The Two Hundred and Seventy-seventh Scientific Meeting of the Nutrition Society was held in the Edward Lewis Lecture Theatre, Middlesex Hospital Medical School, London W1P 7PN, on Friday, 14 March 1975, at 13.30 hours, when the following papers were read:

The effects of feeding starch, sucrose, glucose or fructose to rats during pregnancy and early lactation. By A. R. BOURNE* and D. P. RICHARDSON, *Queen Elizabeth College, London W8 7AH*, and K. R. BRUCKDORFER, *Department of Biochemistry and Chemistry, Royal Free Hospital School of Medicine, London WC1*, and JOHN YUDKIN, *Servier Research Institute, Horsenden Lane South, Greenford, Middlesex*

An increase in the concentration of plasma triglyceride occurs in the rat when it is given diets with sucrose or fructose (Bruckdorfer, Khan & Yudkin, 1972) and also during pregnancy (Hamosh, Clary, Chernick & Scow, 1970). We report here some of the effects of feeding various carbohydrates to Sprague-Dawley rats during pregnancy and early lactation.

The diets contained (g/kg): carbohydrate 680, casein 230 and maize oil 16, together with added vitamins and mineral salts. In the first experiment, with 129 rats, the carbohydrate was either maize starch or sucrose; in the second experiment, with ninety-one rats, it was glucose or fructose. The rats were fed on these diets from weaning and throughout pregnancy and lactation. At days 7, 14 and 20 of pregnancy, and 2 d after parturition, non-fasting plasma samples from the rats were assayed for triglyceride, cholesterol, free fatty acid and glucose; in addition, lipogenic potential was assayed by measuring the activity of fatty acid synthetase in the liver and in adipose tissue. The results were compared with those from non-pregnant female rats.

Dietary sucrose or fructose produced a higher concentration of plasma triglyceride in non-pregnant rats and also in pregnant and lactating rats than did dietary starch or glucose. With all dietary carbohydrates, pregnancy resulted in an increase in triglyceride concentration at day 20, which returned to normal after parturition; this hypertriglyceridaemia was enhanced by sucrose or fructose.

The concentration of plasma glucose was depressed, and that of free fatty acid increased, at day 20, but the nature of the dietary carbohydrate did not affect these values, nor the concentration of plasma cholesterol.

Replacement of starch in the diet by sucrose, or of glucose by fructose, increased the activity of fatty acid synthetase in the liver and decreased it in the adipose tissue. This effect in the liver was intensified by the end of pregnancy.

These metabolic changes do not appear to affect foetal growth.

*Present address: Department of Physiology, Monash University, Clayton, Victoria, Australia.

REFERENCES

Bruckdorfer, K. R., Khan, I. H. & Yudkin, J. (1972). *Biochem. J.* **129**, 439.
Hamosh, M., Clary, T. R., Chernick, S. S. & Scow, R. O. (1970). *Biochim. biophys. Acta* **210**, 473.

The effect of dietary sucrose and dietary cholesterol on hyperlipidaemia and atherosclerosis in White Leghorn cockerels (*Gallus domesticus*).

By NANCY ANN WORCESTER and K. R. BRUCKDORFER, *Department of Biochemistry and Chemistry, Royal Free Hospital School of Medicine, London WC1*, and JOHN YUDKIN, *Servier Research Institute, Horsenden Lane South, Greenford, Middlesex*

Compared with dietary starch, dietary sucrose increases the concentration of plasma triglyceride in the rat (Bruckdorfer, Khan & Yudkin, 1972), baboon (Macdonald & Roberts, 1967), monkey (Lang & Barthel, 1972) and man (Macdonald, 1964). It has also been reported that it increases the concentration of cholesterol in the rat (Al-Nagdy, Miller, Qureshi & Yudkin, 1966) and in man (Yudkin & Szanto, 1972). In the cockerel, plasma cholesterol concentration is increased by dietary cholesterol, which also produces atherosclerosis (Katz & Pick, 1961).

We have examined the effects in cockerels of dietary sucrose, with or without dietary cholesterol.

Two groups each of six White Leghorn cockerels aged 6 weeks were given low-fat, cholesterol-free diets with maize starch or sucrose (500 g/kg). After 4 weeks, plasma from non-fasted birds was assayed for triglyceride and cholesterol. In a second experiment, diets with starch or sucrose, and with or without cholesterol (5·2 g/kg), were fed to each of four groups of ten cockerels aged 8 weeks. Plasma samples for lipid assay were taken after an overnight fast, at 3 weeks and 12 weeks from the start of the experiment. The birds were killed at 16 weeks, and the aortas were examined for atherosclerosis before and after staining with Sudan IV.

Plasma concentrations of triglyceride and cholesterol were increased by dietary cholesterol, but were unaffected by the different carbohydrates. No atherosclerosis was seen in cockerels given cholesterol-free diets. The diets with cholesterol produced in the abdominal aorta rather more atherosclerosis when sucrose was present than when it was absent, but the variation between birds was so great that the effect of sucrose was not statistically significant.

In similar experiments, Tashev & Patscheva (1968) reported a considerable increase of atherosclerosis in cockerels given sucrose. It may be that this occurred because of the larger amounts of cholesterol in their diets. In addition, carbohydrate metabolism in birds is characterized by a high conversion of glucose and fructose to fat by the liver (O'Hea & Leveille, 1968). Again, different species show a varying sensitivity to dietary cholesterol, so that for example it readily produces atherosclerosis in the cockerel and in the rabbit, but not in the rat.

REFERENCES

Al-Nagdy, S., Miller, D. S., Qureshi, R. U. & Yudkin, J. (1966). *Nature, Lond.* **209**, 81.
Bruckdorfer, K. R., Khan, I. H. & Yudkin, J. (1972). *Biochem. J.* **129**, 439.
Katz, L. N. & Pick, R. (1961). *J. Atheroscler. Res.* **1**, 93.
Lang, C. M. & Barthel, C. H. (1972). *Am. J. clin. Nutr.* **25**, 470.
Macdonald, I. (1964). *Proc. Nutr. Soc.* **23**, 119.
Macdonald, I. & Roberts, J. B. (1967). *Metabolism* **16**, 572.
O'Hea, E. K. & Leveille, G. A. (1968). *Comp. Biochem. Physiol.* **26**, 111.
Tashev, T. & Patscheva, G. (1968). *Ernährungsforschung* **13**, 339.
Yudkin, J. & Szanto, S. (1972). *Horm. Metab. Res.* **4**, 417.

The effect of dietary sucrose and different dietary fats on hyperlipidaemia and atherosclerosis in White Leghorn cockerels (*Gallus domesticus*).

