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ABSTRACTS OF COMMUNICATIONS

The Two Hundred and Fifty-eighth Scientific Meeting of the Nutrition Society was held in the Windeyer Building, The Middlesex Hospital Medical School, Cleveland Street, London W1P 7PN, on Friday, 18 *May* 1973, *at* 10.30 *hours, when the following papers were read:*

Aversion to sucrose in obesity. By P. J. UNDERWOOD, ELIZABETH BELTON and PAT HULME, *Clinical Research Centre, Northwick Park Hospital, Harrow, Middlesex*

The hypothesis that obese people are highly sensitive to external stimuli, but relatively insensitive to internal signals (Schachter, 1968; Stunkard, 1968) was investigated by Cabanac & Duclaux (1970).

Our experimental design was similar to that of Cabanac & Duclaux. We tested eighteen obese subjects admitted to hospital for weight control: fifteen adult females, mean weight 94 kg, range 72–137 kg; three adult males, mean weight 109 kg, range 91–125 kg; eleven lean adult volunteers (ten female, mean weight 51 kg, range 40–60 kg and one male, weight 76 kg).

A set of six solutions containing 10–400 g sucrose/l was presented to each subject in random order before and 1 h after an oral load of 50 g glucose. The change in affective response is shown in Fig. 1: in the obese group there was no significant

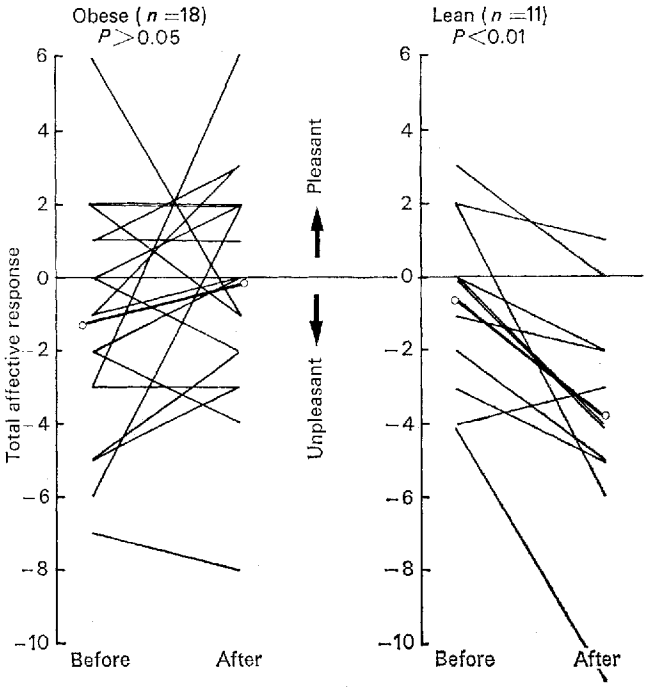

Fig. 1. Total affective response to sucrose solutions before and after a glucose load in obese and lean subjects.

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change ($P>0.05$), but in the normal group there was significant aversion ($P<0.01$). The one lean subject who failed to show aversion had anorexia nervosa.

Contrary to the results of Cabanac & Duclaux (1970), most of our subjects found the solutions initially unpleasant.

The absence of a sucrose aversion response in obese subjects is consistent with the hypothesis that obesity is associated with decreased sensitivity to internal signals.

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Factory accidents and carbohydrate supplements. By J. D. BROOKE and S. TOOGOOD, *Human Performance Laboratory, Physical Education Section, University of Salford*, and L. F. GREEN and R. BAGLEY, *Beecham Products, Beecham House, Great West Road, Brentford, Middlesex*

The present paper communicates details of carbohydrate provision and the incidence of accidents in forge workers briefly described earlier (Brooke, Toogood, Green & Bagley, 1973).

Over 18 weeks, fifty-eight male forge workers, acting as their own controls, were given, each working morning, supplements of either 500 ml glucose syrup drink (2·972 MJ (0·710 Mcal) energy value) containing added salts, or 500 ml of a low-energy fluid (<40 kJ (9·6 kcal) energy value) including the salts. Treatments were reversed after 4, 8 and 13 weeks. In addition, seventeen men who refused to take the dietary supplements were studied. Reported accidents and body-weights before and after experiment were recorded and random determinations were made of blood glucose (glucose-oxidase method) and respiratory quotient (Kofranyi-Michaelis respirometers and Haldane analysis).

