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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Fifty-fourth Meeting of the Nutrition Society was held in The Witts Lecture Theatre, Radcliffe Infirmary, Woodstock Road, Oxford, on Thursday, 5 *February,* 1981, *when the following papers were read:*

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Estimates of energy intake by obese women on reducing diets. By MERRIL L. DURRANT, *Clinical Research Centre, Harrow, Middlesex*

Thirty-five obese women were confined to a metabolic unit for a 3-week course of weight reduction. Procedures were approved by the Northwick Park Hospital Ethical Committee. Subjects were not informed of their weight loss or the outcome of any tests until the end of their stay.

Acceptable diets were designed for each subject from a range of foods which had a threefold difference in energy density and were indistinguishable in appearance and taste (e.g. soups, milkshakes, sandwiches, etc.). Subjects were told that the energy content of their food might or might not vary during their stay.

On days 3–5 subjects were told what their intake was so they had a standard with which to compare the subsequent days. At the end of each day (6–21) subjects estimated the energy content of their diet and wrote this down on a recording sheet provided. Week 1 values are the mean of estimates on days 6–7, weeks 2 and 3 are the means of 7 d. Three dietary studies were run.

Six subjects were given 3·35 MJ/d with no change in energy intake. Their estimates showed no change. Twelve subjects were fed 3·35 MJ/d in the first week followed by either 0·84 MJ/d protein (PR) or 0·84 MJ/d carbohydrate (CHO) in weeks 2 or 3. A cross over design was used for weeks 2 and 3 so sequence was alternated.

No. of subjects	Actual intake (MJ/d)			Estimated intake (MJ/d)		
	Week 1	Week 2	Week 3	Week 1	Week 2	Week 3
6	3·35	3·35	3·35	3·35	3·27	3·34
12	3·35	0·84PR ×	0·84CHO	3·35	2·43** ×	2·14**
17	4·18	2·09	4·18	3·85	3·44*(*)	3·51*(*)

× denotes cross over design for alternate subjects.
*(*) $P<0·02$, (with respect to week 1) **$P<0·01$.

Estimates of intake were significantly lower in both weeks 2 and 3 compared with week 1 ($P<0·01$). Seventeen subjects ate 4·18 MJ/d in week 1, followed by 2·09 MJ/d in week 2 and 4·18 MJ/d in week 3. Subjects gave significantly lower estimates of intake in week 2 ($P<0·02$) but failed to detect the increase in intake in week 3.

These results show that obese patients on a reducing diet will detect a further covert decrease in energy intake, although estimates of the magnitude of the decrease are imprecise.

0029–6651/81/4002–354A $00.35

Weight gain of young rats fed on a cafeteria diet following excision of interscapular brown adipose tissue. By D. N. STEPHENS (introduced by JOHN DOBBING), *Department of Child Health, The Medical School, Manchester M13 9PT*

Unlike adults, young rats fed on a cafeteria diet fail to become obese (Stephens, 1980). The resistance to obesity has been attributed to dietarily-induced thermogenesis in which brown adipose tissue (BAT) plays a predominant role (Rothwell & Stock, 1979). The present study examines the effect of excision of BAT from one site upon weight gain in young rats fed on a cafeteria diet.

Ten litters of Lister hooded rats were weaned at 21 d and four male pups from each litter allocated to four groups. Two groups had interscapular BAT excised under phenobarbitone sodium anaesthesia (IBATX groups), the others (C group) receiving only anaesthesia. From 30 d one IBATX and one C group were fed on the cafeteria diet, the other IBATX and C groups continuing to receive stock diet (Porton Mouse Diet) *ad lib.* Body-weights and food intakes were recorded daily for the next 35 d.