By NANCY ANN WORCESTER and K. R. BRUCKDORFER, *Department of Biochemistry and Chemistry, Royal Free Hospital School of Medicine, London WC1*, and JOHN YUDKIN, *Servier Research Institute, Horsenden Lane South, Greenford, Middlesex*

Dietary sucrose and saturated fats have a synergistic effect upon the concentration of plasma triglyceride in the rat (Bruckdorfer, Kari-Kari, Khan & Yudkin, 1972) and in patients with hyperlipoproteinaemia (Antar, Little, Lucas, Buckley & Csima, 1970). In cockerels, no effect was found on the concentration of either triglyceride or cholesterol when hydrogenated coconut oil was substituted for safflower oil (Bruckdorfer *et al.* 1972). There is some disagreement about the effect of saturated fat on atherosclerosis in cockerels, negative results having been reported by Stamler, Pick & Katz (1959) and positive results by Cembrano, Mardones, Hegsted & Stare (1967).

Four groups each of ten White Leghorn cockerels aged 8 weeks were given diets containing maize starch or sucrose (about 400 g/kg), and hydrogenated coconut oil or maize oil (about 100 g/kg). All diets contained cholesterol (5·2 g/kg). After 3 weeks and 12 weeks, triglyceride and cholesterol were assayed in fasting plasma. At 3 weeks, but not at 12 weeks, triglyceride concentrations were elevated in the sucrose-fed birds; cholesterol concentrations were not affected. In both sets of samples, the concentrations of both lipids were higher in the cockerels given hydrogenated coconut oil than in those given maize oil. At 12 weeks, there was also an interaction between dietary sucrose and fat on the concentration of triglyceride but not on that of cholesterol.

At 16 weeks, the cockerels were killed and the aortas examined. As in the previous experiments (Worcester, Bruckdorfer & Yudkin, 1975), there was a wide variation in the extent of atherosclerosis seen in the unstained arteries, and in the area of staining with Sudan IV. Thus, although there were more diseased arteries in the sucrose groups than in the starch groups, the difference did not reach a statistically significant level. On the other hand, the greater amount of atherosclerosis in the cockerels given hydrogenated coconut oil compared with maize oil, as measured both in stained and unstained aortas, was highly significant.

The possible reasons for the failure to produce significant differences in atherogenesis between dietary starch and dietary sucrose are discussed in the preceding paper (Worcester *et al.* 1975).

REFERENCES

Antar, M. A., Little, J. A., Lucas, C., Buckley, G. C. & Csima, A. (1970). *Atherosclerosis* **11**, 191.
Bruckdorfer, K. R., Kari-Kari, B. P. B., Khan, I. H. & Yudkin, J. (1972). *Nutr. Metab.* **14**, 228.
Cembrano, J., Mardones, J., Hegsted, D. M. & Stare, F. J. (1967). *J. Atheroscler. Res.* **7**, 387.
Stamler, J., Pick, R. & Katz, L. N. (1959). *Circulation Res.* **7**, 398.
Worcester, N. A., Bruckdorfer, K. R. & Yudkin, J. (1975). *Proc. Nutr. Soc.* **34**, 81A.

Serum lipids and some aspects of diet in young female survivors of myocardial infarction. By MARGARET THOROGOOD and J. I. MANN, *Department of Social Medicine, University of Oxford, Oxford*

Myocardial infarction (MI) is uncommon in young women and little information is therefore available concerning risk factors for the condition in this group of people.

We have studied fasting serum lipids and lipoprotein patterns in forty-four female survivors of acute MI under the age of 45 years and eighty-four matched control subjects. Limited information concerning some dietary practices before and after the acute episode was obtained. Mean cholesterol and triglyceride concentrations are shown in Table 1. Subjects with hyperlipoproteinaemia were identified taking the upper limits of normal as 2 SD above the mean for our control subjects (Table 1) and the typing based on the lipid levels and the lipoprotein pattern on electrophoresis.

Table 1. *Mean serum concentrations of cholesterol and triglyceride in control and myocardial infarction (MI) subjects*

	Controls			
	Mean	Upper limit of normal (2 SD above mean)	MI subjects	Significance of difference between groups
Cholesterol (mmol/l)	5·05	6·92	5·93	$P<0·001$
Triglyceride (mg/l)	1090	2070	1330	$P<0·02$

Type IIa hyperlipoproteinaemia was present in 30% of the MI subjects and type IIb in 11%. Neither of these abnormalities was found in any of the controls. There was no difference in the prevalence of the type IV abnormality, which was present in 7% of the MI subjects and 8% of the controls; the higher mean triglyceride levels observed amongst the MI subjects must therefore be attributable to the excessive prevalence of type IIb. There was no difference in the reported consumption of eggs between the MI and control subjects, and similar numbers in both groups reported an attempt to control the saturated fat content of their diet before their hospital admission. MI subjects, however, drank more cups of coffee per d than did the

controls (14% of MI subjects *v.* 7% of controls drank more than six cups per d). Tea-drinking did not differ between the two groups.

No information is available concerning dietary advice given to the MI subjects; however, the fact that 38% of them (*v.* 14% of the controls) were eating fewer eggs at the time of study than at the time of admission suggests that many were given at least some dietary instruction. Mean body-weight (at the time of study) was similar to the weight recorded on admission, but the four subjects who weighed more than 80 kg at the time of admission had all lost weight at the time of study.

Some results concerning the association between population food consumption and ischaemic heart disease mortality will also be presented.

Is there a 'set point' for human body-weight? By J. S. GARROW and SUSAN STALLEY, *MRC Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ*

It has been suggested that obese people regulate their adipose mass about a reference 'set point' which is inappropriately high (Shapiro, 1973). However, despite intensive research, the nature of the control system is not understood, and it has been suggested elsewhere (Garrow, 1974) that there is no physiological 'set point'. An