Accidents to the men on the glucose-syrup treatment were significantly lower ($P<0.05$) than on the low-energy treatment (a rate of 14/100 men compared with 34/100 men). The rate in the non-co-operative group (88/100 men) was significantly higher than the rate in either of these two treatment groups. The differences were more apparent in the morning than in the afternoon work sessions. Changes in body-weights were found to be not significant, the mean increase being 0·36 kg (± 1.96, s $= \pm 4.47$ kg). Reduced weight gain or reduced deposition of adipose tissue with glucose *v.* sucrose intake in rats (Feyder, 1935) and baboons (Brook & Noel, 1969) has been reported.

Because of the many problems in carrying out this programme in the environment of forge hot-shops, the relations between the measures of metabolism and accident incidence must be treated with care.

Blood sugar concentrations and amount of carbohydrate combusted (Brooke *et al.* 1973), for the men receiving the glucose-syrup treatment in the morning, were higher than for those who had the low-energy treatment and non-co-operative

group. However, the mean values of these measurements for the three groups do not show sufficient disparity to relate them directly to accident incidence.

Group mean blood glucose concentrations were most disparate in the early morning (975, 926 and 875 mg/l) and similarly respiratory quotient values were most disparate in the 10.00–12.00 hours period (0·92, 0·86 and 0·83). Sensitive interpretation of these results is not justified.

This work suggests that there may be a relationship between reduction in factory accidents and the appropriate intake of this glucose-syrup drink. We are studying the problem further by a laboratory research of controlled performance with appropriate changes in metabolic conditions.

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Food intake and response to diet therapy of 650 adult obese patients.

By G. L. S. PAWAN, *Metabolic Division, Department of Medicine, The Middlesex Hospital Medical School, London W1P 7PN*

A study on the dietary intake and response to treatment of 650 adult non-diabetic obese subjects, more than 140% ideal body-weight, who were referred to the author by various physicians, showed that they could be classified into two main groups.

In the first group, conveniently designated 'overeaters', the mean daily food intake of each subject was more than 10·5 MJ (2500 kcal), with a range of 10·5–20·4 MJ (2500–4850 kcal). Carbohydrate intake of the subjects provided 55–66% of the total metabolizable energy value of their diets. Of the obese patients, 82% were in this group. The majority of these subjects had a history of adult-onset obesity, generally associated with a relatively low level of physical activity. On a reducing diet of 4·2 MJ (1000 kcal) daily, satisfactory weight loss occurred. Of these subjects, 62% showed regain of the lost weight in a follow-up study some months later. This was mainly due to inadequate control of food intake.

In the second group, which comprised 18% of the obese population, the mean daily food intake of the subjects was less than 10·5 MJ (2500 kcal) with a range of 5·0–10·5 MJ (1200–2500 kcal). The majority of these individuals had a history of juvenile-onset obesity, and exhibited an eating pattern of less than three meals daily, with the larger meal taken usually at night and generally high in carbohydrate. These patients lost weight slowly and with difficulty on a diet of 2·1–4·2 MJ (500–1000 kcal)/d, even when encouraged to increase their physical activity. Rapid weight regain was a common finding on a diet of 8·4 MJ (2000 kcal)/d. This group may be called 'energy conservers'.

Nine subjects (that is, less than 2% of all the obese patients studied) demonstrated some adrenocortical hyperactivity with abnormal sodium and water retention

and expansion of the total body water volume. In these individuals, dietary sodium restriction produced an improved body-weight (water) loss.

The metabolic significance of these findings was discussed.