Excision of BAT did not affect intake of either diet, but cafeteria-fed rats consumed 38% more energy than did stock-fed animals ($13{\cdot}5 \pm 0{\cdot}6$ *v.* $9{\cdot}8 \pm 0{\cdot}7$ MJ; $P<0{\cdot}01$). Despite their increased intake, the cafeteria-fed C group initially gained less weight than the stock-fed C group ($P<0{\cdot}02$), possibly as a consequence of increased thermogenesis since deep-body temperature at this time was elevated in the cafeteria-fed C group ($38{\cdot}7 \pm 0{\cdot}1$ *v.* $38{\cdot}1 + 0{\cdot}1°$; $P<0{\cdot}01$). The two IBATX groups did not differ in either weight or temperature.

From 45 d, the IBATX cafeteria-fed group gained weight more rapidly than the IBATX stock-fed group and by 65 d weighed 22 g more (7%; $P<0{\cdot}01$). By contrast, weights of cafeteria-fed C rats began to catch up with the stock fed animals only after 55 d, when there were no longer differences between the groups in body temperature, and did not differ from them at 65 d.

However, at 65 d, both the cafeteria-fed groups had 59% more fat than the stock-fed groups ($P<0{\cdot}001$) and the IBATX groups 10% less fat than their controls ($P<0{\cdot}05$). These findings do not entirely support the hypothesis that BAT-mediated thermogenesis can account for the failure of cafeteria diets to induce excess weight gain in young rats, but this may be related to a tendency for other BAT sites to show greater hypertrophy in the cafeteria-fed IBATX group than in the cafeteria-fed control group ($P<0{\cdot}05$).

Rothwell, N. J. & Stock, M. J. (1979). *Nature, Lond.* **281**, 31.
Stephens, D. N. (1980). *Br. J. Nutr.* **44**, 215.

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Measurement of body size—an evaluation of indices based on anthropometric observations. By M. D. ELSTON (Introduced by MARY GRIFFITHS), *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

This paper reports an analysis of anthropometric observations on a sample of pre-schoolchildren, their parents and siblings, designed to evaluate alternative indices of body size, particularly with reference to overweight.

During the course of a longitudinal study of growth in pre-schoolchildren, conducted by this department for the Department of Health and Social Security, measurements were made in the last year of the study of the height, weight, triceps skinfold, arm circumference, wrist and knee breadths of the natural parents and siblings of the study children. This provided a data set covering the age range from the first year of life through to adults in their fifties. The data were subsequently used to study familial and socio-economic patterns of overweight.

Assessment of overweight in biomedical investigations has in practice been based on a variety of measures. Where only anthropometric measurements were available, these have included easily computed measures based on weight and height such as the ratio of weight to height, more complicated measures of fat-free weight such as those derived by regression analysis of experimentally determined fat-free weight and anthropometric measurements, and indices based on anthropometric standards for height and weight.

In this paper the use of such measures is discussed and the relationships between them examined; their relative values in the assessment of overweight are compared, both in adults and in children.

For adults it is found that weight over height squared is a convenient and adequate measure of overweight, and that more complicated measures show no significant advantages. For children there appears to be no attractive alternative to the use of anthropometric standards. It is recommended that z-scores or centiles are used in preference to the more common use of percentages of median standard value.

Skinfolds constitute an obvious measurement directly related to body fat and their use in addition to these indices may be important for individual assessment of obesity.

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Some characteristics of obese British adults and their children. By MARY GRIFFITHS, M. D. ELSTON and P. T. FOX, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

During the period 1973–79 the Department of Human Nutrition at the London School of Hygiene and Tropical Medicine conducted a longitudinal growth study of over 3000 pre-schoolchildren for the Department of Health and Social Security as part of a system of nutritional surveillance. In the last year of the study anthropometric measurements were made of all the natural siblings. Height, weight, triceps skinfold, wrist and knee breadth were measured. Socio-economic information was also collected.

Various indicators of fatness were derived from the anthropometric data. Triceps skinfold were poorly related to other measures. Weight over height squared was best related with the remaining measures and cut-off points based on this index were used to indicate obesity. For children weight for height was used as an indicator of fatness.

It was found that mothers were more likely to be obese if (a) they came from social classes III, IV and V, (b) they had left school at the earliest opportunity, (c) they had had large families, (d) they did not smoke.