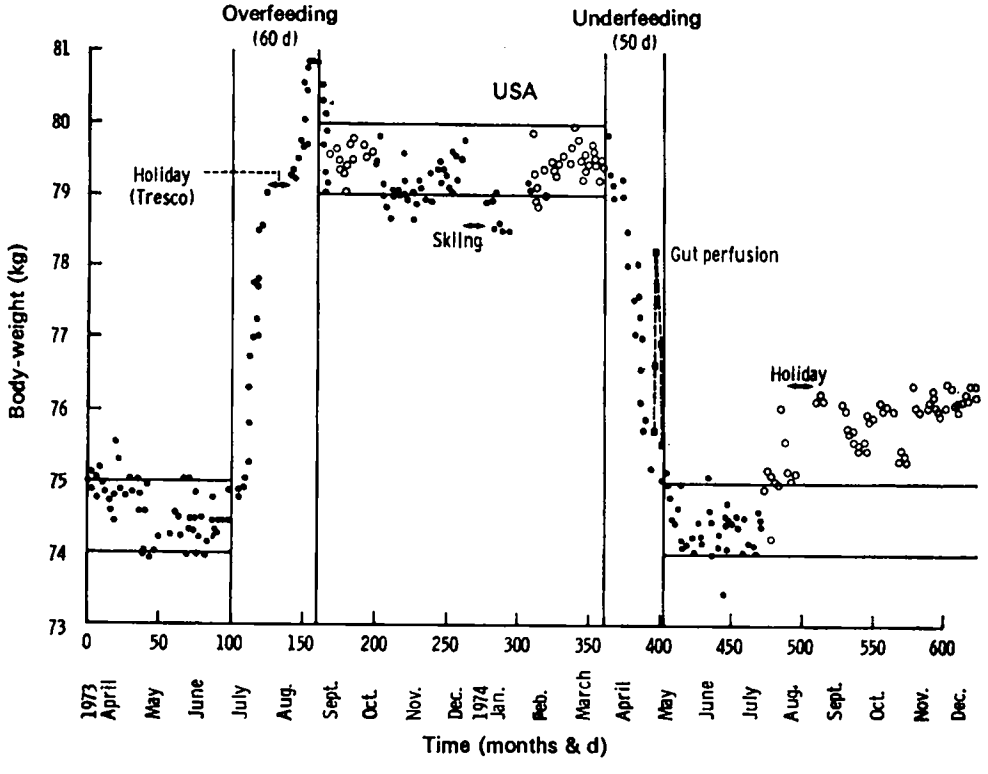

Fig. 1. Weight change in a man of average body-weight during a period of 21 months; ○, times during which he did not know, and could not guess, his weight change.

https://doi.org/10.1079/PNS19750027 Published online by Cambridge University Press

alternative hypothesis is that there is a 'buffer' control system, which tends to oppose weight change when energy intake is changed, but, once a new level is reached by overfeeding or underfeeding, the energy stores will tend to remain at that level until a sufficiently great energy imbalance is imposed to override the 'buffer' control.

Fig. 1 shows an attempt to test this theory experimentally. A normal adult male was overfed by about 5 MJ daily in order to increase his weight by 5 kg. After 200 d there was no evidence of a tendency to revert to the baseline weight, so weight was reduced by underfeeding. In this subject weight seemed to be subject to 'buffer' control, and was just as stable when he was weighed 'blind' as when he knew his weight.

REFERENCES

Garrow, J. S. (1974). *Energy Balance and Obesity in Man.* Amsterdam: North Holland.
Shapiro, B. (1973). In *Energy Balance in Man*, p. 247 [M. Apfelbaum, editor]. Paris: Masson.

Resistance to slimming: adipose tissue cellularity studies. By MARGARET ASHWELL, PAULINE PRIEST, MARINETTE BONDOUX and J. S. GARROW, *MRC Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ*

It has recently been suggested (Björntörp, 1974) that 'there may be some association between adipose tissue cellularity and the possible caloric expenditure regulation' which might explain why 'hyperplastic obese patients are those who are most difficult to reduce to a normal body-weight by introduction of a negative caloric balance'. We were interested to use the needle biopsy technique to compare the cellular characteristics of: (1) a group of twenty-one obese subjects who believed they could lose weight on a suitable negative energy balance (sample A) and a group of fifteen obese subjects who were selected as being particularly resistant to further weight reduction (sample B); and (2) a sub-group of sample B (good losers) ($n=8$) who lost more than 1·5 kg during a 3-week trial where daily energy intake was strictly limited to 6·3 MJ (1500 kcal) and a second sub-group (poor losers) ($n=7$) who gained weight or lost less than 1·5 kg during the 3-week trial. (Further details of this 3-week trial will be given by Miller & Parsonage (1975).)

There was no significant difference between the subjects in samples A and B with respect to (mean $\pm$ SE): age (A $36{\cdot}6\pm2{\cdot}59$ years; B $41{\cdot}7\pm2{\cdot}65$ years), relative weight (A $1{\cdot}37\pm0{\cdot}036$; B $1{\cdot}38\pm0{\cdot}075$) or body fat content (A $29{\cdot}0\pm1{\cdot}38$ kg; B $29{\cdot}0\pm2{\cdot}69$ kg). Neither was there any significant difference between average fat cell weight (A $0{\cdot}707\pm0{\cdot}056$ μg; B $0{\cdot}628\pm0{\cdot}042$ μg) or the total number of fat cells (A $4{\cdot}407\pm0{\cdot}276\times10^{10}$; B $4{\cdot}739\pm0{\cdot}258\times10^{10}$).

The good losers and poor losers differed significantly with respect to their average weight loss ($2{\cdot}58\pm0{\cdot}196$ *v.* $0{\cdot}097\pm0{\cdot}552$ kg) ($P<0{\cdot}001$) but they showed no significant differences with respect to age ($39{\cdot}75\pm2{\cdot}55$ *v.* $43{\cdot}85\pm4{\cdot}56$ years), relative weight ($1{\cdot}49\pm0{\cdot}124$ *v.* $1{\cdot}24\pm0{\cdot}04$), body fat ($33{\cdot}5\pm4{\cdot}38$ *v.* $23{\cdot}9\pm1{\cdot}54$ kg) or adipose tissue cellularity characteristics (fat cell weights $0{\cdot}641\pm0{\cdot}061$ *v.* $0{\cdot}612\pm0{\cdot}061$ μg; total fat cell number $5{\cdot}29\pm0{\cdot}483\times10^{10}$ *v.* $4{\cdot}10\pm0{\cdot}448\times10^{10}$). The only significant difference ($P<0{\cdot}01$) between the good losers and the poor

losers was their resting metabolic rate (5565±154 kJ (1330±36·7 kcal) *v.* 4615±190 kJ (1103±45·3 kcal)/24 h).

The preliminary results reported here do not lend support to Björntörp's (1974) observations or conclusions since the supposed resistant group (sample B) showed no greater evidence of hyperplasia than sample A and there were no significant cellularity differences between good losers and poor losers.

REFERENCES

Björntörp, P. (1974). *Acta med. Scand.* **196**, 145.
Miller, D. S. & Parsonage, S. (1975). *Lancet* i, 773.

Glucose tolerance, plasma insulin levels and insulin sensitivity in geriatric patients. By C. R. C. HEARD, W. S. SOERJODIBROTO and SYLVIA M. FRANGI, *Clinical Nutrition and Metabolism Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*, and A. N. EXTON-SMITH, *Department of Geriatrics, University College Hospital, London*

Venous blood glucose values, 120 min after an oral load of 50 g glucose (G_{120}) are used to diagnose diabetes (WHO, 1965). Values <6·11 mmol/l are classified as normal and >7·22 mmol/l as diabetic. However, G_{120} increases steadily with age and this raises problems in the elderly both of clinical significance and of physiological interpretation.

This communication reports the results of an investigation of twenty-four geriatric patients (mean age 79 years) in whom both availability of insulin and insulin sensitivity were assessed.