The relevance of glycerol metabolism to the hyperlipidaemic property of sucrose. By D. J. NAISMITH and BERTHA P. B. KARIKARI, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

The hyperlipidaemic effect of sucrose has been attributed to differences between the metabolism of glucose and fructose—a constituent monosaccharide of sucrose but not of starch. Macdonald (1970) has suggested that fructose ingestion leads to a greater production of α-glycerophosphate, which is necessary for fat synthesis, and thus promotes an increase in plasma triglycerides. In support of this hypothesis, he showed that the ingestion of glycerol, which is also a precursor of α-glycerophosphate, increased the plasma triglyceride concentration in young men.

An alternative explanation, that glycerol might act as a precursor of fatty acids, was tested in the present experiment.

Twelve litter-mate pairs of weanling rats were fed, *ad lib.*, on diets containing 750 g starch/kg or 500 g starch + 250 g glycerol/kg. After 70 d the rats, in the fed state, received 10 μCi [U-^{14}C]glycerol by intraperitoneal injection. Two hours later, the animals were killed by stunning, and blood was withdrawn for measurements of radioactivity and for lipid analyses. The activities of pyruvate kinase (PK) and glucose-6-phosphate dehydrogenase (G-6-PD), enzymes which regulate lipogenesis, were measured in liver and adipose tissue. The results are summarized in the table.

Dietary carbohydrate	Wt of liver (g)	Enzyme activities in liver (I.U./g)		Plasma lipids (g/l)			Specific activity of triglycerides (counts/min per mg)
		PK	G-6-PD	Cholesterol	Triglycerides	Phospholipids	
Starch	12·2	34·9	5·6	0·70	0·38	1·15	226
Starch + glycerol	13·6	78·5	12·0	0·77	0·61	1·43	260

Feeding with glycerol more than doubled the activities of both enzymes in the liver, indicating that the synthesis of fatty acids was greatly increased. In contrast, lipogenesis in adipose tissue was depressed. Values for PK and G-6-PD were 1·91 and 0·44 I.U./g for the control animals, and 1·57 and 0·34 I.U./g for the rats given glycerol. The pattern of lipogenesis in these tissues was therefore similar to that found in sucrose-fed rats (Naismith, 1971). All plasma lipid fractions were increased in the group which received glycerol. The differences reported were all statistically significant.

No difference was found in the specific activity of the plasma triglycerides, and hydrolysis of the triglycerides revealed that the proportional distribution of radioactivity within the molecule was the same in the two groups; approximately

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75% of the count was recovered in the fatty acid fraction. These findings suggest that glycerol and glucose follow the same metabolic pathway leading to the synthesis of fat.

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Depot fatty acids of gnotobiotic lambs. By W. M. F. LEAT, *ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*, and R. J. LYSONS and T. J. L. ALEXANDER, *Department of Animal Pathology, University of Cambridge, Cambridge CB3 0ES*

The depot fatty acids of ruminants are characterized by a high content of stearic acid, which is mainly a result of the hydrogenation of dietary lipids in the rumen (see Hilditch & Williams, 1964). However, the proportion of stearic acid derived from endogenous and exogenous sources is still relatively undefined. To provide further information on the endogenous contribution of stearic acid to depot fatty acids, the tissues of gnotobiotic lambs were analysed. Three lambs were obtained by hysterectomy and reared in isolators by gnotobiotic techniques (Alexander & Lysons, 1971) until they were killed at 18–20 weeks of age. They were fed on sterile cow's milk until they were 7 weeks old and on a sterile solid ration from 3 weeks of age to death. The main constituents of this ration were (g/kg): barley straw 100, hay 50, barley 500, protein supplement 300 and molasses 50. The lipid content of this diet was 24 g/kg (calculated).

One of the gnotobiotic lambs (17a) was maintained free from normal rumen microflora; a second gnotobiotic lamb (16a) was dosed with seven species of rumen bacteria (Lysons, Alexander, Hobson, Mann & Stewart 1971) at 6–7 weeks of age; and a third gnotobiotic lamb (17b) was dosed with fresh rumen contents from an adult conventional sheep at 6–7 weeks of age. Samples of subcutaneous and perinephric fat were taken at post-mortem examination from all three gnotobiotic lambs and compared with those from a 5-month-old lamb (20) grazing pasture (Table 1).

