ABSTRACTS OF COMMUNICATIONS

The Two Hundred and Eighty-seventh Scientific Meeting of the Nutrition Society was held in the Physiology Lecture Theatre, Guy's Hospital Medical School, London SE1 9RT, on Thursday, 4 December 1975, at 11.00 hours, when the following papers were read:

Postnatal protein malnutrition: a major reduction in brain-cell size or cell number? By FRANK M. CREMIN* and SANFORD A. MILLER, *Department of Nutrition and Food Science, Massachusetts Institute of Technology, Cambridge, Mass., USA*

Eighty Sprague-Dawley rat pups were randomized and divided into ten litters of equal weight on day 1 of life. Two groups of five litters were nursed on dams given diets with either 250 g (control) or 80 g (experimental) casein/kg. The diets were isoenergetic and adequate in all nutrients except protein, which was deficient in the diet with 80 g casein/kg. At weaning (21 d) ten rat pups from control and experimental groups were weighed, killed and the weight, DNA, RNA and protein contents of the liver, kidney and brain were estimated.

In addition, twelve male weanlings from each group were subdivided in random fashion into two groups of equal weight. Thus, postweaning there were four groups of rats and these were either given the 250 g casein/kg or 80 g casein/kg diets that their dams had before weaning (groups 25–25 and 8–8 respectively) or given the alternative diet to that which their dams had before weaning (groups 25–8 and 8–25). At 65 d of age, the rats were weighed, killed and the weight, RNA, DNA and protein contents of the liver, kidney and brain were estimated.

The results confirm earlier observations in showing that: (1) protein malnutrition before weaning results in an irreversible reduction of body-weight and tissue weights, and of tissue DNA, RNA and protein; (2) rehabilitation postweaning on a high-protein diet results in some reversibility of the earlier retardation, but not to control levels, and (3) while protein malnutrition postweaning results in a retardation of growth rate and a reduction in tissue weight, DNA, RNA and protein content, this effect is less severe than that due to malnutrition applied before weaning.

A 3·3-fold reduction in total brain protein and in brain-cell size (total protein÷total DNA) was observed in group 8–8, while 2·3- and 1·4-fold reductions, respectively, were observed in these indices in groups 8–25 and 25–8. The results indicate that: (1) a major effect of preweaning protein malnutrition in rats is to almost completely retard the brain protein growth spurt that normally occurs postweaning; (2) malnutrition postweaning (group 25–8) results in a significant ($P<0·01$) but less severe retardation of brain cell hypertrophy and (3) the primary

*Present address: Department of Dairy and Food Chemistry, University College, Cork, Republic of Ireland.

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effect of malnutrition in reducing brain-cell size (total protein÷total DNA) is to cause a large reduction in total protein and a small reduction in total DNA. The slight reduction in brain-cell number (total DNA) obtained after preweaning protein malnourishment makes sense in view of the fact that the brain has completed most of its hyperplastic phase very early in the postnatal period. The reduction in total brain protein and in brain-cell size was not observed in 21-d-old rats as the brain protein growth spurt is a postweaning one, and therefore had not yet occurred.

The results presented suggest that the major effect of preweaning protein malnutrition in rat brain is to reduce brain-cell size by reducing total brain protein primarily.

Origin of amino acids in rumen bacteria. Studies using [^{15}N]urea. By K. DANESHVAR, D. N. SALTER and R. H. SMITH, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

A Friesian calf was given doses of [^{15}N]urea with isonitrogenous, isoenergetic feeds in which the nitrogen was supplied as: (A) decorticated groundnut meal (DCGM)–urea, 0·83:0·17; (B) DCGM–urea, 0·39:0·61; and (C) urea only. Amino acids and ammonia representing amide-N were separated from acid hydrolysates of the bacteria and ^{15}N determined by optical emission spectroscopy. Results, expressed as relative ^{15}N abundance (^{15}N isotopic abundance in amino acid (or amide)÷^{15}N isotopic abundance in total bacterial N) indicated the relative proportions of ^{15}N incorporated into different bacterial constituents.

For diet C, relative ^{15}N abundance increased for most amino acids (except methionine, tyrosine, phenylalanine and serine, for which there was little change) between 1 and 3 h after feeding (from 0·45–0·79 to 0·50–0·88), whereas the value for amide-N fell (from 1·41 to 1·13). It appeared that ^{15}N moved from a labile pool into the amino acids. Amide-N apparently formed at least part of this pool. After 3 h, values for most individual amino acids increased to only a small extent. Similar patterns of change with time after feeding were generally found after giving the other diets but there were marked absolute differences.

For diet A, mean values for samples taken 3–7 h after feeding were highest for amide-N (1·13) and aspartate (1·06), intermediate for alanine, isoleucine, valine, glutamate, lysine, leucine, glycine, threonine, serine, histidine, phenylalanine and tyrosine (ranging, in order, from 0·85 to 0·49) and lowest for proline (0·39) and arginine (0·20). Values for relative ^{15}N abundance for individual amino acids showed different patterns of change when different diets were compared. For example, for amide-N and aspartate they tended to decrease with increasing urea in the diet while the reverse was true for proline and arginine. The over-all effect of these changes was to make the differences between amino acids less marked on diet C (0·89–0·54) than on diet A, presumably because nearly all bacterial N compounds originated from urea with diet C.

The results indicated that varying proportions of the N of different bacterial amino acids were derived directly from dietary protein when this was given; in

particular, high proportions of the N of proline and arginine were obtained in this way. It is known that certain rumen bacteria in pure culture need the carbon skeletons of branched-chain amino acids in a preformed state (Bryant, 1973). The possibility that mixed rumen bacteria might satisfy such a need by directly incorporating such amino acids was not supported by the present evidence.

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Turnover of bacterial nucleic acids in the rumen. By R. C. SMITH* and R. H. SMITH, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Exogenous nucleic acids are rapidly degraded in the rumen so that most of the nucleic acids entering the ruminant duodenum are microbial (McAllan & Smith, 1973). Purine bases of microbial origin may be incorporated directly into the tissue nucleic acids of the host (Smith, Moussa & Hawkins, 1974). It is recognized that rumen bacteria turn over in the rumen, although to a poorly understood extent, so that some part of their nucleic acids would presumably be degraded there.

To investigate the extent of this degradation, mixed rumen bacteria, separated by centrifugation from the rumen contents of a young steer receiving a diet of flaked maize and hay (1:1, w/w) were incubated in a growth medium containing [U-^{14}C]adenine (Smith *et al.* 1974). A washed suspension of these bacteria (containing over 90% of their ^{14}C activity in the nucleic acid fraction) was incubated with: (A) a medium containing only salts and glucose, (B) the supernatant fluid from rumen contents centrifuged at 650 *g*, or (C) strained rumen contents. Samples were taken at intervals and separated by centrifuging into supernatant fluid and residue. Residues were extracted with dilute trichloroacetic acid (50 g/l) at 0° (cold TCA) and then at 100° (hot TCA). Radioactivity in the nucleic acid fractions (soluble in hot TCA) always decreased up to 6 h while that in the other fractions, particularly those in the supernatant fluid, showed corresponding increases. From 6 to 20 h, radioactivity soluble in hot TCA decreased further but was accompanied by a decrease in total radioactivity largely accountable as evolved CO_2. Average rates of decrease in radioactivity soluble in hot TCA up to 6 h (mean values (%/h)±SE for six experiments) were 10·6±1·5, 5·6±0·9 and 3·9±1·4 for cultures A, B and C respectively.

It appeared that degradation of bacterial nucleic acids, although less than in culture A, occurred at an appreciable rate in incubated rumen contents. If it is assumed that this rate was representative of that for the bacterial population in the rumen then it can be calculated, from known values for the size of the rumen nucleic acid pool and the rate at which nucleic acids pass into the duodenum, that more than 30% of the bacterial nucleic acids synthesized in the rumen of a steer given flaked maize and hay would be degraded in this way.

