical factors in 30 patients undergoing major surgery. These patients were randomly allocated into two groups: Elemental diet group (ED), fifteen patients who were fed with elemental solutions in the immediate postoperative period (days 1 to 7); and Control group, fifteen patients who were treated nutritionally in the conventional manner. Postoperative recovery of the patients in the Elemental diet group was quicker than the patients in the Control group, median 14 d *v.* 19 d in the control group). Weight change was less in ED group (median 0 *v.* median −1·85 kg). The nitrogen balance in both groups was significantly different—over-all mean deficit of 50·8 g in controls; 20·9 g in ED group. Patients in ED group were in a positive balance on the seventh day whilst Control group were still −2·8 g.

These are the preliminary clinical and metabolic results of this study. Postoperative nitrogen balance in these surgical patients will be discussed together with the value of early elemental feeding.

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Retinol content of human livers from autopsies in London. By T. HUQUE and A. S. TRUSWELL, *Nutrition Department, Queen Elizabeth College, London W8 7AH*

The retinol content of human liver tissue, obtained at autopsy, was determined in a group of 281 subjects. The histogram of frequency distribution was sharply skewed to the left. The mean value was 242 μg/g, with a median of 181 μg/g and a range of 6–1201 μg/g. 50% (*n* 141) of the values were in the 100–300 μg/g range, which is sometimes considered to be the 'normal' range for liver retinol.

Only 6% (*n* 18) of the values were below 40 μg/g, the arbitrary cut-off point below which individuals are considered to be at risk. On the other hand, nearly 10% (*n* 27) of the subjects had stores exceeding 500 μg/g.

There were no sex differences—the median values for 173 male and 108 female subjects were 189 and 181 μg/g respectively.

Median retinol stores varied markedly with age. They were relatively low (121 μg/g) in infancy (0–1 year) but were about three times greater in childhood (1–9 years) and adolescence (10–19 years). Reserves reached a peak (402 μg/g) in young adulthood (20–29 years) before declining gradually with increasing age to a low of 85 μg/g in those above 90 years.

Two hundred and fifty-eight subjects were classified according to primary cause of death, and the results are shown in the Table.

Retinol content of human livers in relation to cause of death

		Retinol (μg/g)		
Cause of death	*n*	Median	Range	%<40 μg/g
Accidental	57	270	69–1201	0
Cardiovascular diseases	137	193	10–1132	3
Respiratory diseases	51	141	6–937	18
Cancer	13	105	18–563	15

Retinol stores in our survey are substantially higher than those reported from North America and are exceeded only by those in New Zealand and Ghana (Underwood, 1974). In sharp contrast to the situation in Canada, where nearly 10% of the subjects were reported to have no detectable retinol stores (Hoppner *et al.* 1969) there were no such individuals in our survey.

Hoppner, K., Phillips, W. E. J., Erdody, R. T., Murray, T. K. & Perrin, D. E. (1969). *Can. med. Ass. J.* **101**, 736.
Underwood, B. A. (1974). *Wld. Rev. Nutr. Diet.* **19**, 123.

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An effect of low- and high-fat diets on the depot fat of rats. By F. P. JENKINS and P. H. HAGUE, *Environmental Safety Division, Unilever Research Laboratory, Colworth House, Sharnbrook, Bedford MK44 1LQ*

Four groups of 4–5 week-old, Colworth–Wistar albino rats were fed a normal diet *ad lib.* during weeks 1, 8 and 9. During weeks 2–7 the rats were restricted to equal energy intakes provided by diets fed twice daily at 09.00 hours and at 16.00 hours. The composition of the diets ensured that protein, mineral and vitamin intakes would be equal for equal energy intakes. Thus a normal diet (N), a high-fat diet (H) and a low-fat diet (L) containing 7·5, 26·0 and 4·8% sunflower-seed oil respectively were fed as shown in Table 1.

Table 1.

	Week 2		Weeks 3–6		Week 7	
Group	09.00 hours	16.00 hours	09.00 hours	16.00 hours	09.00 hours	16.00 hours
HH	N	N	H	H	N	N
LL	N	N	L	L	N	N
HL	N	N	H	L	N	N
LH	N	N	L	H	N	N

At the end of week 9 the rats were killed and testicular fat pads were weighed, mean values for groups of sixteen rats are shown in Table 2.

Table 2.

Group	Final body-wt (g)	Weight of testicular fat pads (g)	Relative weight of testicular fat pads (g/100 g body-wt)
HH	307·1	4·978	1·618
LL	304·4	5·591	1·839
HL	300·4	4·904	1·633
LH	303·0	5·077	1·679
Significant difference ($P<0·05$)	8·8	0·445	0·148

Energy intakes and body-weights were almost identical for all groups in each week of the test. However, at the end of week 9 the weights of testicular fat pads differed between groups; those of group LL, which were fed the low-fat diet during weeks 3–6, were heavier than those of other groups ($P<0·05$).