Table 1. *Major fatty acids (% of total) of subcutaneous (S) and perinephric (P) fat from gnotobiotic (17a, 16a and 17b) and pasture-fed (20) lambs*

	17a		16a		17b		20		Dietary
Fatty acid	S	P	S	P	S	P	S	P	fatty acids
16:0	24·0	24·1	26·5	24·0	26·8	25·5	21·9	18·3	20·8
18:0	9·3	16·2	9·4	15·9	15·6	21·8	14·9	29·4	5·1
18:1	43·8	38·7	45·5	42·0	41·7	37·7	45·4	36·7	28·4
18:2	7·5	8·6	6·3	6·2	1·7	4·0	3·2	3·9	31·8

These results indicate that, in lambs, endogenous synthesis could account for an appreciable proportion of the stearic acid found in perinephric and subcutaneous fat. In the gnotobiotic lambs, values for stearic acid were higher than those recorded

for non-ruminant herbivores (Hilditch & Williams, 1964) but lower than those found in ruminants fed on fat-free diets (Reiser, Choudhury & Leighton, 1959; Duncan, Garton & Matrone, 1971).

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Lipolytic enzymes of sheep pancreatic juice. By G. ARIENTI, F. A. HARRISON and W. M. F. LEAT, *ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

In the sheep, micro-organisms in the rumen hydrolyse dietary lipids to free fatty acids (see Garton, 1967) and the role of pancreatic juice in the subsequent digestion and absorption of lipids in the small intestine is obscure. In cattle and sheep the output of pancreatic lipase has been reported to be low compared with that in the non-ruminant animal (Keller, Cohen & Neurath, 1958; Taylor, 1962).

Pancreatic juice was obtained by the method of Taylor (1960) and immediately freeze-dried. Subsequent experiments showed that this juice contained powerful lipolytic activity when incubated with a triolein emulsion at a pH optimum of 7·5. Under our experimental conditions, the lipolytic activity was equivalent to the release of 2·5 mequiv. fatty acid/ml pancreatic juice per min. The other reaction products were almost exclusively monoglyceride and 1:2 diglyceride. The hydrolysis of triolein was stimulated by Ca^{++} and bile salts with maximum stimulation at concentrations in the ranges found in the duodenum of the sheep.

Freeze-drying of pancreatic juice resulted in a 20% decrease in lipolytic activity but the activity of the freeze-dried powder was retained for up to 10 years when stored at $-20°$. Lipolytic activity against triolein was heat-labile, and less than 5% of the activity was present after heating for 10 min at 60°. The measurements of the lipolytic activity so far made are consistent with the properties of a lipase.

Lipase activity was detectable at pH 5 but not at more acid values. When pancreatic juice was pretreated by incubation at different pH levels for 10 min at 39° before lipase estimation at pH 7·5, 100% of the lipolytic activity was lost by pretreatment at pH 2·5; 70% lost at pH 3·0; and only 10% lost at pH 3·5. Duodenal secretions, bile and pancreatic juice buffer the acidity of abomasal digesta and, as Harrison & Hill (1962) showed, the pH of digesta increases from pH 2·5 on leaving the abomasum to pH 3·5 immediately posterior to the common bile duct. Considerable lipase activity would therefore be preserved even in the comparatively acid conditions of the sheep duodenum.

Pancreatic juice was also shown to be lipolytic against bile or a bile salt–lecithin micelle, with the fatty acids of both 1 and 2 positions of lecithin being hydrolysed.

After heating for 10 min at 60° more than 50% of the lipolytic activity against lecithin was destroyed and the residual activity caused almost exclusive hydrolysis of fatty acids from the 2 position, suggesting the presence of a heat-stable phospholipase A.

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The estimation of tryptophan. By N. A. MATHESON, *Department of Protein Biochemistry, Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Few of the presently available procedures for estimating tryptophan in purified proteins are entirely satisfactory and when applied to tryptophan determination in feeding-stuffs usually prove even less satisfactory. A method has been developed which incorporates several features from other procedures as well as some original ones. It allows determinations of the tryptophan content of many samples daily and is not sensitive to interference by other components.