*On leave from the Department of Animal and Dairy Sciences, Auburn University, Auburn, Alabama, USA.

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Platelet aggregation in rats made hyperuricaemic with nucleic acid-rich diets containing oxonate, an inhibitor of uricase. By P. D. WINOCOUR, K. A. MUNDAY, T. G. TAYLOR and M. R. TURNER, *Department of Physiology and Biochemistry, The University, Southampton SO9 3TU*

The epidemiological association between gout and cardiovascular disease has long been known, but attempts to demonstrate that the effect results from the higher plasma uric acid concentration affecting platelet function have yielded equivocal results both with epidemiological studies and with experimental procedures involving either the injection of uric acid into animals, or in vitro work. Therefore, we have determined platelet aggregation in plasma from rats made hyperuricaemic by feeding nucleic acid-rich diets containing oxonate, a uricase (urate oxidase; *EC* 1.7.3.3) inhibitor; this experimental situation more nearly represents that found in gout than does the intravenous injection of uric acid.

Female Wistar rats weighing about 170 g were fed for 21 d on diets containing a *Fusarium* mould (Lord Rank Research Centre, High Wycombe) as the source of protein, fibre and nucleic acid. The composition of the diets was (g/kg): mould 400, sucrose 150, maize starch 300, maize oil 100, mineral mix 40, vitamin mix 10, and supplied 200 g protein and 35 g nucleic acid/kg. In the experimental diet 30 g oxonate/kg was included at the expense of maize starch. It has been shown in previous experiments that oxonate per se has no effect on platelet function.

Platelet aggregation was measured using a modification of the method of Born (1962), with ADP (2μM) or thrombin (0·4 U/ml) as aggregating agents. Results are expressed as the maximum rate of aggregation ($V_{A_{max}}$). Uric acid was determined in plasma and in the kidney using an AutoAnalyzer II (Technicon Instruments Corporation, 1971). In rats receiving the diet containing oxonate, plasma uric acid and $V_{A_{max}}$ were both significantly increased:

(Mean values with their standard errors for eight observations)

	Uric acid		$V_{A_{max}}$ (cm/min per 10^8 platelets)	
Addition to diet	Plasma (mg/l)	Kidney (mg/g)	ADP-induced	Thrombin-induced
None	19·5±1·7	0·33±0·01	5·3±0·3	6·2±0·4
Oxonate	35·6±4·3**	1·62±0·22***	7·1±0·6*	7·5±0·4*

Significance of differences: *$P<0·05$, **$P<0·01$, ***$P<0·001$.

There was a significant correlation between the plasma uric acid concentration and $V_{A_{max}}$ for ADP-induced (r 0·88, $P<0·001$) and for thrombin-induced aggregation (r 0·74, $P<0·01$).

In a second experiment, the time-course of the effect of the nucleic acid-rich diet containing oxonate on plasma uric acid and $V_{A_{max}}$ was examined by feeding rats

for 4, 14 or 21 d prior to investigation. The plasma uric acid concentration had increased 2·5-fold after 4 d of feeding but it was only after 14 d that there was any significant change in $V_{A_{max}}$, and that only for thrombin-induced aggregation. However, after 21 d of feeding, the $V_{A_{max}}$ was significantly increased for ADP-induced aggregation. Although there was a significant correlation between plasma uric acid and $V_{A_{max}}$ when the 21 d results were considered alone (ADP-induced aggregation, r 0·65, $P<0·001$; thrombin-induced aggregation, r 0·56, $P<0·05$), the correlation when results from animals fed for 4 or 14 d were also included was non-significant.

An association between plasma uric acid and platelet aggregation has been demonstrated when the plasma uric acid concentration was raised for 21 d. However, there appears to be a time-lag between the effect of the experimental diet on plasma uric acid and the increase in $V_{A_{max}}$ that merits further investigation.

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The effects of 1α-hydroxycholecalciferol on the calcium and phosphorus metabolism of dairy heifers. By B. F. SANSOM, M. J. VAGG and W. M. ALLEN (Introduced by W. LITTLE), *ARC Institute for Research on Animal Diseases, Compton, Newbury, Berks.*

1α-Hydroxycholecalciferol (1-HCC) is a synthetic analogue of 25-hydroxy- and 1α, 25-dihydroxycholecalciferol, the active metabolites of cholecalciferol (vitamin D_3). Because it already has a hydroxyl group in the 1α position it may be expected to stimulate absorption of calcium by the intestine and mobilization of bone Ca more rapidly than cholecalciferol itself, and thus may be of value for the treatment or prevention of milk fever in dairy cattle.

In order to establish its effects on the concentrations of Ca, phosphorus and magnesium in plasma, and on Ca and P balances, 1-HCC was administered to groups of four pregnant heifers at dose rates of 1 or 2·5 μg/kg body-weight, either intramuscularly, dissolved in sesame-seed oil, or intravenously, in propylene glycol. Four heifers were used as controls, and all the animals received a conventional diet containing approximately 42 g Ca and 28 g P/d.

After intramuscular administration, the concentrations of Ca and P in plasma increased more rapidly than after doses of cholecalciferol; the first response was observed after 24 h, the peak after 3–4 d and the increased concentrations were maintained with only a slow decline for at least 14 d. The larger dose produced a significantly greater effect than the smaller. After intravenous administration, increases in plasma Ca and P concentrations were apparent after only 12 h, similar peak concentrations were reached after 2–3 d and concentrations then declined slowly. These results are summarized in Table 1.

Table 1. *Mean increases in plasma calcium and phosphorus concentrations (mmol/l) of dairy heifers after intramuscular or intravenous administration of 1 or 2·5 μg 1α-hydroxycholecalciferol/kg body-weight*

	Intramuscular dose				Intravenous dose			
	Ca		P		Ca		P	
Time after dose (d)	1 μg	2·5 μg	1 μg	2·5 μg	1 μg	2·5 μg	1 μg	2·5 μg
0·5	0·045	0·02	—	—	0·10	0·29	0·14	0·36
1	0·11	0·08	—	—	0·22	0·36	0·45	0·64
2	0·16	0·32	0·03	0·34	0·47	0·78	0·68	0·64
3	0·205	0·61	0·67	0·78	0·51	0·62	0·76	0·91
4	0·32	0·42	0·59	0·83	0·32	0·60	0·77	0·89
5	0·27	0·53	0·78	0·97	0·33	0·51	0·67	0·79
7	0·30	0·57	0·75	0·80	0·27	0·44	0·59	0·78
10	0·24	0·30	0·61	0·94	0·19	0·32	0·39	0·50
13	0·045	0·23	0·49	1·00	0·11	0·19	0·34	0·71

The 1 μg/kg dose, by either route of administration, resulted in an approximately 10% reduction in food intake during the 14 d after administration, and the 2·5 μg/kg dose caused a 20% reduction; the largest depressions of appetite coincided with the maximum increases in plasma Ca and P concentrations, and with small reductions in plasma Mg concentrations which were maintained for a few days thereafter.

There were no other indications of toxicity. Both the Ca and P balances of the heifers receiving 2·5 μg 1-HCC/kg increased by approximately 2 g/d but there were no significant changes in the heifers receiving the lower dose. Such small changes appear unlikely to provide sufficient Ca and P to account for the observed increases in the concentrations of these minerals in plasma, and it may be presumed that the elements are also being actively mobilized from bone.

The rapidity of action of intravenously administered 1-HCC makes it potentially useful for the prevention or treatment of milk fever.

We thank Leo Laboratories Ltd (Copenhagen) for generously supplying the 1-HCC.