Each patient received two tests on successive days: (1) oral glucose tolerance test (GTT) (50 g glucose) lasting 150 min; (2) intravenous (i.v.) GTT (0·33 g glucose/kg) lasting 60 min followed immediately by i.v. glucose+insulin (0·33 g glucose and 0·133 units insulin/kg) with blood sampling for a further 60 min (Heard & Henry, 1969). Half the patients had the oral test on the first day, half had the i.v. test. Patients were fasted from midnight. Blood was sampled and glucose and insulin injected via an indwelling butterfly needle in the arm. Glucose (glucose oxidase method) and insulin (Radiochemical Centre method) were estimated in plasma. Glucose disappearance rates (k; % per min) in the i.v. tests were calculated to give k_G for glucose alone and k_{G+I} for glucose + insulin. Normal values are about 2 and 5% per min respectively (Franckson, Malaise, Arnould, Rasio, Balasse, Conrad & Bastenie, 1966).

Although only one patient was overtly diabetic (fasting glucose 8·10 mmol/l), sixteen of the twenty-four had G_{120} values >7·77 mmol/l. Of these, fourteen also had severely impaired i.v. glucose tolerance (k_G <1·0% per min). Only two other patients had k_G <1·0% per min. Clear signs of glucose malabsorption occurred in three patients, who therefore showed very low plasma insulin levels during the oral GTT compared with values during the i.v. GTT. Another five patients without

malabsorption also showed low plasma insulin levels during the oral GTT suggesting impairment of insulinogenic gut factors. Although plasma insulin levels during both tests were lower than those reported for young normal subjects, insulin sensitivity (k_{G+I}) was also low in all the patients (mean 2·4% per min). The shape of the oral GTT curves suggested that failure to suppress hepatic glucose release was a feature of this insulin insensitivity.

The extent to which these changes are typical of old age per se and whether deterioration in nutritional status contributes to the effects remain to be established.

REFERENCES

Franckson, J. R. M., Malaise, W., Arnould, Y., Rasio, E., Balasse, E., Conrad, V. & Bastenie, P. A. (1966). *Diabetalogia* **2**, 96.
Heard, C. R. C. & Henry, P. A. J. (1969). *Clin. Sci.* **37**, 37.
WHO (1965). *Wld Hlth Org. tech. Rep. Ser.* no. 310.

'Added lactose' and 'added sucrose' cow's milk formulas in neonatal nutrition. By B. A. WHARTON and A. S. FOSBROOKE, *Institute of Child Health, Guilford Street, London WC1N 1EH*

During the manufacture of infant feeding formulas, carbohydrate may be added to dried cow's milk to lower the relative proportions of protein and minerals to levels nearer those in human milk. The particular carbohydrate added varies, though in other circumstances it is known that the type of dietary carbohydrate affects intestinal tolerance, deposition of body fat (in rats) and concentrations of plasma lipids (in man).

In this study a comparison has been made between feeding either an added lactose formula only or an added sucrose formula only to a total of twenty-three low-birth-weight babies during the first 3 months of life. The 'added lactose' group experienced more diarrhoea (five were withdrawn from the trial with diarrhoea compared with one of the 'added sucrose' group) and a greater degree of metabolic acidosis during the first week of life (base excess (mequiv./l) (mean $\pm$ SD at 7 d), 'added lactose' group -7 ± 4, 'added sucrose' group -3 ± 4; $P<0{\cdot}05$). The 'added sucrose' group did not become fatter nor were their concentrations of plasma lipids higher than in the 'added lactose' group. The results at 3 months were, for the 'added lactose' and 'added sucrose' groups respectively (mean $\pm$ SD): total skinfold thickness (mm) $10{\cdot}9\pm2{\cdot}6$, $8{\cdot}7\pm2{\cdot}2$ ($P<0{\cdot}03$); serum cholesterol (mmol/l) $4{\cdot}25\pm1{\cdot}24$, $4{\cdot}14\pm0{\cdot}70$ (not significant); serum triglyceride (mg/l) 1090 ± 390, 680 ± 350 ($P<0{\cdot}02$).

Despite the teleological arguments in favour of lactose we have found no objective contraindications to the use of 'added sucrose' cow's milk formulas in the nutrition of low-birth-weight babies.

Both milks contained only very low proportions of linoleic acid and the poly-unsaturated fatty acid content of the plasma and adipose tissue lipids fell to low levels, but no clinical evidence of essential fatty acid deficiency was found.

Oxidation and metabolism of linoleic acid in the sheep. By W. M. F. LEAT, D. B. LINDSAY and G. VALERIO, *Biochemistry Department, ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

In many simple-stomached animals the requirement for essential fatty acids, mainly linoleic acid, is equivalent to 1–2% of the dietary energy (see Holman, 1968). In ruminants, however, most of the dietary polyunsaturated fatty acids are hydrogenated in the rumen and the amount of linoleic acid entering and absorbed from the intestine is only 0·3% of the dietary energy (Leat & Harrison, 1972). However, essential fatty acid deficiency has never been noted in adult ruminants, which suggests that linoleic acid is conserved in ruminants and utilized more efficiently than other long-chain fatty acids.

To test this hypothesis [1-^{14}C]linoleic acid was infused intravenously into two starved (72 h) sheep continuously for 2–3 h, and the entry rate, extent of oxidation and incorporation into other plasma lipids was estimated. Comparison was made with results obtained from the infusion of [1-^{14}C]palmitic and [1-^{14}C]stearic acid into these sheep (Table 1), and with published values for these fatty acids (Leat & Ford, 1966; Annison, Brown, Leng, Lindsay & West, 1967).

The entry rate of linoleic acid (0·05 mg/min per kg) was very low compared to that of palmitic acid (0·43 mg/min per kg) and stearic acid (0·47 mg/min per kg). The contribution of linoleic acid to carbon dioxide production was also very low and the proportion of the entry rate that was oxidized appeared to be lower than values obtained for palmitic and stearic acid, both in this experiment and those published elsewhere (25–40%).

Table 1. *Oxidation and metabolism of linoleic, palmitic and stearic acids infused intravenously into two starved (72 h) sheep*

Fatty acid infused	Plasma concentration (μmol/l)		Fatty acid oxidized		Maximum percentage isotope incorporation into plasma	
	Total free fatty acid	Fatty acid studied	(%CO_2 output)	(% entry rate)	Phospho-lipids	Cholesteryl esters
[1-^{14}C]linoleic	801·8	18·3	0·37	16·6	—	—
[1-^{14}C]linoleic	309·6	12·5	0·39	14·4	1	1
[1-^{14}C]stearic	683·9	209·9	9·8	21·1	0·2	<0·1
[1-^{14}C]palmitic	830·5	132·9	11·0	31·3	—	—

A possible explanation for this finding is suggested by the preliminary observation that, in contrast to stearic acid, an appreciable proportion of the infused [1-^{14}C]-linoleic acid is sequestered in some form, and subsequently appears in plasma phospholipids and cholesteryl esters. This transfer is even more marked in fed sheep.