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Ascorbic acid nutrition on a British Antarctic base. By S. VALLANCE* (Introduced by M. A. K. Westland), *British Antarctic Survey, Madingley Road, Cambridge CB3 0ET*

In response to reports of suspected ascorbic acid deficiency in some men on British bases in Antarctica, an investigation into the ascorbic acid content of the diet and the nutritional status of the men living on one British Antarctic Survey base (Adelaide Island, 67°46'S, 68°54'W) was undertaken between February 1972 and February 1973.

The base diet consisted mainly of canned and dried foods. These were analysed by the 2:6 dichlorphenolindophenol method of Harris & Olliver (1942) and found generally to be poor sources of ascorbic acid. Diet sheets completed on twenty occasions by the eight men who wintered on the base showed the basic diet to provide $12 \cdot 3 \pm 8 \cdot 0$ mg ascorbic acid/man per d. This was supplemented by small quantities of canned fruit juices and cordials, which some men took only rarely, while others drank them on all available occasions.

Plasma and leucocyte ascorbic acid levels were measured by the 2:4 dinitrophenylhydrazine method of Denson & Bowers (1961) on eight occasions from the eight wintering men. These reflected the poor dietary intake. Twenty-two (35%) of the plasma levels were $<2 \cdot 5$ mg/l and thirty-eight (60%) were $<4 \cdot 0$ mg/l. Twenty-two (35%) of the leucocyte levels were <15 $\mu g/10^8$ leucocytes. One man intermittently took 500 mg tablets of ascorbic acid from a private supply and four of the men received a course of 1 g ascorbic acid daily for one week before the fourth analysis.

Clinical signs of scurvy were not seen in the men on base, but two men from another base (Stonington Island) who spent five months in the field during the austral summer, living off field rations, reported signs of scurvy which responded to vitamin supplement. These men did not routinely take the vitamin supplement capsules provided in their field rations, which were otherwise very deficient in ascorbic acid.

Previous workers have suggested an increased requirement for ascorbic acid in men living in polar regions. However, comparison of ascorbic acid intake to blood levels gave similar results to those of workers in more temporate climates.

It was concluded that, without vitamin supplement capsules normally available to, but not always taken by, men on British bases, ascorbic acid deficiency was a distinct danger.

I am indebted to the British Antarctic Survey who supported this work.

Denson, K. W. & Bowers, E. F. (1961). *Clin. Sci.* **21**, 157.
Harris, T. L. & Olliver, M. (1942). *Biochem. J.* **36**, 155.

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Meal viscosity, gastric emptying and glucose absorption in the rat. By A. R. LEEDS*, N. R. BOLSTER, R. ANDREWS and A. S. TRUSWELL, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

The magnitude of postprandial hyperglycaemia in man after test meals containing absorbable and unabsorbable carbohydrates has been shown to be related to meal viscosity (Jenkins *et al.* 1978) but it is not known if increasing meal viscosity delays gastric emptying or slows movement of glucose to the absorptive mucosal surface or both.

In this study seventy-two hooded Lister rats (Queen Elizabeth College strain, thirty-six females, body-weight range 172–213 g, and thirty-six males, body-weight range 180–260 g) were investigated after 16 h fasts. A meal composed of 500 mg glucose, 16 mg phenol red, 80 mg of one of three guar gum preparations, and 4 ml water was given by orogastric intubation. The animals were killed humanely after 0·5 and 1 h (n 8 for each) 1·5 or 2 h (n 4 for each), and immediately afterwards the abdominal cavity was opened, the whole gut excised after clamping, and cut into parts, viz: stomach, upper, mid and lower thirds of the small gut, caecum and colon, which were diced into iced cold water and homogenized. After protein precipitation and centrifugation supernatants were analysed for glucose by a glucose oxidase method, and phenol red. Solutions of the three guar gum preparations had viscosity ranges at 37° as follows: purified guar gum (Meyprogat 150^R) 9400–79 400 cP, purified and depolymerized guar gum (Meyprogat 60^R and 7^R) 360–470 cP and 10–20 cP respectively.

Gastric emptying of phenol red and glucose was slowed by increasing meal viscosity; glucose half emptying time being 41±5·8 min (mean ± SEM) and 16±1·7 min after high and low viscosity meals ($P<0·001$, n 46). Glucose disappearance from the gut as a whole was significantly slower after the most viscous meal than after the least (glucose absorbed at 30 min: 30±5·1% of glucose in most viscous meal and 70±7·9% of glucose in least, $P<0·001$, n 15) and the rate of disappearance of glucose was dependent on the rate of gastric emptying. However, meal viscosity did not significantly affect disappearance of glucose from the small gut when this was expressed as a proportion of glucose emptied from the stomach.

These results show that in the rat increasing meal viscosity slowed glucose disappearance from the gut and this was due mainly to a slowing of gastric emptying. One possible mechanism of action for viscous forms of dietary fibre which modify postprandial glycaemia is thus indicated.

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Jenkins, D. J. A., Wolever, T. M. S., Leeds, A. R., Gassul, M. A., Wainsman, P., Dilawari, J., Goff, D. V., Metz, G. L. & Alberti, K. G. M. M. (1978). *Brit Med. J.* 1, 1392.

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