The influence of dietary sodium and fibre on mineral absorption in the small and large intestines of growing pigs. By I. G. PARTRIDGE, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

It has been reported that cereal and purified diets resulted in different patterns of intestinal sodium absorption (Partridge, 1975). The possibility that dietary fibre intake may influence mineral absorption by altering digesta volume was investigated. The response to reduced Na intake was also studied.

Seven pigs of 30 kg initial live weight, fitted with ileal re-entrant cannulas, were given diets containing starch, sucrose, maize oil, cellulose and casein, plus minerals and vitamins. Dietary cellulose and Na levels were: (A) 30 and 2·7, (B) 30 and 0·9, (C) 90 and 2·7, and (D) 90 and 0·9 g/kg respectively. Different feeding scales ensured equivalent intakes of other nutrients. Each animal received each of the

diets in turn. After adaptation to diet A or C, a 24 h collection of digesta and a 3 or 4 d collection of faeces commenced. At the end of the faeces collection an abrupt change from diet A to B or from C to D was made and a 48 h collection of digesta was started. Mean apparent absorption coefficients from analyses of ileal digesta and faeces, with their standard errors, were:

	Diet with 30 g cellulose/kg				Diet with 90 g cellulose/kg			
	Ileal digesta		Faeces		Ileal digesta		Faeces	
Diet with 2·7 g Na/kg	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Na	0·46	0·050	0·99	0·003	0·15	0·079	0·98	0·010
Calcium	0·43	0·020	0·74	0·016	0·44	0·080	0·63	0·097
Phosphorus	0·64	0·022	0·81	0·029	0·69	0·027	0·73	0·026
Potassium	0·90	0·037	0·96	0·006	0·88	0·025	0·86	0·041
Magnesium	−0·01	0·030	0·73	0·026	0·03	0·127	0·61	0·044
Diet with 0·9 g Na/kg, day 1								
Na	−0·56	0·111			−1·57	0·407		
Diet with 0·9 g Na/kg, day 2								
Na	−0·35	0·075			−1·27	0·242		

There was a greater volume of ileal digesta with the high-fibre diets and a concomitant reduction in Na net absorption in the small intestine, with no effect on other elements. In the large intestine Na absorption was enhanced while that of other elements was reduced by the high-fibre diet. Reduced Na intake caused net secretion of Na anterior to the cannula, this being more pronounced with the high-fibre treatment. Na concentrations in ileal digesta were similar for all treatments.

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Ascorbic acid concentrations in human plasma and cerebrospinal fluid.

By B. D. RIDGE and E. FAIRHURST, *Beecham Products Applied Research Department, Randalls Road, Leatherhead, Surrey,* and D. CHADWICK and E. H. REYNOLDS, *University Department of Neurology, Institute of Psychiatry, De Crespigny Park, London SE*5 8*AF*

Certain anticonvulsant drugs depress folic acid concentrations in the serum and cerebrospinal fluid (CSF) (Reynolds, Gallagher, Mattson, Bowers & Johnson, 1972). A collaborative study into possible drug-interference in ascorbic acid (AA) metabolism has given us the opportunity to determine AA in the plasma and CSF of a group of epileptic patients. This is of nutritional interest, especially in view of Hammerstrom's (1966) evidence that brain tissue AA derives from the CSF.

Eighteen epileptic subjects were being treated with drugs, and nine were untreated. Treated subjects received diphenylhydantoin, phenobarbitone and primidone, either alone or in combination.

No statistically significant difference was observed between the treated and untreated groups in respect of either plasma or CSF AA.

In the absence of a drug effect, the results were amalgamated into a single group in which the relationship between plasma and CSF AA concentrations can be represented by Fig. 1.

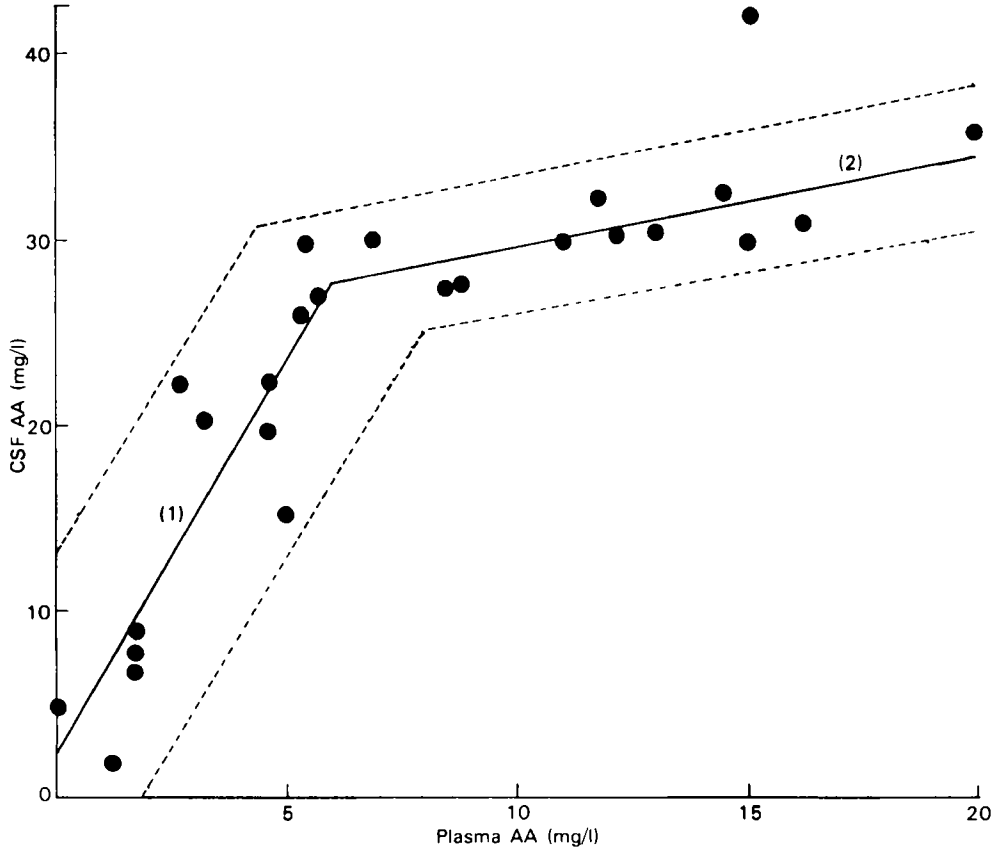

Fig. 1. Relationship of cerebrospinal fluid (CSF) ascorbic acid (AA) level to plasma AA level in twenty-five subjects being treated for epilepsy. (1), $y=0{\cdot}24+4{\cdot}22x$ (r 0·90); (2), $y=2{\cdot}47+0{\cdot}49x$ (r 0·84), where y is CSF AA and x is plasma AA; ·····—·—·—, 95% confidence limits.

In Fig. 1, the line of steep slope probably represents active transport of AA from plasma to CSF, and that of shallow slope an approach to saturation of this active process. The intersections of the 95% confidence limits show that, in order to ensure saturation of the active process, the plasma AA level must be >8 mg/l.

According to the classic Sheffield study (Bartley, Krebs & O'Brien, 1953) maintenance of this plasma level requires an intake of at least 70 mg AA/d.

The authors are grateful for the technical assistance of Miss Margery Williams, a Nutrition student in the Department of Biochemistry, University of Surrey.