High income and non-smoking were the only indicators of obesity in fathers which were statistically significant. The socio-economic indicators bore little relationship to the prevalence of overweight in the children under 5 years of age. However, significant relationships were found between obesity in parents and overweight in their children. The relationship was particularly strong between the mother and child.

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The relationship between the growth pattern of children in Britain during the first five years of life and the fatness of their parents. By P. T. Fox, M. D. Elston and Mary Griffiths, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

This paper reports an analysis of the association between the pattern of growth in a sample of pre-schoolchildren and a measure of fatness in their parents, using information collected for a national longitudinal survey of pre-schoolchildren's growth.

During the period 1973–79 the Department of Human Nutrition conducted a longitudinal growth study of over 3000 children, in four parts of Britain, as part of a system of nutritional surveillance for the Department of Health and Social Security. In the last year of the study, anthropometric measurements were also made of natural parents and siblings of the subjects. These included height, weight, skinfold thickness, arm circumference, and wrist and knee breadth measurements.

The growth pattern over the following five years of those subjects who, at three months of age, were above the NCHS 90th percentile for height (length), weight and weight-for-height, was examined and its association with obesity in the parents noted. A similar study in reverse was undertaken looking at the earlier growth pattern of those subjects who were above the 90th centile of the NCHS standards at five years.

The analysis suggests that children above the 90th centile for weight for height at five years are more likely to have been in this position during their earlier life than children in this position at three months are likely to remain so. In addition children who are above the 90th centile at three months, and also have obese parents, are more likely to be above the 90th centile subsequently than are those children whose parents are not obese. However, it is observed that there is considerable crossing of centiles whichever way growth is traced. When socio-economic as well as familial factors are included in the analysis it is clear that obesity in the parents, particularly in the mother, is the only factor to display a consistent relationship with long-term obesity in these pre-schoolchildren.

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Increased resistance to obesity following early exposure to the cafeteria diet. By SARAH L. BROOKS, NANCY J. ROTHWELL and M. J. STOCK, *Department of Physiology, St. George's Hospital Medical School, London SW17*

Three groups (A, B, C) of male, weanling Sprague–Dawley rats were allowed *ad lib.* access to a pelleted stock diet but one group (C) was also fed on the cafeteria diet (Rothwell & Stock, 1979) for 30 d (period 1). In period 2 (30 d), all groups received stock diet only and in period 3 (22 d) groups B and C were both given the cafeteria diet. In the final period (period 4, 26 d) all groups were fed on stock diet alone.

Rats fed on the cafeteria diet at weaning consumed 42% more energy than stock-fed controls but showed a similar rate of weight gain. The efficiency of weight gain (g/MJ) was reduced by 44% and resting oxygen consumption ($\dot{V}O_2$) was elevated by 40%. When returned to stock diet these animals gained less weight than controls and at the start of period 3 had a significantly lower body-weight (mean±SE) (410±10 g, $P<0.05$) than either of the stock-fed groups (A, 445±14; B, 432±14).

During period 3, rats given the cafeteria diet for a second time (C) increased energy intake by 58% but gained significantly more weight (84±5 g) than controls (49±3, $P<0.001$). The hyperphagia of naive rats (B) fed the cafeteria diet was less (42% increase) but they gained significantly more weight (108±2 g) than groups A and C and the efficiency of weight gain was 40% ($P<0.01$) greater than that of stock-fed controls. Resting $\dot{V}O_2$ and the thermogenic responses to noradrenaline were significantly elevated in both cafeteria groups but were greatest in group C. Sub-groups were sacrificed at the end of period 3 and hypertrophy (mass) and hyperplasia (DNA content) of interscapular brown adipose tissue (IBAT) was seen in both cafeteria groups but this was greatest in group C. During the recovery period on stock diet groups B and C showed significantly lower weight gains and efficiency of gain and the final body-weights were similar in all groups. *IBAT* mass was also similar but DNA content remained significantly greater in group C.

This study suggests that early exposure to the cafeteria diet, results in a greater thermogenic capacity and resistance to obesity when hyperphagia is induced in later life.