Plasma phospholipids and cholesteryl esters are rich in linoleic acid, but in ruminants the free fatty acid fraction contains very low levels of linoleic acid (see Leat, 1966); this could explain the low oxidation of this fatty acid in starved sheep. The lower percentage of the entry rate of linoleic acid which is oxidized may be the result of the greater incorporation of linoleic acid into plasma phospholipids and cholesteryl esters, where it would be protected from oxidation.

REFERENCES

Annison, E. F., Brown, R. E., Leng, R. A., Lindsay, D. B. & West, C. E. (1967). *Biochem. J.* **104**, 135.
Holman, R. T. (1968). In *Progress in the Chemistry of Fats and Other Lipids*, vol. 9, p. 278. Oxford: Pergamon Press.
Leat, W. M. F. (1966). *Biochem. J.* **98**, 598.
Leat, W. M. F. & Ford, E. J. H. (1966). *Biochem. J.* **101**, 317.
Leat, W. M. F. & Harrison, F. A. (1972). *Proc. Nutr. Soc.* **31**, 70A.

The influence of high dietary intakes of calcium on lead retention and release in rats. By J. QUARTERMAN, J. N. MORRISON and W. R. HUMPHRIES, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

The retention of dietary lead by rats is increased when the dietary calcium content is decreased to levels below requirement (Six & Goyer, 1970; Quarterman, Morrison & Carey, 1974). A decrease of dietary Ca also inhibits the release and excretion of Pb already incorporated into the body of the rat (Quarterman *et al.* 1974). It is not certain that increases of dietary Ca above requirement affect the retention and release of Pb.

Rats weighing about 75 g were given a diet (Quarterman *et al.* 1974) supplemented with calcium carbonate to provide 7 or 18 g Ca/kg and with lead acetate as required. In Expt 1, rats were given diets containing 400 mg Pb and 7 or 18 g Ca/kg for 3 weeks. In Expt 2, three groups of rats were given the diet with 7 g Ca/kg and with 200 mg Pb/kg for 6 weeks. One group was then killed for analysis. The remaining two groups were then given diets with 7 or 18 g Ca/kg but with no Pb supplements for a further period of 6 weeks. At the end of each experiment the rats were killed with sodium pentabarbital and Pb was estimated in the gut-free carcase of each rat.

In Expt 1 supplementary Ca greatly reduced the carcase content of Pb at the end of the experiment. Food consumption and weight gain were not influenced by dietary Ca content (Table 1). The net retention of Pb had thus been reduced by increased dietary Ca. In the 1st part of Expt 2 the rats accumulated Pb in the carcase. During

Table 1. *Effects of diets with different calcium and lead contents on carcase Pb contents and body-weights of rats at the end of each experimental period*

(Mean values with their standard errors where given)

	Diet	No. of rats	Period of feeding (weeks)	Dietary Ca (g/kg)	Dietary Pb (mg/kg)	Carcase Pb (μg)	Body-wt (g)
Expt 1	Control	5	3	7	400	952±18 *	126±1
	High-Ca	5	3	18	400	529±30	126±2
Expt 2 (1st part)	Control	5	6	7	200	713±40	257±3
(2nd part)‡	Control	6	6	7	0	466±19† *	384±9
	High-Ca	5	6	18	0	554±32†	386±5

*Difference statistically significant ($P<0.05$).
†Difference from 713±40 significant ($P<0.05$).
‡Immediately before the 2nd part of Expt 2 the rats had been treated as the rats in the 1st part had been, in order to allow accumulation of Pb in the carcase.

https://doi.org/10.1079/PNS19750027 Published online by Cambridge University Press

the period in which the Pb-free diet was given and Pb was lost from the carcase, supplementary Ca reduced the amount of Pb lost (Table 1).

From this work and that quoted above it seems that the net retention of Pb decreases as dietary Ca increases from below to above requirement. The loss of Pb already incorporated into the carcase is greater when the dietary Ca is near to requirement than when it is greater or less than requirement.

REFERENCES

Quarterman, J., Morrison, J. N. & Carey, L. F. (1974). In *Trace Substances in Environmental Health*, vol. 7, p. 347 [D. D. Hemphill, editor]. Columbia, Missouri: University of Missouri.
Six, K. M. & Goyer, R. A. (1970). *J. Lab. clin. Med.* **76**, 933.

Glycosyl ureides in ruminant feeding. By A. B. McALLAN, R. J. MERRY and R. H. SMITH, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Milligan, Worsley, Elofson, Young & Atwal (1972) showed that ammonia was formed only slowly when glucosyl urea was added to the rumen of the sheep. The apparent resistance to degradation was confirmed when we incubated glucosyl urea anaerobically with strained calf rumen contents; more than 0·8 g/g remained intact after 4 h, although degradation of both glucose and urea in a mixture similarly incubated was nearly complete.

Glucosyl urea (m.p. 206–208°) was prepared according to Hynd (1926). Lactosyl urea (m.p. 233–234°) was prepared similarly from lactose and urea; it appeared in a peak close to the sucrose position when it was treated with borate and passed through an anion-exchange column (Smith & McAllan, 1971). Lactosyl urea and an equimolar mixture of lactose and urea were each incubated anaerobically and at a concentration of 6 mmol/l with whole rumen contents from a calf given chopped straw and barley. For the mixture, after 0·5 h, a peak ammonia concentration of about 9 mmol/l was achieved, urea had disappeared and lactose concentration had fallen to 3·5 mmol/l; lactose had disappeared after 2 h. For lactosyl urea, ammonia concentration remained below 1 mmol/l and the compound, identified and measured chromatographically, survived to extents of 0·75 and 0·50 g/g after 2 and 4 h respectively.

Although adaptation to continued feeding of glycosyl ureides needs to be studied (Milligan *et al.* (1972) reported appreciable increases in ammonia release from glucosyl urea after adaptation), it seems likely that these compounds would be of value in ruminant feeding because they release ammonia slowly and, at the same time, provide a supply of energy for microbial growth. Glucosyl urea is expensive and unlikely to be of practical value but we have examined the possibility of preparing a product rich in lactosyl urea directly from whey.

Urea (96 g) was added to 2 l rennet-precipitated whey, sulphuric acid was added to a final concentration of 0·2 M and the mixture was kept at 50° for 7 d. Lactosyl urea (m.p. 232–233°) was prepared from the reaction mixture by evaporating down

and recrystallizing from water. Chromatography of the mixture showed that 70% of the lactose in the whey was converted to the ureide. It is proposed to assess the value of the total product from whey as a nitrogenous supplement in ruminant feeding.