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A futile energy cycle in adult rats given a low-protein diet at high levels of energy intake? By K. J. McCracken, *Agricultural and Food Chemistry Research Division, Department of Agriculture, Northern Ireland, and The Queen's University of Belfast, Belfast BT9 5PX*, and R. Gray, *Agricultural and Food Chemistry Research Division, Department of Agriculture, Northern Ireland, Belfast BT9 5PX*

Whereas the studies of McCracken (1975*a*, *b*) indicate that the energy expenditure of rats given low-protein diets at moderate levels of energy intake or normal-protein diets at very high levels of energy intake is explicable in terms of a strictly classical approach, there are a number of reports which suggest that low-protein diets in conjunction with excessively high levels of energy intake give rise to poor utilization of energy and increased heat production (Miller & Payne, 1962; Pond, Barnes, Bradfield, Kwong & Krook, 1965; Miller & Mumford, 1967; McCracken, 1975*a*). Because of difficulties of interpretation of the results in the growing rat owing to differences in carcass composition (McCracken, 1975*a*), and in order to obtain a more direct comparison with the results of Miller & Mumford (1967), an experiment has been conducted on adult female rats using the diet described by McCracken (1975*b*) (control diet) or a low-protein diet containing 25 g crude protein (nitrogen×6·25)/kg, obtained by replacing part of the casein by dextrin. Two groups of three female rats averaging 230 g body-weight were fasted for 24 h and heat production was then measured for a further 22 h in a closed-circuit respiration chamber. Group 1 was given the control diet and group 2 the low-protein diet for 6 d at a level of 175 kJ/rat per d by gastric intubation, and during the sixth day the heat production of both groups was determined. The energy intake was increased over a 4 d period to 350 kJ/rat per d. Heat production was measured on the first day at this level and at intervals over the next 4 months. There was no difference in the initial fasting heat production of the two groups, or in daily heat production on the lower plane of intake (159 and 158 kJ/rat per d) but the heat production of group 2 rats increased to 218 kJ/rat per d on the first day at the higher level of intake compared to 183 kJ/rat per d for the animals in group 1. This corresponds to an efficiency of utilization of the extra energy intake of only 0·66 on the low-protein diet compared to 0·86 on the normal diet. The difference in fed heat production of the two groups when compared on a body-weight basis was maintained throughout the experiment.

Energy retention of the rats in group 1 was 160 kJ/rat per d at the beginning of the experiment and decreased to 100 kJ/rat per d after 4 months, whereas that of the rats in group 2 was 130 kJ/rat per d, falling to 70 kJ/rat per d.

Possible mechanisms whereby the extra energy expenditure arises under conditions of protein inadequacy will be discussed and the relevance of these results to studies on the overfed adult human will be considered.

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The influence of plane of nutrition and environmental temperature on heat loss and energy retention in the pig. By W. H. CLOSE and L. E. MOUNT, *ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

Thirty-eight series of calorimetric determinations, each of 2 weeks' duration and each involving one animal on a given plane of nutrition and at a given environmental temperature, were carried out on Large White pigs with initial body-weight of 21·7–38·2 kg, and final body-weight 24·2–50·0 kg. Before its entry into the calorimeter, each animal was habituated to the conditions of measurement for a minimum period of 14 d. The planes used were approximately one, two and three times maintenance food requirement, or *ad lib.*, at 10, 15, 20, 25 or 30°; at 30° the three times maintenance plane represented the *ad lib.* regimen, since voluntary food intake was reduced at that temperature. Two animals were exposed singly to each combination of plane of nutrition and environmental temperature. The calorimeters and experimental arrangements were those described by Close & Mount (1975). In addition to the continuous measurement of heat loss (H) during

Table 1. *Mean values for metabolizable energy* (ME) *intake, heat loss* (*H*), *energy retention* (*ER*), *protein* (*P*) *and fat* (*F*) *deposition* (*kJ/kg body-weight*$^{0.75}$ *per d*), *and mean body-weights, for pigs on different planes of nutrition and at different environmental temperatures*

Environmental temperature(°)	Plane of nutrition*	ME intake	H	ER	P	F	Mean body-wt (kg)
10	1M	571	593	−22	14	−36	31·0
	2M	976	760	216	100	116	35·1
	3M	1446	786	660	170	490	41·0
	ad lib.	1965	1009	956	249	707	34·6
15	1M	504	604	−100	28	−128	29·9
	2M	893	671	222	92	130	24·8
	3M	1476	763	713	173	540	35·6
	ad lib.	1591	853	738	178	560	38·6
20	1M	495	469	26	22	4	27·3
	2M	943	601	342	111	232	30·4
	3M	1412	791	621	154	467	31·8
	ad lib.	1655	863	792	196	596	37·3
25	1M	455	425	30	32	−2	30·2
	2M	941	588	353	114	239	30·4
	3M	1494	763	731	177	554	34·6
	ad lib.	1406	714	692	154	538	38·0
30	1M	455	484	−29	43	−72	30·2
	2M	928	637	291	103	188	29·1
	3M	1202	765	437	125	312	32·4

*Pigs were given one (1M), two (2M) or three (3M) times maintenance requirement, or fed *ad lib.* At 30° 3M was equal to or above *ad lib.* intake.

consecutive 24 h periods, weekly energy balances were determined. This allowed the estimation of total energy retention (ER) as the difference between metabolizable energy (ME) intake and H. The nitrogen balance was determined by Kjeldhal estimations on food, faeces and urine, with N lost to the air as ammonia estimated from continuous sampling through acid of the inlet and exhaust air of the calorimeter. Protein deposition in the animal's tissues (P) was determined from the N balance, and it was assumed that the difference between total ER and P gave the fat deposition.

The mean results of the measurements are given in Table 1. Mean H was at a minimum and mean ER was at a maximum at 25° on all planes of nutrition; 30° was in the hyperthermic zone on all planes of nutrition. The relation between ME, H and ER will be discussed.

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Close, W. H. & Mount, L. E. (1975). *Br. J. Nutr.* **34**, 279.

The effect of sodium chloride and sodium bicarbonate on food intake, growth rate and acid–base balance in calves. By R. C. KELLAWAY, *Dairy Research Unit, University of Sydney, Camden, New South Wales 2570, Australia,* and D. J. THOMSON and D. E. BEEVER, *The Grassland Research Institute, Hurley, Maidenhead, Berks. SL6 5LR*

Calves given cereal-based diets containing 20 g sodium bicarbonate and 20 g sodium chloride/kg ate considerably more than calves given pellets without the buffer salts (Kellaway, Grant & Chudleigh, 1973). Apart from their effect on rumen pH and buffering capacity, it is likely that these salts would have increased rumen osmolality. Increases in rumen osmolality were associated with an increased flow of α-linked glucose polymers and amino acids into the small intestine of sheep (Harrison, Beever, Thomson & Osbourn, 1975). The work reported here was designed to differentiate between osmotic and buffering effects on food intake.

NaCl or $NaHCO_3$ was incorporated into two basal diets at levels of 2, 11, 20 and 29 g Na/kg dry matter (DM). The first basal diet comprised ground barley, meat-and-bone meal, molasses, urea, trace minerals and vitamins; in the second basal

Table 1. *Food-pellet intake and growth rate of calves given dietary supplements of sodium as the chloride or bicarbonate*

	Na (g/kg diet DM)				SE of mean
Pellet intake (g/kg body-wt $^{0.75}$ per d)	2	11	20	29	
NaCl	63·9	74·0	67·0	70·4	3·6
$NaHCO_3$		75·6	86·0	88·2	
Growth rate (g/d)					
NaCl	633	721	626	688	66·6
$NaHCO_3$		833	910	852	

DM, dry matter.

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diet 40% of the barley was replaced by grass meal. Diets were pelleted and fed with chopped straw, *ad lib.*, to forty-eight calves in individual pens for 4 weeks before and 5 weeks after weaning, which was at 5 weeks of age. Animal responses are given only in terms of source and level of Na, because the grass-meal treatment did not interact significantly with these treatments.

During the 5-week period post weaning, the pellet intake and growth rate of calves receiving NaCl were not significantly affected by level of NaCl inclusion in the diet. With calves receiving $NaHCO_3$ there were significant linear increases in pellet intake, pellet and straw intake and growth rate with levels up to 20 g Na/kg DM (Table 1).