The method is based on the colorimetric reaction between tryptophan and dimethylbenzaldehyde for the estimation (Spies & Chambers, 1948, 1949) but uses an alkaline hydrolysate rather than whole protein for the reaction (cf. Spies, 1967); 5 N-barium hydroxide rather than sodium hydroxide is used for the hydrolysis which is done in polypropylene vessels in an autoclave overnight (Miller, 1967; Oelshlegel, Schroeder & Stahmann, 1970). Most of the barium hydroxide which interfered with colour development was removed by cooling, and a corrected calibration curve was prepared from pure tryptophan in presence of the appropriate concentration of barium hydroxide. The results for purified proteins were obtained

Table 1. *Tryptophan content of feeding-stuffs*

Sample and no.	Tryptophan content (g/kg protein)† NAM*	ARC‡
Fish meal: FM101	9·4	11·1
FM102	8·3	9·5
FM122	11·5	13·5
FM123	12·4	14·0
Hydrocarbon yeast: HY101	13·4	12·2
HY104	14·7	12·2
Meat meal: MM101	8·7	10·5
Groundnut: GN101	12·0	13·1
Sunflower-seed meal: SF101	13·7	13·6

*Method outlined in this paper; each result is the mean of two replicates on each of two hydrolysates (four values per sample).
†(N × 6·25).
‡Analytical Panel of the Protein Evaluation Group of the Agricultural Research Council, by a chemical method (Miller, 1967) with correction for loss during hydrolysis.

on a tenth of the scale. Hydrogen sulphide is produced from cystine and cysteine during alkaline hydrolysis and acidification. It interferes with the colour reaction used to determine tryptophan but may easily be removed from a hydrolysate under reduced pressure after acidification.

When the method was applied to purified proteins their apparent tryptophan content was usually within 10% of the accepted value. Samples of feeding-stuffs which had been already examined by the Analytical Panel of the Agricultural Research Council's Protein Evaluation Group were analysed by the above method (Table 1). Agreement here was still reasonable when it is remembered how unreliable most tryptophan methods are with crude materials.

The method above has also been applied to single-cell protein, barley, feather-meal and fatty tissue, with reproducible results in the expected ranges. One worker can carry out duplicate tryptophan determinations on at least fifteen samples daily without any special apparatus or skills.

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Investigations on favism-inducing toxins in broad beans (*Vicia faba* L.)

By J. JAMALIAN, F. AYLWARD and B. J. F. HUDSON, *Department of Food Science, University of Reading, Reading*

The importance of broad bean seed as a source of protein for human consumption needs no emphasis. However, a haemolytic disease, favism, in certain subjects whose red blood cells are deficient in glucose-6-phosphate dehydrogenase (G-6-PD) has been known for more than 100 years to be associated with the consumption of fresh or dry broad beans. Comparatively little attention has been paid to the toxic principles in broad beans as opposed to the disease itself. As part of the investigations on toxic principles in broad beans the following studies have been carried out.

Preparations of dry, ground broad-bean seeds from a favism-prone area were suspended in phosphate buffer solutions the pH of which ranged from 5·3 to 7·7. Samples were centrifuged. The precipitates were homogenized in distilled water and treated with 95% ethanol; the supernatants were freeze-dried and extracted with 95% ethanol. The ethanolic extracts were further extracted with light petroleum (b.p. 40°–60°) and the light petroleum-soluble materials in both fractions were discarded because they had been shown previously to have no activity. The ethanolic extracts were then evaporated to dryness at low temperature and reduced pressure. They were taken up in known volumes of phosphate-buffered isotonic saline (pH 7·4).

Samples of extracts in buffered saline were incubated for 3 h at 37° with blood (packed cell volume adjusted to 30% with ACD (acid citrate–dextrose solution)) from two G-6-PD-deficient, favism-sensitive subjects and a non-sensitive control subject.

Blood incubated with buffered isotonic saline served as internal control for each subject. Reduced glutathione (GSH), known to be unstable in blood from G-6-PD-deficient subjects, and the packed cell volume values of the mixtures were determined immediately after incubation.