REFERENCES

Hynd, A. (1926). *Biochem. J.* **20**, 205.
Milligan, L. P., Worsley, M., Elofson, M., Young, B. A. & Atwal, A. S. (1972). *J. Anim. Sci.* **34**, 906.
Smith, R. H. & McAllan, A. B. (1971). In *Automation in Analytical Chemistry : Technicon Symposium, London* 1969, p. 20. Tarrytown, N.Y.: Technicon Instruments Corporation.

The turnover rate of muscle and liver protein in sheep. By P. J. BUTTERY, A. BECKERTON and R. M. MITCHELL, *Department of Applied Biochemistry and Nutrition, University of Nottingham*, and K. DAVIES and E. F. ANNISON, *Unilever Research Laboratory, Colworth House, Sharnbrook, Bedford*

In an attempt to quantify certain aspects of amino acid metabolism in sheep we have investigated the fractional synthetic rate of protein in various muscle tissues and the liver of mature wethers (40–50 kg). L-[4, 5-^{3}H]lysine was continuously infused into the jugular vein for 12 h. Blood samples were taken at intervals for the measurement of the specific radioactivity of lysine. The animal was then killed, and the specific radioactivity of the intracellular and the protein-bound lysine was determined in liver, diaphragm, heart and the longissimus dorsi and gastrocnemius muscles by preparative ion-exchange chromatography. Care was taken to separate the lysine from the *N*-ε-monomethyl-lysine normally present in the intracellular fluid (see Beckerton, Buttery, Bailey & Bolton, 1974). The change in the specific radioactivity of the plasma lysine during the infusion was also monitored.

The procedure developed for rats by Waterlow & Stephen (1968) was used to calculate the fractional synthetic rate of the mixed protein in each tissue. Results (mean values with their standard errors where given; no. of samples in parentheses) were:

Tissue	Fractional rate of protein synthesis (/d)		Half-life (d)
	Mean	SE	
Liver	0·101	0·036 (3)	6·9
Diaphragm	0·022	0·003 (4)	31·5
Heart	0·032	0·016 (3)	21·6
Longissimus dorsi	0·018	0·006 (4)	38·5
Gastrocnemius	0·017	0·005 (9)	40·7

The alternative method of calculation of Garlick, Millward & James (1973) gave almost identical results. If the mean rate obtained for the longissimus dorsi and the gastrocnemius muscles is representative of all skeletal muscle, then in a 40 kg sheep, 68 g skeletal muscle protein is synthesized and degraded daily (assuming 47% of the

body is muscle and 20% of the muscle is protein). Similar calculations in the liver suggest that 23 g protein is synthesized and degraded daily (assuming 1·1 kg liver contains 0·2 g protein/g).

Analysis of the time-course of the increase of specific radioactivity of plasma lysine showed that the flux of lysine through the plasma pool of a 40 kg sheep was 64 ± 17 (8) mmol/d. The lysine reaching the duodenum in sheep given an identical diet was found in other experiments to be 75 ± 11 (4) mmol/d.

Although the increase in the specific radioactivity (disintegrations/min per μmol) of the lysine in the plasma could approximately be described by a single exponential (see Garlick *et al.* 1973), it was better defined by three exponential terms:
$y = -13{\cdot}3 \times 10^3(1-e^{-0.865t}) + 43{\cdot}4 \times 10^3(1-e^{-0.0670t}) + 415{\cdot}2 \times 10^3(1-e^{-0.00254t})$; (t in min).

A.B. and R.M.M. wish to thank the SRC (UK) for CASE Studentships.

REFERENCES

Beckerton, A., Buttery, P. J., Bailey, F. J. & Bolton, N. (1974). *J. Chromat.* (In the Press.)
Garlick, P. J., Millward, D. J. & James, W. P. T. (1973). *Biochem. J.* **136**, 935.
Waterlow, J. C. & Stephen, J. M. L. (1968). *Clin. Sci.* **35**, 287.

The optimal time for enrichment of dietary protein with tryptophan in relation to feeding time in the rat. By H. RAFALSKI, E. JABLONSKI and W. PONOMARENKO, *Department of Human Nutrition, Institute of Social Medicine, Medical Academy of Lodz, 83 Zachodnia, Lodz, Poland*

The biological effects of tryptophan enrichment of the diet were determined. Tryptophan was administered to rats at various times in relation to the time of intake of food. The effect of tryptophan on the nutritional value of the diet was assessed by measuring net protein utilization (NPU), energy supplied by utilizable protein: total metabolizable energy (NDP:E), food intake, body-weight gain, and chemical composition of the bodies of the rats.

Groups of four rats of the Wistar strain aged 30 ± 1 d were given, for 15 d, a basic diet with NDP:E 0·184. The composition (g/kg) was: maize grits 522, gelatin 80, sodium caseinate 72, sucrose 122, pork lard 122, L-methionine 2, mixture of vitamins and mineral salts 80. The limiting amino acid of this diet was tryptophan. The chemical score in relation to the ideal protein, as recommended by Bender (1965), was calculated according to data from the literature to be 50%.

The basic diet was given to the rats twice daily for 30 min, from 09.00 to 09.30 hours and from 20.00 to 20.30 hours. Tryptophan enrichment was given in an aqueous solution in amounts that raised the tryptophan content of the diet to 10 g L-tryptophan/kg protein, equivalent to Bender's (1965) standard.

Rats were given the following dietary treatments: (1) basic diet; (2) basic diet + tryptophan administered 1 h before intake of diet; (3) basic diet + tryptophan

administered at the same time as the diet; (4) basic diet + tryptophan added 1 h after intake of diet; (5) basic diet + tryptophan added 2 h after intake of diet.

The results are given in Table 1. The administration of tryptophan 1 h after the meals gave the highest weight gain and NPU.

Table 1. *Effect of tryptophan supplements on net protein utilization* (NPU), *energy supplied by utilizable protein : total metabolizable energy* (NDp : E) *and relative body-weight gain in rats*

(Mean values with their standard errors for groups of twelve rats)

Treatment no.	Tryptophan given	NPU		NDp:E		Body-weight gain relative to control (g)
		Mean	SE	Mean	SE	
1	None	0·335	0·018	0·062	0·0004	—
2	1 h before diet	0·345	0·014	0·063	0·0004	+39·4
3	With diet	0·408	0·015	0·075	0·0004	+53·4
4	1 h after diet	0·455	0·015	0·084	0·0004	+87·0
5	2 h after diet	0·419	0·015	0·077	0·0004	+61·4

REFERENCE

Bender, A. E. (1965). *Proc. Nutr. Soc.* **24**, 190.