Rothwell, N. J. & Stock, M. J. (1979). *J. Comp. Physiol. Psychol.* **93**, 1024.

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Effect of β-adrenergic blockade on thyroid hormone metabolism and resting oxygen consumption in fasted and refed rats. By M. ELIZABETH SAVILLE and M. J. STOCK, *Department of Physiology, St. George's Hospital Medical School, London SW17*

Male Sprague-Dawley rats (300–350 g) were divided into three groups and one (group 1) fed normally whilst the others (groups 2 and 3) were fasted for 72 h. Rats in group 3 then received a slurry of cornflour and water (42 kJ) by intragastric tube. Half of the animals in each group were injected with propranolol (20 mg/kg, s.c.) 30 mins before and 2 h after the time of refeeding. All animals were sacrificed 1 h later and blood taken for analysis of serum triiodothyronine (T_3).

Serum T_3 levels in fed and fasted rats were (mean±SE) 587±28 and 252±29 ng/l ($P<0.001$) respectively and were not significantly different in animals treated with propranolol (fed, 573±41 ng/l; fasted, 318±22 ng/l). In refed rats, T_3 levels were significantly higher (467±51 ng/l, $P<0.01$) than in fasted animals and not significantly different from fed values. However, in refed rats treated with propranolol, T_3 levels remained low (348±43).

Other groups of animals were used to investigate the effects of these treatments on the change in resting oxygen consumption ($\dot{V}O_2$) following the carbohydrate meal. Refeeding caused a significant rise in $\dot{V}O_2$ (10·43 ml/min per $W^{0.75}$, $P<0.002$) above fasting values (9·24±0·20 ml/min per $W^{0.75}$) 24 h after the meal but this effect was blocked by propranolol treatment at the time of refeeding ($\dot{V}O_2$ at 24 h, 9·19±0·19 ml/min per $W^{0.75}$). In untreated refed animals the rise in $\dot{V}O_2$ could also be inhibited by treatment with propanolol 24 h after the meal.

The effect of propranolol on T_3 levels following refeeding is probably caused by decreased 5′-deiodinase activity which results from either β-receptor blockade or the membrane stabilizing effects of the drug. The post-prandial rise in T_3 is required for the subsequent increase in $\dot{V}O_2$ but this delayed thyroid-dependent effect on metabolic rate also appears to be a β-receptor mediated response.

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The influence of dietary obesity on milk composition in the rat. By B. A. ROLLS, J. D. EDWARDS-WEBB and M. I. GURR, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT* and BARBARA J. ROLLS and E. A. ROWE, *Department of Experimental Psychology, University of Oxford*

Obesity can be induced in rats by the consumption of palatable foods (Rolls & Rowe, 1977). Rolls *et al.* (1980) have demonstrated that the pups of obese mothers grew less well and had a higher mortality than those of lean controls. Moreover, the obese mothers did not exhibit the usual hyperphagia during lactation.

Following the suggestion that the poor growth of the young may be related to impaired mammary gland lipogenesis (Rolls *et al.* 1980), it seemed appropriate to investigate the composition of the milk of lean and obese rats.

Two groups of female hooded Lister rats were given laboratory diet alone (control group) or laboratory diet supplemented with high energy palatable foods (obese group). After parturition at 25–30 weeks of age litter size was adjusted to eight. On the eighth day of lactation the pups were separated from the dam for 6 h and the rat was milked under light ether anaesthesia following an oxytocin injection. Proximate analysis of the milk showed that whereas there was little difference in protein and carbohydrate content between the two groups, the proportion of fat in the milk from obese rats was about twice that of milk from the lean controls (mean$\pm$SE) ($24{\cdot}4\pm2{\cdot}3$ *v.* $12{\cdot}5\pm0{\cdot}8\%$, v/v).

When the milk fats were analysed by gas–liquid chromatography, it was apparent that there were striking differences between the proportions of individual fatty acids in milks from the two groups. The milk of the obese group contained a lower proportion of the characteristic medium chain fatty acids and a higher proportion of long chain fatty acids than that of the lean group. For example, the value for decanoic:octadecenoic acids was 1·01 and 0·16 in the lean and obese groups respectively.