Extracts were also subjected to thin-layer chromatographic and spectrophotometric examination and tested chemically for pyrimidines and related compounds.

In general, extracts of precipitates from treatments at different pH were less active and their difference in activities less pronounced than those of the supernatant fractions. The presence of two short-wave uv-absorbing compounds in the precipitates and three in the supernatant fractions was demonstrated. Correlations were observed between relative quantities of the uv-absorbing compounds (maximum at 270 nm) in the extracts of supernatant fractions and the activity of these extracts as determined by the fall in GSH concentration in blood from sensitive subjects during the course of incubation.

Dichlorophenolindophenol, phosphomolybdate and ferric chloride–ammonia tests were negative, both with untreated extracts of precipitates and after acid hydrolysis. Weakly positive reactions were noted for extracts of supernatant fractions before acid hydrolysis, and strong positive reactions after acid hydrolysis.

The value of dietary protein for hyperplasic growth at a near-maintenance energy intake. By D. J. NAISMITH and B. L. G. MORGAN, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

Under conditions of food shortage, the use of high-protein supplements for the infant population is frequently advocated, although the diet may be severely lacking in energy. Whether, under such conditions, protein is of value other than as a source of energy has not been determined. This dietary situation was simulated in rats weaned at 19 d of age. The pups were distributed, as litter-mates, into three groups of eight. The first group was fed, *ad lib.*, on a diet containing 400 g casein/kg. The second received the same diet, but intake was restricted to 3·5 g/d. The third group was given a diet containing 100 g casein/kg, again restricted to 3·5 g/d. After 16 d on the diets, the animals were killed and the liver, kidneys and two muscles were analysed for protein and DNA. The content of protein and fat in the carcasses was also measured.

The mean daily food intake of the rats fed *ad lib.* was 14·0 g. Thus the animals on restricted rations received only 25% of the voluntary food consumption.

The results of the analyses are summarized in the table.

		Liver		Kidney		Muscle	
Diet	Gain in body-wt (g)	Protein (mg)	DNA (mg)	Protein (mg)	DNA (mg)	Protein (mg)	DNA (mg)
High-protein (*ad lib.*)	61·9	970	6·21	200	4·74	160	0·54
Low-protein (restricted)	2·8	390	2·72	100	2·36	80	0·32
High-protein (restricted)	10·5	450	3·15	130	2·77	110	0·39

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Restricting food intake had a very marked effect on growth. Nevertheless, the rats given the high-protein diet gained almost four times as much weight as their littermates fed on the low-protein diet. Carcass protein was raised by 24%, but no difference in body fat was detected. The benefit of an increased proportion of protein in the diet was also seen in the values for protein in the liver (+15%), the kidneys (+30%) and in the muscles (+37%). Total DNA in the liver and kidneys was raised by 16% and 17% respectively, while muscle DNA was increased by 22%. The differences we report were all statistically significant.

The experiment shows clearly that growth by hyperplasia is influenced by the concentration of protein in the diet even when the intake of energy approximates to the maintenance level.

The long-term effects of marginal protein–energy deficiency. By R. J. C. STEWART, R. F. PREECE and HILDA G. SHEPPARD, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*

In addition to the low birth weights (Stewart, Preece & Sheppard, 1972) brought about by protein-energy deficiency there are also long-term, and possibly permanent, stigmata. Many young rats of the deprived colony died during suckling and the remainder grew very slowly, probably resulting, in part, from the reduced quantities of milk produced by their mothers. In the malnourished colony, weight at weaning was 58% and body length 20% below that of well-fed controls. Part of this deficit was made good by an increased period of growth, but 6-month-old animals of the colony maintained on the low-protein ration (NDp:E 6·8) weighed about 35–40% less than those of the well-fed colony. The deficits in adult body-weight increased with succeeding generations.

The coats of the animals were poor and easily damaged. For instance, rubbing of the head and neck against food hoppers during feeding caused no ill-effects in the control rats, but led to bald patches in the deficient animals. Changes in hair colour were common, usually with the development of grey rather than black hair, but occasionally the hair had a distinct brownish hue. Although their size was not altered, the eyes of the malnourished rats appeared to be prominent and bulging.