Influence of prolonged ingestion of some organochlorated pesticides on digestibility and nutritive value of a diet in rats. By G. VARELA, AMALIA TORRALBA and J. ESCRIVÁ, *Nutrition Department, Patronato 'Juan de la Cierva', C.S.I.C. Veterinary Faculty, Madrid* 3, *Spain*

Recently, in 1973 at the Symposium of the American Institute of Nutrition, dealing with the action of organochlorate molecules in nutrition and metabolism, Sell & Davison (1973) pointed out changes in the activity of certain microsomatic hepatic enzymes due to the action of various organochlorate pesticides, and Davis (1973) suggested the advisability of studying the possible effect that these contaminants might exercise on the nutrients of a diet.

We studied the effect of three pesticides: Lindane, DDT and Dieldrin, using one dose of Lindane (10 mg/kg) and two doses of the other two (0·5 and 10 mg/kg) added to the diet. In all instances we compared the results obtained with those for a group of controls consuming a diet with the same characteristics but without a contaminant. For each one of the doses under study, we used one group of ten Wistar rats, male and female, with the same initial characteristics of age and weight. Each group consumed after weaning a laboratory-prepared diet, containing in the instance of the test groups, the pesticides in the corresponding doses. These conditions were maintained for 3 months, until an average body-weight of 250 g had been reached. At this point digestibility and nitrogen balance tests were carried out, to determine the digestibility ratios of the dry matter, organic matter, protein (true and apparent), fat, crude fibre, N-free extracts, and net protein utilization (NPU) and total digestible nutrients (TDN) (Table 1).

Table 1. *Digestibility ratios of diets containing organochlorate pesticides given to rats*

(Mean values for groups of ten animals)

	Lindane		DDT			Dieldrin		
	Control	10 mg/kg	Control	0·5 mg/kg	10 mg/kg	Control	0·5 mg/kg	10 mg/kg
Protein								
True digestibility	0·834	0·846	0·820	0·811	0·812	0·799	0·778	0·800
Apparent digestibility	0·799	0·809	0·773	0·778	0·773	0·767	0·743	0·769
Fat	0·943	0·948	0·888	0·850	0·827	0·880	0·860	0·832
Crude fibre	0·085	0·087	0·080	0·083	0·085	0·087	0·091	0·086
Nitrogen-free extract	0·884	0·888	0·855	0·853	0·849	0·887	0·883	0·888
Net protein utilization*								
True	0·385	0·403	0·412	0·380	0·406	0·371	0·373	0·374
Apparent	0·225	0·231	0·295	0·278	0·300	0·229	0·253	0·237
Total digestible nutrients	87·2	87·6	78·0	77·7	77·1	80·8	79·8	80·3

*Determined by N balance method: Apparent, not corrected for endogenous N losses.

Our results show that with 0·5 mg DDT/kg the digestibility of fat diminished significantly ($P<0·01$). With 10 mg DDT/kg, the decrease was greater and significant compared with the controls ($P<0·001$) and compared with the dose of 0·5 mg/kg ($P<0·05$). Dieldrin had a similar effect; the presence of 0·5 mg/kg caused a decrease in the digestibility of fat ($P<0·01$), and on increasing the content of the pesticide to 10 mg/kg, this effect was greater when compared with the controls ($P<0·001$) and with the level of 0·5 mg/kg ($P<0·01$). Lindane (10 mg/kg) had no action on digestibility of fat.

We observed no effect on digestibility of the remaining nutrients, NPU and TDN.

REFERENCES

Davis, G. K. (1973). *Fedn Proc. Fedn Am. Socs exp. Biol.* **32**, 1993.
Sell, J. L. & Davison, K. L. (1973). *Fedn Proc. Fedn Am. Socs exp. Biol.* **32**, 2003.

A comparison of the apparent digestibility of amino acids from measurements of digesta in the terminal ileum and of faeces in growing pigs.

By A. G. Low, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

In view of the role of the microflora of the large intestine in metabolizing certain nutrients, it has been suggested that the apparent digestibility of nutrients may be better estimated from measurements of digesta in the terminal ileum than of faeces (Payne, Combs, Kifer & Snyder, 1968). Comparisons between these sites for the major nutrient groups have been reported (Braude, Low, Partridge & Sambrook, 1975). Six pigs with re-entrant cannulas in the terminal ileum were fed in turn on

barley, weatings and fish meal (diet A) and starch, sucrose, maize oil and casein (diet B). Digesta were collected during four 24 h periods from each pig on each diet. Faeces were collected during four 5 d periods, from six pigs (without cannulas) per diet.

The apparent digestibility of all amino acids was lower for diet A than diet B, at both sites. The results suggest that for most essential amino acids, apparent digestibility measurements in faeces are of similar value to those in ileal digesta.

Of the essential amino acids in diet A, only histidine was apparently absorbed in the large intestine (apparent digestibility $0{\cdot}81 \pm 0{\cdot}030$ in ileal digesta, $0{\cdot}87 \pm 0{\cdot}014$ in faeces); in all other instances the values were similar or lower in faeces than ileal digesta, suggesting that microbial transformation occurred. A similar situation was found for non-essential amino acids except for proline and glycine ($0{\cdot}83 \pm 0{\cdot}014$ and $0{\cdot}72 \pm 0{\cdot}032$ in ileal digesta and $0{\cdot}88 \pm 0{\cdot}002$ and $0{\cdot}83 \pm 0{\cdot}004$ in faeces, respectively); the latter are important components of mucus and bile respectively, which are known to be digested to some extent in the large intestine.

Of the essential amino acids in diet B, only threonine was apparently absorbed in the large intestine (apparent digestibility $0{\cdot}91 \pm 0{\cdot}008$ in ileal digesta, $0{\cdot}98 \pm 0{\cdot}004$ in faeces). The only non-essential amino acids apparently absorbed in the large intestine were serine and glycine (apparent digestibility $0{\cdot}91 \pm 0{\cdot}008$ and $0{\cdot}79 \pm 0{\cdot}022$ in ileal digesta, and $0{\cdot}98 \pm 0{\cdot}004$ and $0{\cdot}91 \pm 0{\cdot}020$ in faeces, respectively). There was no evidence of microbial transformation in the large intestine (cf. diet A): this may have been because of the smaller amount of digesta and the almost complete digestion and absorption of protein anterior to the terminal ileum for diet B.

REFERENCES

Braude, R., Low, A. G., Partridge, I. G. & Sambrook, I. E. (1975). *Proc. Nutr. Soc.* **34**, 46A.
Payne, W. L., Combs, G. F., Kifer, R. R. & Snyder, D. G. (1968). *Fedn Proc. Fedn Am. Socs exp. Biol.* **27**, 1199.

The rates of tissue protein synthesis in vivo in hypophysectomized and pair-fed control rats. By D. J. MILLWARD, D. O. NNANYELUGO and A. K. CHATTERJEE, *Clinical Nutrition and Metabolism Unit, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*

The growth failure in hypophysectomized rats is not fully understood. We have therefore measured the rates of tissue protein synthesis in vivo in muscle, liver, heart and kidney in hypophysectomized rats and in normal rats pair-fed or fed *ad lib.* In this way we can evaluate separately the effects of the operation and the reduced food intake.