It seems probable that the additional long chain fatty acids were of dietary origin but it is not clear from these results whether the striking differences in values found were also influenced by impaired endogenous synthesis of medium chain length fatty acids.

Future work will be concerned with the interrelationships between dietary fatty acids and endogenous milk fat synthesis in obese animals and the possible effects on the young.

Rolls, B. J. & Rowe, E. A. (1977). *J. Physiol., Lond.* **272**, 2P.
Rolls, B. J., Rowe, E. A., Fahrbach, S. E., Agius, L. & Williamson, D. H. (1980). *Proc. Nutr. Soc.* **39**, 51A.

0029-6651/81/4002-354A $00.35

Effects of obesity on maintenance of body-weight in the lactating rat. By BARBARA J. ROLLS, E. A. ROWE and S. E. FAHRBACH, *University of Oxford, Department of Experimental Psychology, South Parks Road, Oxford OX1 3UD*

Obesity and the availability of high-energy diets plus chow (Rolls, Rowe & Turner, 1980) affect the performance of rats during lactation (Rolls, Rowe, Fahrbach *et al.* 1980). Control chow-fed hooded Lister rats (*n* 10) gained weight during lactation (mean ±SE) (5·0±3·7 g in three weeks) and lost weight after weaning (19·0±2·4 g in two weeks). All of the litters (culled to eight pups) of these controls survived and the mean pup weight at weaning was 32·6±1·0 g. Three different responses of the obese mothers during lactation were observed. 29% of these mothers lost all of the pups within 6 d and their post-partum weight remained stable (+6·0 g in three weeks). The obese mothers which retained litters (culled to eight pups) showed weight losses in lactation which were related to the body-weight immediately after parturition. Obese mothers (*n* 11) which had similar non-foetal weight gains in pregnancy as the controls weighed 330·7±3·6 g after parturition (compared to 290·7±5·9 g for controls), lost 27·7±2·4 g during lactation, and the pups weighed 26·7±1·8 g at weaning. The remaining obese mothers (*n* 11), which had gained significantly more non-foetal weight during pregnancy ($P<0·05$) weighed 374±7·4 g after parturition, lost substantial amounts of weight during lactation (59·1±6·1 g), and their pups weighed 25·5±0·9 g at weaning. At weaning the weights of the mothers in the different groups did not differ significantly, but the obese rats subsequently gained weight (3·0±3·9 g (*n* 11), and 13·3±6·1 g (*n* 11) in two weeks).

The substantial weight loss of the obese mothers during lactation and the poor growth of the offspring were associated with a lower energy intake during lactation which was only 61% greater than premating intakes, while the control mothers showed a 116% increase. These obese mothers ingested less protein and more lipid than the controls during lactation. Thus although chow and high-energy foods were freely available, obese rats selected inappropriate amounts of nutrients and energy to maintain maternal body-weight during lactation. The poor pup growth and survival also suggests an impairment of lactation in these obese rats.

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Rolls, B. J., Rowe, E. A., Fahrbach, S. E., Agius, L. & Williamson, D. H. (1980). *Proc. Nutr. Soc.* **39**, 51A.
Rolls, B. J., Rowe, E. A. & Turner, R. C. (1980). *J. Physiol., Lond.* **298**, 415.

0029–6651/81/4002–354A $00.35

The status of education on salt iodization in New Zealand. By J. A. BIRKBECK and M. F. GILLIES, *Department of Human Nutrition, University of Otago, Dunedin, New Zealand*

Iodine-deficiency goitre was formerly endemic in both islands of New Zealand (Kelly & Snedden, 1960), but the use of iodized salt combined with a vigorous education programme in the 1930s and 1940s essentially eliminated the condition. In more recent years the programme has not been promoted so actively. The National Diet Survey (Birkbeck, 1979) of a stratified random sample of adults aged 20–64 years provided an opportunity to assess the current status. Respondents were asked about their use of iodized salt, and the packet in use checked if possible. They were also asked why they thought salt was iodized. Of 1379 white respondents, 85% of 588 men were using iodized salt, and 6% were not; the rest being uncertain. 94% of 791 women did use iodized salt and 2·5% did not. The Table shows the proportion of respondents giving a reasonable explanation of its purpose.