Food intake and growth rate were increased with $NaHCO_3$ but not with NaCl, which suggests that beneficial responses to dietary inclusions of buffer salts are attributable to their buffering capacity rather than to their osmotic activity. The optimum level of inclusion appears to be about 66 g $NaHCO_3$/kg DM (18 g Na/kg DM). Measurements of acid–base balance indicated that elevated levels of base excess associated with this rate of $NaHCO_3$ inclusion were compensated by changes in partial pressure of dissolved CO_2 which maintained blood pH within the normal range.

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Success and failure of breast-feeding in relation to energy intake. By MARGARET J. WHICHELOW, *Unit for Metabolic Medicine, Department of Medicine, Guy's Hospital, London SE1 9RT*

It has been shown that lactating women have much higher daily energy intakes than bottle-feeding mothers (Thomson, Hytten & Billewicz, 1970; Naismith & Ritchie, 1975). Various authorities recommend an increase in daily energy intake during lactation, ranging from 2·09 MJ (500 kcal) (Department of Health and Social Security, 1969) to 4·18 MJ (1000 kcal) (FAO, 1957). However, none of over 100 mothers questioned were aware of any extra energy requirement for lactation. As insufficient milk supply is a commonly reported cause of failure of lactation, a study was carried out to determine whether there was any relationship between energy intake and adequacy of milk supply.

The daily energy intakes of twenty-six healthy breast-feeding mothers, with babies aged from 4 to 40 weeks, were compared with their normal, non-lactating intakes.

Of the twenty-one successfully lactating mothers, eight were losing weight, one was gaining weight and in the remaining twelve weight was steady. There were five mothers with an inadequate milk supply, three of whom were losing weight:

one was gaining weight and one was maintaining a steady weight. The daily energy intakes were:

(Mean values; no. of mothers in parentheses)

	Intake (MJ (kcal)/d)			Significance of increase (paired *t* test)
	Lactating	Non-lactating	Increase	
Successful breast-feeders				
Weight steady (12)	12·33 (2946)	8·38 (2002)	3·95 (944)	$P<0·001$
Losing weight (8)	10·50 (2509)	8·03 (1920)	2·46 (589)	$P<0·001$
Unsuccessful breast-feeders (5)	8·20 (1959)	7·07 (1688)	1·35 (271)	$P<0·025$

All the successful mothers were eating considerably more than normal, with those whose weight was steady or increasing consuming the most. The unsuccessful mothers were eating very little more than normal.

A close inverse correlation was observed in the successful mothers, between the rate of change of body-weight and the extra energy intake (r −0·766, $P<0·001$). This suggests that although in many women body-fat can be mobilized to subsidize milk production, the rate of mobilization is limited, as evidenced by the unsuccessful group and by three successful mothers who when trying to diet found an immediate reduction in milk supply.

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Long-chain polyunsaturated fatty acids in the erythrocyte lipids of breast-fed and bottle-fed infants. By T. A. B. SANDERS, *Department of Pathology, Kingston Hospital, Kingston, Surrey KT2 7BD,* and D. J. NAISMITH, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

It has been postulated that the long-chain polyunsaturated (LCP) fatty acids in human milk may play an important role in meeting the lipid requirements of the human infant (Crawford, Sinclair, Msuya & Munhambo, 1973). It was suggested that the production of LCP fatty acids, in particular arachidonic (20:4ω6) and docosahexenoic (22:6ω3) acids from the parent short-chain polyunsaturated fatty acids, linoleic (18:2ω6) and linolenic (18:3ω3) respectively, may be limited by the rate of desaturation. As 20:4ω6 and 22:6ω3 acids are found in substantial quantities in the grey matter of the brain it has been argued that any limitation in the supply of these fatty acids might impair the development of the nervous system (Crawford & Sinclair, 1972); 20:4ω6 and 22:6ω3 acids occur in considerably greater amounts in human milk than in cow's milk (Crawford *et al.* 1973). The lipids of the erythrocyte contain a high proportion of LCP fatty acids (Dodge & Phillips, 1967) and, owing to their relatively rapid rate of turnover, would be a

good indicator of tissue LCP fatty acid status. We decided therefore to look for differences in the LCP fatty acid composition of the erythrocyte lipids between four infants who had been wholly breast-fed and eight infants who were bottle-fed on a cow's milk formula.

The infant subjects of the study were those described by Naismith, Deeprose & Ma (1976). The results of the analyses are shown in Table 1. The proportion of 22:6ω3 was significantly lower, and that of docosapentaenoate (22:5ω3) and eicosatrienoate (20:3ω9) was significantly higher in the bottle-fed infants than in the breast-fed infants. The distribution of fatty acids in the linoleic (ω6) series was similar for both groups, 22:4ω6 always exceeding 22:5ω6. The triene : tetraene ratio (20:3ω9/20:4ω6) did not approach 0·4, the value which is taken to indicate essential fatty acid deficiency (Mohrhauer & Holman, 1963), in any of the infants. The distribution of LCP fatty acids in the linolenic (ω3) acid series differed markedly between the two groups. The ratio 22:5ω3/22:6ω3 was 0·70±0·23 (mean±SD) in the bottle-fed group and 0·22±0·03 in the breast-fed group (difference significant; $P<0.01$).

These results confirm the findings of Naismith *et al.* (1976) that an infant milk containing cow's milk fat appears to satisfy the requirements for fatty acids of the linoleic acid (ω6) series. These results also suggest that the ability to insert a double bond between the 18th and 19th carbon atoms from the terminal methyl group of the fatty acid may be the rate-limiting step in the formation of 22:6ω3 and possibly 22:5ω3. The lower proportion of 22:6ω3 in the bottle-fed infants does not appear to affect their rate of growth or their general health.

Table 1. *The long-chain polyunsaturated (LCP) fatty acid composition (mmol/mol LCP fatty acids) of the erythrocyte lipids of four breast-fed and eight bottle-fed infants*

(Mean values and standard deviations)

	LCP fatty acids							
	20:3ω9	20:3ω6	20:4ω6	22:4ω6	22:5ω6	20:5ω3	22:5ω3	22:6ω3
Breast-fed								
Mean	8·8	35·0	537·6	79·3	8·3	35·0	53·5	243·0
SD	4·5	9·6	31·1	34·2	8·1	11·6	13·4	26·4
Bottle-fed								
Mean	43·5	48·6	525·5	55·6	12·8	53·8	104·8	154·1
SD	4·7	13·2	42·0	14·4	10·6	21·4	32·3	17·3
Significance of difference	$P<0.001$	NS	NS	NS	NS	NS	$P<0.02$	$P<0.001$

NS, not significant.

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The linoleic acid requirement of the human infant. By D. J. NAISMITH, SUSAN P. DEEPROSE and M. C. F. MA, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

From the experiments of Hansen and his colleagues (Hansen, Haggard, Boelsche, Adam & Wiese, 1958) it is generally accepted that the minimum requirement for linoleic acid in the diet of the human infant is 1% of the total energy. The optimum intake is taken to be 4%, the amount found in the breast milk of mothers eating an average American diet. Infant milks containing butterfat fail to meet the minimum requirement, yet clinical signs of linoleic acid deficiency in artificially-fed infants have not been reported, and biochemical evidence is conflicting (Woodruff, Bailey, Davis, Rogers & Coniglio, 1964; Pikaar & Fernandes, 1966).

We have studied eleven infants fed exclusively on a cow's milk formula, and five wholly breast-fed infants, for 14 weeks. Body-weights and lengths were measured at 2 week intervals, and records were made of the food intakes of the bottle-fed infants. Fasting blood samples were taken at the end of the study for analysis of the total plasma lipids by gas–liquid chromatography.