Male Wistar rats were hypophysectomized when they weighed 100 g, having been purchased together with unoperated controls. The groups of control rats were either pair-fed the food intake of the hypophysectomized rats or fed *ad lib.* After 7–10 d, rates of tissue protein synthesis were measured by the constant-infusion method (Garlick, Millward & James, 1974) in all three groups. Results are shown in Table 1.

Table 1. *Tissue protein synthesis rate, RNA concentration and synthetic efficiency in hypophysectomized rats and in control rats pair-fed to the test animals or fed* ad lib.

(Mean values and standard deviations)

Tissue	Treatment	Synthesis rate (/d) Mean	SD	RNA:protein ratio ($\times 10^3$) Mean	SD	Synthetic efficiency (g protein/g RNA per d) Mean	SD
Muscle	Fed *ad lib.*	0·121	0·022	6·83	0·55	17·73	3·21
	Hypophysectomized	0·058	0·013	8·91	0·63	6·81	1·53
	Pair-fed	0·088	0·014	9·87	1·68	8·81	1·45
Liver	Fed *ad lib.*	0·489	0·086	52·42	7·20	9·55	2·56
	Hypophysectomized	0·574	0·138	43·82	2·49	13·03	2·76
	Pair-fed	1·071	0·170	52·90	4·30	20·26	3·86
Heart	Fed *ad lib.*	0·196	0·044	12·26	1·09	16·12	3·09
	Hypophysectomized	0·127	0·013	12·42	0·95	10·30	0·50
	Pair-fed	0·133	0·022	12·30	0·56	10·81	1·55
Kidney	Fed *ad lib.*	0·508	0·097	27·04	1·61	18·76	3·11
	Hypophysectomized	0·283	0·051	30·82	1·92	9·38	2·50
	Pair-fed	0·471	0·048	33·40	2·05	14·20	2·20

The reduced food intake in the operated rats was illustrated by the growth failure in the pair-fed rats. Furthermore, the pattern of protein synthesis in the pair-fed control rats was typical of malnourished rats with reduced synthesis rates in muscle and heart and an increased rate in liver. In the operated rats there were further reductions in synthesis rates in muscle and kidney, but the rates of liver protein synthesis were nearly normal.

REFERENCE

Garlick, P. J., Millward, D. J. & James, W. P. T. (1974). *Biochem. J.* **136**, 935.

Effect of protein deficiency on glucagon–stimulated insulin secretion, and on glycogen storage and release in rabbits. By K. A. ALLEN, C. E. AYRES, K. A. MUNDAY and M. R. TURNER, *Department of Physiology and Biochemistry, The University, Southampton SO9 3TU*

Glucagon has two important metabolic roles: (a) the stimulation of insulin secretion during a meal and (b) the maintenance of the blood glucose level during times of nutrient need, by the stimulation of glycogenolysis and gluconeogenesis. Protein-energy malnutrition has profound effects both on hormone secretion and on hormone action.

New Zealand White rabbits were fed from weaning on diets containing 200 g (HP) or 100 g (LP) protein derived from isolated soya-bean protein/kg. The total food intake was similar in both groups, but in LP animals growth was impaired. After 12 weeks on the LP diet, insulin secretion in response to intravenous glucagon (20 ng/kg body-weight) was impaired, the peak insulin value being reduced and delayed.

At this time the insulin secretory response to both glucose and amino acids is completely lost (Turner, Allen & Munday, 1974). After 18 weeks on diet, the fasting insulin level in LP animals was significantly reduced and the secretory response to glucagon was completely lost. Circulating growth hormone levels are normal after 12 weeks on the LP diet, but become elevated after 18 weeks on diet (Turner *et al.* 1974). The plasma insulin values (μU/ml) ± SE for six HP and five LP rabbits following glucagon administration were:

	12 weeks on diet				18 weeks on diet			
Time after glucagon (min)	HP		LP		HP		LP	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
0	5·9	0·9	6·9	1·3	14·8	0·8	3·2***	0·6
1	35·0	2·8	8·3***	1·2	42·0	11·2	4·4*	0·8
2	74·3	9·0	15·3***	2·5	86·8	15·2	5·0***	1·0
4	22·5	2·1	17·8	1·8	38·5	9·8	4·3*	1·0
8	9·5	3·6	42·8**	5·0	21·5	4·2	4·0**	1·0
16	9·7	2·2	16·5	4·9	15·7	2·3	4·8**	0·9

Significance of difference from HP: $*P<0.05$, $**P<0.01$, $***P<0.001$.

There was no difference between the dietary groups in the plasma glucose increment following glucagon administration. After 18 weeks on diet, liver from fed LP animals contained more glycogen than that from fed HP animals (HP 52, LP 93 mg/liver; $P<0.05$) despite a 35% reduction in liver weight in LP rabbits, but after a 20 h overnight fast, there was little detectable glycogen in liver from either dietary group. Therefore, in these protein-deficient animals, the insulin secretory response to glucagon stimulation was progressively lost, but the ability of the liver to store glycogen and to release glucose both in response to glucagon stimulation, and during a fast, was unimpaired throughout.

REFERENCE

Turner, M. R., Allen, K. A. & Munday, K. A. (1974). *Proc. Nutr. Soc.* **33**, 56A.

Sex difference in muscle growth after undernutrition during the sucking period. By J. P. G. Williams and P. C. R. Hughes (introduced by P. A. McAnulty), *Department of Growth and Development, Institute of Child Health, Guilford Street, London WC1N 1EH*

Black-hooded rats were reared in litters of eight or sixteen pups; at 21 d the pups were weaned and given unlimited access to food and water. The calf muscle width was measured from serial radiographs as previously described (Williams & Hughes, 1975). A total of 410 observations were made between the age of 3 d and 228 d. The sixteen-pup group was termed undernourished and the eight-pup group, controls. A sex difference was apparent in the growth velocity curves, the males being more

affected than the females by the undernutrition and the females showing a greater and earlier catch-up velocity on rehabilitation. In females the muscle length (=tibia length) had caught up to the control group by 70 d but in the males the length was still slightly but significantly less even at 228 d. Muscle width in the females reached 98% of the control group by 65 d and was not significantly different from control values at 228 d. In the males, however, the muscle width was 96% of the control group at day 65 and was still significantly different ($P<0{\cdot}001$) on day 228. At the end of the experiment the females had caught up in body-weight but the males were still significantly lighter than their controls. The degree of catch-up observed in the male body-weight is less than that seen in muscle width.

This work was supported by a long-term grant to Professor J. M. Tanner from the Medical Research Council.

REFERENCE

Williams, J. P. G. & Hughes, P. C. R. (1975). *Proc. Nutr. Soc.* **34**, 39A.