		20–34 years	35–49 years	50–64 years	All
Males	*n*	272	185	131	588
	%	36·4	54·6	66·4	
Females	*n*	332	255	204	791
	%	53·0	78·4	78·9	

Although they had probably learned of it many years previously, older subjects are significantly more likely to give an accurate explanation. This suggests that such nutrition education had been effective. In the light of present concepts of iodide requirements, the recommended adult intake of 200 μg/d may well be provided by the unsupplemented diet.

Our figures for milk intake suggest that this source alone may provide up to 200 μg/d based on recent analyses (Sutcliffe & Davidson, 1979). Since the entire recommended intake is provided by 4 g of iodized salt, a review of supplementation is warranted.

Since adventitious iodide sources, such as iodophors, are subjected to large and chance variation, it is recommended that salt iodization should remain the cornerstone of prophylaxis. Adventitious sources should be subject to strict regulation.

Birkbeck, J. A. (1979). *New Zealanders and their diet.* Auckland: National Heart Foundation.

Kelly, F. C. & Snedden, W. W. (1960). *Monograph Ser. W.H.O.* **44**, 27.

Sutcliffe, E. R. & Davidson, F. (1979). *Proc. 2nd N.Z. Sem. Trace elements and health*, 123.

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The maximum capacity of the bovine liver to metabolize ammonia. By H. W. SYMONDS, DENISE L. MATHER and K. A. COLLIS, *Institute for Research on Animal Diseases, Compton, Newbury, Berkshire RG16 0NN*

The maximum capacity of the bovine liver to metabolize ammonia was measured in three non-lactating Friesian cows. The technique involved the use of silicone rubber cannulas inserted into the mesenteric, hepatic, jugular and portal veins and a carotid artery. Each cow received 4·5 kg hay and 3·5 kg dairy cake in two equal portions at 07.30 and 14.00 hours daily. A solution of ammonium acetate (2 mmol NH_3/ml) was infused into the mesenteric vein at a rate which was increased every 15 min until the cows became recumbent due to NH_3 intoxication. The effect of the acetate in the infusate was controlled by infusing a solution of sodium acetate at a similar rate 7 d later. The rate of uptake of NH_3 and production of urea and glucose by the liver was calculated from plasma NH_3 and urea and whole blood glucose concentrations in portal, hepatic and carotid vessels and hepatic blood flow which was measured by an indicator dilution technique using *p*-aminohippurate.

Time (min)	Mean NH_3 infusion rate (mmol/min)	Hepatic NH_3 uptake (mmol/min)			Hepatic urea output (mmol/min)			Hepatic glucose production (mean of A, B and C; mmol/min)
		A	B	C	A	B	C	
0	—	2·8	3·2	3·3	3·1	4·0	1·6	4·5
30	3·7	4·5	5·7	4·3	1·5	2·3	−0·3	4·3
60	10·0	11·7	8·0	7·8	8·2	5·7	3·0	3·8
90	15·5	12·2	18·6	11·2	7·1	10·9	2·8	4·9
120	22·6	10·0	5·0	21·7	7·9	7·2	3·9	6·0
160	27·9	9·3	5·8	10·9	8·4	9·7	5·8	15·7

The NH_3 concentrations in carotid and portal plasma before infusion of ammonium acetate averaged 0·04 and 0·19 mmol/l respectively. Severe signs of intoxication occurred when arterial concentrations exceeded 0·80 mmol NH_3/l. The concentration of NH_3 in carotid and hepatic plasma began to increase when the infusion rate reached approximately 16 mmol NH_3/min in cows A and B and 19 mmol NH_3/min in cow C. As shown in the Table the maximum capacity of the livers of cows A, B and C to metabolize NH_3 was respectively 12·2, 18·6 and 21·7 mmol/min (1·7, 2·1 and 2·6 mmol/min per kg liver wet weight). Urea production in cows A and B reached maxima of 9·4 and 10·9 mmol/min respectively while in cow C production reached a maximum of 5·8 mmol/min only at the end of the infusion. The mean (±SE) ratio of hepatic urea production to NH_3 uptake for cows A and B was 0·7±0·1 and 0·9±0·2 respectively. Hepatic glucose production did not increase until the cows were severely intoxicated.