Linoleic acid deficiency is characterized by an increase in voluntary food intake, indicating impaired utilization of energy. The mean food intake of our bottle-fed infants declined from 477 kJ (114 kcal)/kg per d at 2 weeks to 422 kJ (101 kcal)/kg per d at 14 weeks. Results of the lipid analyses are shown in Table 1.

Table 1. *Levels of the major fatty acids (mg/g total plasma lipid fatty acids) in breast-fed and bottle-fed infants*

	14:0	16:0	16:1	18:0	18:1	18:2ω6	18:3ω3	20:3ω9	20:4ω6
Breast-fed									
Mean	34	242	42	91	299	166	12	1·7	63·2
SE	4·7	10·7	6·7	3·7	20·1	13·1	3·9	0·2	10·8
Bottle-fed									
Mean	42	279	56	82	341	99	11	7·1	32
SE	5·0	5·6	5·9	4·9	7·0	2·8	1·4	0·7	2·2

The proportions of linoleic (18:2ω6) and arachidonic (20:4ω6) acids in the plasma lipids were significantly lower ($P<0·02$), whereas eicosatrienoic (20:3ω9) acid was significantly raised ($P<0·01$) in the artificially-fed infants. In none, however, did the triene:tetraene ratio exceed 0·4, the value taken to represent the upper limit of normality (Holman, 1960). The mean ratio for breast-fed infants was 0·03, and for bottle-fed infants, 0·22.

We conclude that infants fed solely on a cow's milk formula over a realistic period of time before weaning are not at risk from linoleic acid deficiency.

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The abnormal metabolism of essential fatty acids in the cat. By J. P. W. RIVERS, A. J. SINCLAIR, D. P. MOORE and M. A. CRAWFORD, *Nuffield Institute of Comparative Medicine, Zoological Society of London, Regent's Park, London NW1 4RY*

Linoleic acid (18:2ω6) and linolenic acid (18:3ω3) are usually thought of as the dietary essential fatty acids (EFA), although it is their chain-elongation and desaturation products that appear to be physiologically essential. This group of derived EFA (d-EFA) are of importance as prostaglandin precursors and as components of membrane phospholipids. The metabolism of linoleic and linolenic acids to d-EFA does not always proceed optimally, the desaturation step in particular having been shown to be rate-limiting (Hassam, Sinclair & Crawford, 1975).

Dietary studies on the domestic cat suggest that this species is unable to desaturate polyunsaturated fatty acids, and therefore exhibits a specific dietary requirement for the d-EFA (Rivers, Sinclair & Crawford, 1975).

Cats were given one of three semi-purified experimental diets differing only in the type of lipid added. These were SBOL (a 5:1(w/w) mixture of soya-bean oil and linseed oil), SSO (safflower-seed oil) and HCO (hydrogenated coconut oil). A control group was maintained on the mixture of proprietary cat foods used to maintain our cat breeding colony. Blood lipids of these animals examined after an average period of 15 months on the diet showed major modification of the fatty acid composition of all lipid classes by the type of diet given. The table illustrates this effect for plasma choline phosphoglycerides (CPG) (mean values; no. of animals in parentheses):

	Fatty acid composition (mg/g total fatty acid)					
	Diet			Plasma CPG		
Diet	18:2ω6	18:3ω3	d-EFA*	18:2ω6	18:3ω3	d-EFA*
Control (4)	72	9	38	118	3	292
SBOL (5)	408	168	0	456	26	31
SSO (2)	636	9	0	482	5	58
HCO (2)	2	0	0	136	6	21

*Chain-elongation and desaturation products of 18:2ω6 and 18:3ω3.

It can be seen that cats given experimental diets had very low levels of d-EFA. Such d-EFA as were present were chiefly chain-elongation rather than desaturation products. The results are compatible with an absence of Δ8 and Δ6 desaturation activity in the cat. Subsequent refeeding experiments demonstrated that Δ5 desaturation was also absent. The Δ9-desaturase, however, appeared to be

unimpaired. The results obtained on blood lipids were substantiated by observations on other tissues where parallel, although less extreme, changes in fatty acid composition occurred.

Animals given the HCO diet received virtually no EFA for 12 months. They had no 5, 8, 11-eicosatrienoic acid (20:3ω9) in any tissue lipid and this substantiates the idea of a desaturase deficiency.

All animals showed many of the clinical signs normally associated with EFA deficiency. However, the animals given the HCO diet were more severely affected, raising the possibility that linoleic and linolenic acids themselves may have a discrete functional role. Refeeding experiments have shown the condition to be reversible, and preliminary experiments with lipid concentrates support the idea that remission was due to a dietary supply of d-EFA.

These results suggest that the cat requires a source of animal lipid for optimal health. Their implication for human nutrition will be discussed.

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The absence of Δ6-desaturase activity in the cat. By J. P. W. RIVERS, A. G. HASSAM and CATHERINE ALDERSON, *Nuffield Institute of Comparative Medicine, Zoological Society of London, Regent's Park, London NW1 4RY*

Domestic cats given diets based on vegetable oils show changes in the fatty acid composition of tissue lipids compatible with an absence of Δ8-, Δ6- and Δ5-desaturase activity (Rivers, Sinclair & Crawford, 1975; Rivers, Sinclair, Moore & Crawford, 1976). In order to eliminate the possibility that desaturase activity was suppressed by the experimental diet, we have examined desaturation in animals given the proprietary meat- and fish-based cat-foods used for maintaining our breeding cat colony.

Two male cats, each weighing approximately 3·5 kg, were given an intraperitoneal injection containing 35 μCi [1-^{14}C]linoleic acid (206 μCi/mg fatty acid, 98% pure; The Radiochemical Centre, Amersham) in 0·5 ml olive oil carrier. Sequential blood samples showed that although blood lipids rapidly became labelled, the peak activities were low. Plasma lipids reached a maximum specific activity of 1000 ^{14}C disintegrations/min per mg lipid in less than 4 h after dosing, and erythrocyte lipids a maximum of 100 disintegrations/min per mg lipid 8 h after dosing.

Radioactivity was mostly associated with plasma phospholipid (PL) and cholesterol ester (CE) fractions. Initially it was evenly distributed between PL and CE, but as fatty acid turnover in PL was 25% faster, CE became progressively the more important fraction.

After 168 h animals were killed and tissues quickly removed and analysed by standard techniques (Sinclair, 1975). Lipids from most tissues contained about 40 ^{14}C disintegrations/min per mg lipid. However, there was very little incorporation in the brain (5 disintegrations/min per mg lipid) and much higher activity in the liver (100 disintegrations/min per mg lipid). In total, less than half of the administered dose was present as lipid, and only 0·79% as liver lipid.

Activity within the liver lipids was evenly distributed between PL and triglyceride fractions. Fatty acid analysis showed that most of the small amount of ^{14}C in both fractions was present as [1-^{14}C]linoleic acid: 91–94% in triglyceride, 73–87% in PL. Comparable values were obtained for plasma PL. A maximum of 7·5–16·7% of the ^{14}C present in PL fatty acids was associated with the metabolic products of linoleic acid. This was, however, a very small amount of radioactivity (10 disintegrations/min per mg fatty acid) and an insignificant proportion (0·005%) of the initial dose. It could be accounted for by the incorporation of [^{14}C]acetate in the chain elongation of dietary fatty acids, and is a marked contrast to comparable observations in laboratory rodents. We conclude that no $\Delta 6$ desaturation occurs in the cat.

These observations lend support to the contention that the cat cannot desaturate linoleic acid and is likely to require dietary sources of its desaturation products, particularly the prostaglandin precursors dihomo-γ-linolenic acid (20:3ω6) and arachidonic acid (20:4ω6).