There were delays of up to 4 or 5 weeks in the sexual maturation of both the males and females. Maternal behaviour was modified, the mother being less industrious in nest building and often allowing the young to become scattered. This dispersal was increased by the enhanced exploratory behaviour of the malnourished young.

Occasionally a nursing mother appeared to undergo a convulsion during which she destroyed the nest and killed her young. This was thought to be related to unusual noise, and certainly all members of the deprived colony responded to noise more markedly than did well-fed controls.

Histological changes similar to, but usually less severe than, those described in malnourished pigs and dogs have been found. As in the dog, the rats passed through

a stage, probably related to dendritic development, when signs such as stiff hind legs, unsteady gait and athetoid movements of the head were exacerbated.

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Recovery from long-term protein–energy deficiency. By R. J. C. STEWART, R. F. PREECE and HILDA G. SHEPPARD, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*

The possibility existed that some of the results obtained from the colony marginally deficient in protein energy, especially the low birth weights, slow growth and increasing deficit in adult size, might be caused by selective survival and breeding. An attempt was made to determine the degree of recovery brought about by re-feeding, at various stages of their development, the rats which had been marginally malnourished for nine generations. Giving the diets of high-protein value from 4 weeks of age led to an increased growth, but at 24 weeks of age the animals attained only 67% of the weight and 90% of the length of those on the well-fed colony. The values for the ninth generation of malnourished rats were 57 and 88%. Fostering to a dam of the well-fed colony within 24 h of birth led to a more marked improvement, 90 and 93% respectively. Clearly, postnatal re-feeding of small-for-dates young had to be started very early to have any real effect on size. During the 3rd week *in utero* conditions are especially adapted for growth, and an attempt was made to take advantage of this situation. Pairs of malnourished females were mated to the same male and 14 d after mating one of each pair was given the diet of higher protein value. Amongst the re-fed group birth weights increased to an average of 5·3 g, 98% of normal, weaning weights were 95% of normal, and at 24 weeks of age the males were 7·5% heavier and 5% longer than rats of the well-fed colony.

An unexpected finding was that the enhanced exploratory behaviour and increased response to noise which differentiated the members of the deprived from those of the control colony were still present in these physically rehabilitated rats. Distinct differences have been found between the deprived and well-fed colonies in visual discrimination tests (Turkewitz and Stewart, unpublished results), and it will be interesting, when sufficient animals are available, to discover whether the 'rehabilitated' animals respond in the same way as the deprived or as the well-fed colony. However, these animals do illustrate the great degree of rehabilitation that can occur and the possibility of separating physical growth from some other indices of development and maturation.

The response of growing, castrated male pigs to various hormones of possible anabolic potential. By M. KATHARINE ELLIOTT and V. R. FOWLER, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Three androgens, methyltestosterone (17α-methyl-17β-hydroxy-androst-4,5-ene-3-one), mesterolone (17β-hydroxy-1α-methyl-5α-androstan-3-one) and

methenolone acetate (17β-acetoxy-1-methyl-5α-androst-1-ene-3-one), were fed to forty-two growing, castrated male pigs with and without 2·0 mg diethylstilboestrol per kg feed. Androgen dose rates were 0, 0·5, 0·9, 1·7, 3·0, 5·5 and 10·0 mg/kg feed. The animals were fed twice daily, starting at 1600 g/d at 35–40 kg live weight, with weekly increments of 140 g/d. The trial ran for 10 weeks, the hormones being withdrawn 1 week before slaughter. Carcass gains and carcass food conversion ratios (kg food/kg gain) were calculated, and gains and food conversion ratios of lean tissue were estimated using the specific gravity of the carcass as a predictor of lean tissue according to formulas from Adam & Smith (1964).