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Growth and development of artificially reared well-fed and underfed rats.

By J. L. SMART, H. B. KATZ and D. N. STEPHENS, *Department of Child Health, The Medical School, Manchester M13 9PT*

The behavioural sequelae of early life undernutrition may possibly be the result of maternal effects other than nutrition *per se* (Smart, 1976). The most direct way to control for this is to rear rats artificially without their mothers. Previous attempts to rear pups artificially have seldom achieved normal growth, thus precluding a comparison between artificially-reared undernourished and artificially-reared well-nourished pups. In our experiment, 4-d-old rat pups were fitted with a gastric cannula and infused with a milk substitute (Hall, 1975). In most cases, two male pups/litter were taken for artificial rearing, one each for the well-fed control (ARC) and underfed (ARU) groups, with the remainder serving as mother-reared controls (MR). Twelve ARC and ten ARU rats were successfully reared out of initial populations of sixteen pups. Unlike Hall's method, the diet was infused intermittently not continuously: twelve times/d from 4–16 d, eight times/d from 16–21 d when rats were disconnected from the infusion pump. A wet mash was available from 18–25 d and solid food thereafter (both Porton Mouse Diet). Underfed pups received in total 44% of the milk given to well-fed pups and a restricted quantity of mash from 18–25 d, after which feeding was *ad lib.*

There were no significant differences in body-weight between ARC and MR males throughout the experiment (i.e. to 140 d). ARU rats were significantly lighter than ARC rats from 6 d onwards (all P values <0.001). Their weight deficit compared to ARC animals was 42% at the end of underfeeding, diminishing to 15% by 140 d. However, the absolute weight difference increased from 22 to 63 g over the same period.

Nose-rump length of conscious animals was measured at 21 and 25 d, but these results were unreliable and are not presented. However, the rats were rated on a four point scale for the intensity of their resistance to the restraint required for length measurements to be made. ARC rats reacted significantly more strongly on both occasions than MR rats ($P<0.02$) and the ARU rats scored intermediately between the other groups but did not differ significantly from either.

The age at which the eyes opened (an index of maturation) was brought forward by artificial rearing (ARC before MR, $P=0.011$) and tended to be retarded by undernutrition (ARC before ARU, $P=0.075$).

The adult behaviour of these animals is currently under test.

Hall, W. G. (1975). *Science, N.Y.* **190,** 1313.
Smart, J. L. (1976). *Physiol. Behav.* **16,** 147.

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Urinary fluoride in the Bedfordshire brickworks area. By G. THOMPSON and C. GEISSLER, *Department of Nutrition, Queen Elizabeth College, London W8*

The clays of the Marston valley in Bedfordshire contain 500–700 ppm fluoride of which about 40% is driven off during the firing of bricks, leading heavy fallout on crops and pastures and fluorosis in livestock (Burns & Allcroft, 1964). Possible high fluoride intakes cause concern to local inhabitants but no measurement of their exposure has been carried out.

As a preliminary survey the fluoride content of 24-h urine collections (1–3 from each subject) from fifty-three adult volunteers in three villages was measured by the fluoride electrode method of Tusl (1970). None of the subjects was employed at the brickworks. Very low air pollution levels were reported during the survey. The results are shown in the Table.