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Changes in plasma lipids of man after substituting glucose-syrup solids for table sucrose. By R. BAGLEY, *Beecham Products Research Department, Slough,* M. FORD, *Beecham Products Research Department, Coleford,* and L. F. GREEN, *Beecham Products Research Department, Leatherhead*

Most dietary sucrose substitution studies have been short-term (e.g. Rifkind, Lawson & Gale, 1966; Macdonald, 1968; Dunnigan, Fyfe, McKiddie & Crosbie, 1970). The study reported here extended over 2 years.

Body-weight, fasting blood glucose and plasma lipids were recorded every 4 weeks for 1 year on eighteen male factory and laboratory workers, aged 31–62 (mean 45 years) whose table sucrose consumption was at least 38% (mean 59%) of their total sucrose intake (mean 138 g/d). It was then substituted by saccharin-sweetened glucose-syrup solids, physically indistinguishable from, and isoenergetic by volume with sucrose. Recording continued for another year. Body-weights were reasonably constant throughout.

The mean fasting plasma cholesterol concentrations (mmol/l) of eighteen men over the full 52 weeks, and also over the last 28 weeks of each treatment, were:

	Over 52 weeks		Over the last 28 weeks	
Table sweetener	Cholesterol	*n*	Cholesterol	*n*
Sucrose	6·314	222	6·309	123
Glucose-syrup solids	6·011	224	5·812	122
Reduction	0·303	---	0·497	—

As comparisons between the second half of each treatment period should avoid any carry-over effect of earlier diet- or subject-adjustment to the technique, the mean fasting values obtained over the final 28 weeks have been compared:

	Glucose		Triglycerides		Cholesterol		Phospholipid-phosphorus	
Table sweetener	mmol/l	*n*	mg/l	*n*	mmol/l	*n*	mmol/l	*n*
Sucrose	4·38	123	1190	123	6·309	123	3·78	123
Glucose-syrup solids	4·44	121	1210	121	5·807	122	3·29	122
Difference	+0·06		+20		−0·497		−0·49	

Reductions in mean fasting cholesterol and phospholipid-phosphorus concentrations were highly significant ($P<0.001$) when glucose-syrup solids replaced table sucrose, but glucose and triglyceride concentrations were unchanged.

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Some immediate metabolic responses in man to fructose ingestion. By I. Macdonald and Deborah Pacy, *Department of Physiology, Guy's Hospital Medical School, London SE1 9RT*

There is evidence that in men an increase in the fructose content of the diet can lead to an increase in the level of triglyceride in the fasting serum (Macdonald, 1973). It was therefore considered to be of interest to study the more immediate metabolic effects following fructose ingestion and to learn whether these were dose-dependent.

Eight men aged 18–21 years were given by mouth, after an overnight fast, 0·25, 0·5, 0·75 or 1·0 g fructose/kg body-weight, made up with water to 4 ml/kg body-weight. Venous blood was obtained at 0, 15, 30, 60 and 90 min after swallowing the fructose solution, and fructose (Bergmeyer, Bernt, Schmidt & Stork, 1970), glucose (Werner, Rey & Wielinger, 1970), triglyceride (Eggstein & Kreutz, 1966), pyruvate (Tfelt-Hansen & Siggaard-Anderson, 1971), lactate (Hohorst, 1970), glycerol (Eggstein & Kreutz, 1966), uric acid (Praetorius &

Poulsen, 1953), and insulin (Hales & Randle, 1963) determinations in the serum were carried out.

It was found that the fructose, pyruvate, lactate, glycerol and uric acid levels in the serum were dose-related. The serum glucose, insulin and triglyceride levels following the fructose load did not seem to be related to the amount of fructose consumed.

With the exception of the triglyceride and glycerol levels, which fell significantly, all the variables measured showed a significant increase after the ingestion of fructose.

We are grateful to the volunteers in this experiment.

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Some effects, in baboons, of chronic glycerol ingestion. By I. MACDONALD and ANNE KEYSER, *Department of Physiology, Guy's Hospital Medical School, London SE1 9RT*

Following the ingestion of glycerol there is an increase in serum triglyceride concentration both immediately after ingestion and in the fasting level during several weeks of adding glycerol to a free-choice diet. These effects are more marked in men than in pre-menopausal women (Macdonald, 1970). As a high-sucrose diet is also associated with an increased level of triglyceride in fasting serum it was decided to learn whether there is any synergistic effect between dietary sucrose and glycerol. Six mature male and six mature female baboons were given: (1) stock diet and 40 g glycerol/l in the drinking-water; (2) a formula diet of (parts by wt) 75 sucrose, 18 calcium caseinate, 5 dried yeast, 2 mineral mixture, 2 glycerol, in water; (3) diet 2 with no added glycerol.

Each diet was given for 10 weeks, fasting blood was taken at weekly intervals and the serum cholesterol (Levine, Morgenstern & Vlastelica, 1967) and triglyceride (Eggstein & Kreutz, 1966) concentrations estimated.

The results show that a diet high in sucrose with added glycerol causes increased levels of serum cholesterol and triglyceride in male baboons when compared with the stock diet with added glycerol or the high-sucrose diet. In the female animals no difference in response between the various diets was found. It thus seems that only in the male animals does a synergism exist between dietary sucrose and glycerol.

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Cholesterol-lowering effect of lignin in rats. By P. A. JUDD, R. M. KAY and A. S. TRUSWELL, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

One of the types of dietary fibre, lignin, was reported by Thiffault, Belanger & Pauliot (1970) to lower plasma cholesterol in patients with primary hypercholesterolaemia, an effect which was not confirmed by Lindner & Möller (1973). We have not been able to find any report of work on lignin and plasma lipids in experimental animals.

Twelve litter-mate pairs of male, weanling Sprague-Dawley rats were fed *ad lib.* on diets containing either 30 g lignin or 30 g cellulose/kg, this being thoroughly mixed into a basal diet containing (g/kg): maize starch 430, casein ('Casumen') 200, groundnut oil 150, sucrose 100, cellulose 30, and vitamins and minerals. The lignin used was 'Indulin ATR-S' (Westvaco, USA); the cellulose was 'Solka floc'. After 21 d the animals were anaesthetized and heart blood was taken by syringe.

Serum cholesterol, as determined by the method of Abell, Levy, Brodie & Kendall (1952), was significantly lower ($P<0.01$) in the twelve rats given lignin; serum triglycerides (not fasting), as determined by the method of Eggstein (1966), were not significantly lowered:

	Lignin diet		Control diet	
	Mean	Range	Mean	Range
Serum cholesterol (mmol/l)	1·86	1·61–2·28	2·18	1·68–2·64
Serum triglycerides (mmol/l)	0·81	0·60–1·56	0·95	0·60–1·58

Analysis of pooled faeces from each group showed faecal fat 15% higher, neutral steroids 14% higher and bile acids, as determined by the method of Evrard & Janssen (1968), not appreciably different (judged by the main gas–liquid-chromatographic peaks) in the lignin group.

Further work is needed on the mechanism. Lignin binds bile acids in vitro (Eastwood & Hamilton, 1968) and has been used to treat the diarrhoea that follows ileal resection (Eastwood, Mitchell, Findlay & McCormick, 1973). The lignin we used was the same as that used by Thiffault *et al.* (1970); it is prepared from pine wood. We have been unable to trace the source of that used by Lindner & Möller (1973). There are many different lignins in plants (Brauns & Brauns, 1959). Studies are needed on the lignins in foods.

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A prospective study of modified-fat and low-carbohydrate dietary advice in the treatment of maturity-onset diabetes. By J. I. MANN, T. D. R. HOCKADAY, J. M. HOCKADAY and R. C. TURNER, *The Radcliffe Infirmary and Department of Social and Community Medicine, University of Oxford, Oxford*

A diet low in carbohydrate is usually recommended to diabetic patients in 'Westernized' countries. However, Japanese, Trappist communities and Yemenite Jews usually consume diets high in carbohydrate and no change in this pattern is recommended for diabetic patients in these communities. In these groups, serum lipid levels tend to be lower than in 'Westernized' countries and ischaemic heart disease is less frequent in both diabetics and the general population.