Table 1. *Means and standard errors (SE) for the effect of hormone treatments on the performance of growing, castrated male pigs*

			Lean tissue		Carcass	
Treatment	Diethyl-stilbestrol	No. of animals	Gain (kg)	Food conversion ratio	Gain (kg)	Food conversion ratio
(1) Methyltestosterone	−	6	13·4	11·7	38·6	4·1
(2) Mesterolone	−	6	12·4	12·6	38·8	4·0
(3) Methenolone acetate	−	6	13·1	12·0	39·0	4·0
(4) Methyltestosterone	+	6	14·8	10·7	40·1	3·9
(5) Mesterolone	+	6	13·6	11·4	37·9	4·1
(6) Methenolone acetate	+	6	14·6	10·6	41·4	3·8
SE of differences for any pair of treatments 1–6			0·83	0·71	1·40	0·15
Significance of differences between 1+4, 2+5 and 3+6			NS	NS	NS	NS
(7) All treatments	−	21	12·9	12·2	38·8	4·1
(8) All treatments	+	21	14·3	10·9	39·7	3·9
SE of differences			0·48	0·40	0·77	0·08
Significance of differences between 7 and 8			($P<0·01$)	($P<0·01$)	NS	NS

NS, not significant.

Results were pooled for each hormone combination, because there was no evidence of any significant response to dose level. There were no differences between pigs on any androgen treatment and those receiving none. The total results for each androgen showed no significant differences (Table 1). Pigs given diethylstilboestrol had significantly greater gains ($P<0·01$) and lower food conversion ratios ($P<0·01$) of lean tissue than those not given this oestrogen, but the differences between these groups for carcass gain and food conversion ratio were not significant. This suggests that increases in lean tissue growth occurred without the improvement in conversion of food to carcass which one would expect from the fact that the heat of combustion of lean tissue is very much less than that of fatty tissue. It appears that, with growing pigs, exogenously supplied oestrogenic hormones could have a greater potential for promoting an anabolic response than do androgens.

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REFERENCE

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Vitamin A and epilepsy: a dietary contretemps. By I. M. SHARMAN, *Dunn Nutritional Laboratory, University of Cambridge and Medical Research Council, Milton Road, Cambridge CB4 1XJ*, and G. STERN, *University College Hospital Medical School, Gower Street, London WC1E 6JJ*

Sharman (1969) reported findings on a unique case of a young man with long-standing epilepsy who by unscientific reasoning believed vitamin A was responsible, and who resolved to eliminate all sources of the vitamin from his diet. He did this so effectively that the concentration of the vitamin in his blood was reduced to the lowest ever recorded in this country. During a period of 4 years he had only one minor attack of epilepsy but complications including progressive bilateral corneal xerosis and severe retinal damage resulted (Bors & Fells, 1971). These disabilities were, however, reversed by vitamin A therapy but a further fit ensued. It appeared possible, though unlikely, that vitamin A might be involved and it was decided to investigate the incidence of fits in severely disabled epileptics in relation to their vitamin A status.

Some difficulty was experienced in finding an adequate number of suitable patients who were sufficiently disabled by frequent fits to justify inclusion in a trial, and since such patients are usually confined to institutions, they also had to be intelligent enough to pursue the proposed dietary restrictions.

Nine patients were studied in detail for nearly 2 years; one died from *status epilepticus*. The rest were first observed on a normal diet and then on a diet of very low vitamin A content. Vitamin supplements, excluding vitamin A, were given throughout the trial. Patients remained on their usual anticonvulsant therapy and their witnessed attacks were recorded. When blood concentrations of retinol reached a lower limit, and at a time unknown to the patient, a supplement of vitamin A was provided with the other vitamin supplements.

A battery of tests was carried out at the commencement, during and at the end of the experimental period. The tests included measurements of blood carotenoids, retinol, haemoglobin, cyanocobalamin, folate, electrolytes, calcium, phosphorus and phosphatase determinations. Urinary amino acids were investigated before and after the trial. Serial electroencephalograms, electroretinograms and tests of dark adaptation and visual acuity were also performed.

Although blood concentrations of retinol fell to about 70 μg/l, no impairment of dark adaptation was observed. Throughout the trial the patients reported that they felt better in themselves and had fewer fits. But in none was there any demonstrable change when vitamin A was introduced, although blood concentrations rose.

Any improvement that did occur must therefore be ascribed to their improved well-being and not to a reduced vitamin A status.

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