Village	Drinking water concentration (ppm)	No. of subjects (no. of 24 h urines)	24 h urinary F excretion (mg/24 h (range))		Urinary F concentration (ppm (range))	
			Mean	SE	Mean	SE
Kempston	0·7	10 (20)	1·7 (0·4–3·7)	0·9	1·4 (0·4–2·1)	0·5
Wootton	0·7	14 (38)	1·5 (0·5–3·6)	0·8	1·0 (0·2–3·2)	0·5
Lidlington	0·2	29 (65)	1·2 (0·2–2·4)	0·5	1·0 (0·2–1·9)	0·4

The mean 24 h fluoride excretion values are considerably higher than the mean excretion of 0·96±0·6 mg/24 h in South-east London and Kent. (Parsons *et al.* 1975). 20% of our subjects had mean excretion values ⩾2 mg/24 h. Also the urinary fluoride concentrations are more than twice the values expected from the often cited comparability of urine and drinking water concentrations in a moderate climate (WHO, 1970). Our range of urinary excretion values, 0·2–3·7 mg/24 h is within the range found in a population including heavy tea drinkers in an area with fluoridated water (Jenkins & Edgar, 1973). However, we found no relationship between urinary fluoride values and the stated quantity of tea consumed nor the use of fluoride toothpaste. On the basis that 50–100% of any absorbed fluoride is excreted in the urine of adults within 24 h (Largent, 1961) our results indicate intakes of 0·2 mg to over 7 mg/24 h.

Burns, K. N. & Allcroft, R. (1964). *Animal Disease Surveys Report* no. 2 part 1 MAFF. London: H.M. Stationery Office.
Jenkins, G. N. & Edgar, W. M. (1973). *J. Dent. Res.* **52**, 984.
Largent, E. J. (1961). *Fluorosis*, Ohio State University Press.
Parsons, V., Choudhury, A. A., Wass, J. A. & Vernon, A. (1975). *Br. Med. J.* **1**, 128.
Tusl, J. (1970). *Clin. Chim Acta* **27**, 216.
World Health Organization (1970). *Monograph Ser. W.H.O.* no. 59.

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Low digestibility of legume nitrogen. By A. E. BENDER and H. MOHAMMADIHA, *Nutrition Department, Queen Elizabeth College, Campden Hill, London W8 7AH*

The true digestibility of the nitrogen (TDN) of certain legumes is as low as 70% but as this investigation indicates this might not be due to failure to absorb dietary N but to increased loss of endogenous N.

Cooked, white kidney beans (*Phaseolus vulgaris*) were incorporated into a basal diet to provide a protein level (N×6·25) of 10% and fed to rats for 10 d periods. TDN was determined by feeding a non-protein diet (NP) to a group of littermates to estimate endogenous losses. TDN was (mean±SE) 74·2 (±1·3%) when fed to animals at 30 d of age and 54·5 (±1·2%) when fed to the same animals at 70 d. Casein TDN values were 97·0 (±0·8%) and 92·2 (±0·3%) respectively.

Faecal N can come from (1) undigested dietary protein (2) residues of digestive juices (3) lining of the intestinal tract and (4) bacteria. (3) and (4) can be estimated from faecal DNA. Determination of the viable bacteria in faeces sampled from the large intestine, of the DNA content of *E. coli* taken as a representative organism and DNA content of scraped mucosal cells permitted the calculations shown in the Table.

Diet		Total DNA (mg)	DNA:N	Bacterial count ($\times 10^{10}$)	DNA from bacteria (% total)
NP	a	49·3	0·63	4·2	1
	b	37·7	0·52	3·8	1
Casein	a	58·3	0·29	73·6	15
	b	43·7	0·23	66·6	18
Beans	a	155·4	0·33	118·4	9
	b	96·0	0·29	84·8	10

a, rats aged 63 d; b, replicated on rats aged 68 d.

Compared with NP diet total DNA excretion is little changed on feeding 10% casein diet while the value of DNA:N falls from 0·55 to 0·25. This indicates increased N from possibly undigested dietary protein and digestive juices. The bean diet, on the other hand, produced a very large increase in total DNA compared with NP and casein diets while the value of DNA:N is similar to that on casein. Approximate calculations show that only a small fraction of the DNA comes from (viable) bacteria suggesting that one of the sources of increased faecal nitrogen may be due to a massive increase in mucosal cell turnover.

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