A prospective study of two dietary regimens has begun in newly diagnosed diabetics to determine their effect on some circulating metabolites and on diabetic complications. Patients have been randomly allocated to receive one of two types of dietary advice. One regimen is a classical low-carbohydrate diet, while the other is aimed at restriction of cholesterol, reduction of total fat and an increase in the proportion of polyunsaturated fatty acids. The recommended energy content is determined from the excess above desirable body-weight.

Table 1. *Plasma cholesterol and triglyceride levels in diabetic patients given either a modified-fat or a low-carbohydrate diet*

(Mean values with their standard errors)

	Modified-fat						Low-carbohydrate					
	Initial		1 month		12 months		Initial		1 month		12 months	
Cholesterol (mmol/l)	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Men	5·15	0·24	4·01	0·20	4·20	0·19	5·21	0·25	4·8	0·19	4·69	0·18
Women	5·18	0·30	4·45	0·24	4·84	0·25	5·31	0·24	5·41	0·24	5·52	0·30
Triglyceride (mg/l)												
Men	1290	104	1200	143	1430	165	1510	141	1080	77	1260	110
Women	1280	115	1210	121	1340	111	1610	206	1300	125	1490	163

Instruction in the modified-fat diet was followed after 1 month by a significant fall in plasma cholesterol in men, but the fall observed in women was not statistically significant. Instruction in a low-carbohydrate diet caused a significant fall in triglyceride in both sexes but little change in cholesterol. After 12 months only the fall in cholesterol observed in men on the modified-fat diet remained statistically significant. The results for the first seventy-seven patients entered are given in Table 1. Both diets produced a significant and sustained decrease in fasting plasma glucose in both sexes.

The relative importance of cholesterol, triglyceride and hyperglycaemia as risk factors in the development of ischaemic heart disease in diabetics is uncertain and can be ascertained only through a long-term prospective trial.

Nutritional knowledge and food habits of families with children attending a state nursery school in Sheffield. By JANE WALSH and R. C. OSNER, *Department of Hotel and Institutional Management, Sheffield Polytechnic, Pond Street, Sheffield S1 1WB*

The work reported here is part of a wider research programme concerned with the formation of food habits and the nutritional importance of the free school meal.

During a study of preschool eating habits, a survey (33% response rate) was carried out on mothers with children at a state nursery school in Sheffield with a catchment area consisting of pre- and postwar council estates. Half the children in the survey dined at a school where 50% were given free school meals. Although 60% of mothers considered that this was the main meal of the day, only 38% bothered to read the menu list.

The nutritional quality of the children's diets was assessed by asking mothers how many times/d foods from the following categories were served (as an average over 1 week): potatoes, bread, meat, fish, cheese, eggs, milk, margarine, butter, fresh fruit, vegetables, salads, cakes, biscuits, sweets, 'fizzy' drinks. Daily consumption of two servings of protein-rich foods (meat, fish, cheese, eggs), three servings of milk and three servings of fresh fruit or vegetables was taken (arbitrarily) to indicate that the child received a good diet and was well-nourished, with under-nourished children receiving poor diets being defined as those consuming one or no serving of each of these categories. The differences between what parents believed (self-accredited) and the actual quality of the diet are shown in Table 1.

Table 1. *Actual and self-accredited nutritional quality of diets of children at a Sheffield nursery school* (%)

Quality of diet ...	Good	Average	Poor
Self-accredited			
Total	75	25	0
Having school lunch	60	40	0
Not having school lunch	90	10	0
Actual			
Total	50	45	5

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The nutritional knowledge of the mothers was established by the use of thirty check questions in which the mothers were asked to recognize the correct foods in certain categories: body-building (protein), protective, energy-giving, fattening, harmful to teeth, 'having no goodness'. There were fifteen correct and fifteen incorrect foods in each category, thus giving scores ranging from −15 to +15 (Table 2). It was found that 55% of mothers knew less and 25% knew more than they thought they knew about nutrition.

Table 2. *Actual and self-accredited nutritional knowledge of the mothers of children at a Sheffield nursery school* (%)

Nutritional knowledge . . .	Good	Fairly good	Fair	Poor	Very poor
Test score* . . .	13–15	9–12	5–8	1–4	−15–0
Self-accredited	0	30	45	25	0
Actual	0	5	60	25	10

*For details of test, see text.

The main sources of nutritional knowledge included the child welfare clinic (27%), general practitioner (22%), television and radio programmes (18%) and the health visitor (13%), but there was a noticeable lack of use of the clinic for reference to eating problems. It is apparent that although mothers have specific knowledge (e.g. toffees are bad for the teeth), they lack an over-all understanding of the importance of nutrition in relation to health.

Food intake and anthropometric data on children under 4 years old living in the south of England. By JANE MORGAN, PAMELA MUMFORD and ELIZABETH EVANS, *Department of Nutrition, Queen Elizabeth College, London W8 7AH,* and JOHN WELLS, *H. J. Heinz Co. Ltd, Hayes, Middx.*

Few studies have been reported on the food intake of healthy infants and additional data are required to assess the present situation (Department of Health and Social Security, 1974). During the period December 1973–January 1974 an investigation of the feeding practices of children aged 1–24 months was undertaken for market-research purposes. Mothers of 707 children, representing a national cross-section of the population, completed a quantitative 7 d record of the child's food intake, and by courtesy of one of the commercial organizations involved, we had access to this information. A subsample of children living within easy reach of London was selected for calculation of nutrient intake. Most of these children were visited in June 1975, when anthropometric and skinfold measurements were made and further information was collected on feeding practices.

Mean energy intakes within each age cohort, expressed either as MJ/d or MJ/kg body-weight, were lower than the Department of Health and Social Security (1969) recommended intakes (Table 1). A two- and even threefold variation in intake was observed between individuals in the same age-group. Mean protein

intake was higher than minimum requirements expressed both as g/d and g/kg body-wt per d. In the group under 6 months of age, the mean intake was 24 g/d, an amount considered by Ritchie & Naismith (1975) to be a factor in the aetiology of accelerated growth.

Table 1. *Body-weights and daily energy intakes of children compared with intakes recommended by the Department of Health and Social Security (DHSS)* (1969)

(Mean values with no. in sample in parentheses)

	DHSS recommended			Survey children			
		Energy intake			Energy intake		
						MJ	
Age (months)	Body-wt (kg)	MJ/kg body-wt	MJ	Body-wt (kg)	MJ/kg body-wt	Mean	Range
0–3	4·6	0·50	2·30	4·77 (3)	0·37	1·70 (3)	1·28–2·59
3–6	6·6	0·48	3·16	6·91 (12)	0·31	2·60 (15)	1·73–3·24
6–9	8·3	0·46	3·80	8·48 (9)	0·36	3·15 (14)	2·35–4·30
9–12	9·5	0·43	4·18	9·29 (15)	0·38	3·55 (22)	2·38–5·89
12–24	11·4	0·43	5·00	11·4 (17)	0·36	4·23 (55)	2·41–6·72

Examination of feeding practices revealed that by 1 week of age 63% of infants were totally bottle-fed, and by 4 months the value was 80%; 3% of mothers introduced solids in the first week accompanied in each instance by bottle-feeding; 84% of babies were receiving solids by 3 months of age.

Data collected in the follow-up study indicated some obesity in the children, then aged 18–42 months, although the literature provided no standard yardstick for defining obesity in this age-group. Skinfold measurements did not provide conclusive evidence; and four male and five female children were considered to be obese on the basis of a weight-for-height ratio, although not all exceeded the 90th weight centile (Tanner, Whitehouse & Takaishi, 1966).

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