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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Nineteenth Meeting of the Nutrition Society was held at the University of Southampton on Thursday and Friday, 20/21 July, 1978 when the following posters were presented:

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Differences in hepatic extraction of lactate in lactating and non-lactating dairy cows. By G. D. BAIRD, M. A. LOMAX*, H. W. SYMONDS and S. R. SHAW, *ARC Institute for Research on Animal Diseases, Compton, Berkshire RG16 0NN*

Use of dairy cows (Friesian×Ayrshire) in which catheters had been permanently implanted in appropriate blood vessels by the method of Symonds & Baird (1973) has led to the observation that there are differences between lactating and non-lactating cows in the ability of the liver to extract lactate from the blood in vivo.

Mean percentage hepatic extraction of lactate in one pass, calculated as $[(F_p C_p + F_a C_a) - F_h C_h]/(F_p C_p + F_a C_a) \times 100$ where F_p, F_a and F_h are the whole-blood flow rates (l/min) in the portal vein, hepatic artery and hepatic veins, respectively, and C_p, C_a and C_h are the lactate concentrations in whole blood (mmol/l) at these locations, was found to be 29·6 and 8·9 for 5 fed lactating cows and 4 fed non-lactating cows respectively (SED 4·7; $P<0.01$). Rates of arrival of lactate at the liver were very similar in these same cows, however, (10·05 and 10·25 mmol/min respectively, SED 0·95).

It was also observed that the magnitude of hepatic extraction of lactate could be readily altered. Increase in hepatic extraction in the non-lactating cows could be achieved by food deprivation, while decrease in extraction in the lactating cows could be achieved by glucose infusion (see Table).

Percentage hepatic extraction of lactate during food deprivation and glucose infusion

(2 lactating and 2 non-lactating cows were deprived of food for 144 h and 3 lactating and 3 non-lactating cows were infused intravenously with glucose at 4·2 mmol/min for 48 h)

	Duration of food deprivation (h)				Duration of glucose infusion (h)		
	0	48	144	SED	0	48	SED
Lactating cows	26·3	55·8***	34·4	3·7	30·0	10·3**	6·0
Non-lactating cows	9·3	46·6***	43·4***	3·7	7·2	5·4	6·0

**$P<0.01$; ***$P<0.001$ compared with 0 h.

The difference in hepatic extraction of lactate could arise from variation in hormonal environment. Thus, changes in the concentration ratio, glucagon:insulin, might affect the ability of the liver to extract lactate, perhaps by means of alterations in the activity of pyruvate carboxylase (Brockman & Manns, 1974). The greater extraction of lactate in the lactating cows in the fed state might reflect a greater need to maintain the hepatic content of glycogen and of intermediates of the tricarboxylic acid cycle (c.f. Baird & Heitzman, 1970).

M.A.L. acknowledges receipt of an Agricultural Research Council studentship.

Baird, G. D. & Heitzman, R. J. (1970). *Biochem. J.* 116, 865.
Brockman, R. P. & Manns, J. G. (1974). *Cornell Vet.* 64, 217.
Symonds, H. W. & Baird, G. D. (1973). *Res. Vet. Sci.* 14, 267.

*Present address: Biological Laboratory, The University, Canterbury, Kent CT2 7NJ.

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Differences in the effect of glucose infusion on insulin and glucose production in lactating and non-lactating dairy cows. By M. A. Lomax*, G. D. Baird, H. W. Symonds, C. B. Mallinson and S. R. Shaw, *ARC Institute for Research on Animal Diseases, Compton, Newbury, Berkshire RG16 0NN*

Three lactating and three non-lactating dairy cows (Friesian × Ayrshire), that had been catheterized by the procedure of Symonds & Baird (1973) and were being maintained under normal husbandry conditions, were infused via the jugular vein with a 50% (w/v) aqueous solution of glucose at a constant rate of 4·2 mmol/min (1·5 ml/min) for 48 h. Two cows from each group were also given control infusions of water. Arterial concentrations of insulin and glucose, portal production of insulin and hepatic production of glucose (see Baird, Symonds & Ash, 1975) were determined before infusion, and after the infusion had been in progress for 4, 24 and 48 h.

		Duration of glucose infusion (h)				
Sampling times (h):		0	4	24	48	SED
Lactating cows:						
Insulin	concentration (mU/l)	16·5	36·2	17·7	18·9	18·6
	portal production (mU/min)	+23·8	+247·7	+89·0	+101·7	214·7
Glucose	concentration (mM)	2·9	4·3***	3·4*	3·3	0·2
	hepatic production (mmol/min)	+6·5	+1·1***	+2·6**	+4·2	1·1
Non-lactating cows:						
Insulin	concentration (mU/l)	37·0	130·1***	145·6***	145·7***	18·6
	portal production (mU/min)	+198·2	+553·7	+947·8**	+850·1*	214·7
Glucose	concentration (mM)	3·3	3·9*	3·8*	3·9*	0·2
	hepatic production (mmol/min)	+3·6	+3·2	+1·4	+4·8	1·1

*$P<0·05$; **$P<0·01$; ***$P<0·001$ compared with 0 h.

The table demonstrates that the insulin response to glucose infusion is far less in lactating than in non-lactating animals. Water infusions caused no significant changes in either groups of animals.

The differences in insulin response to glucose infusion may be determined by the necessity in the lactating cow to direct metabolites towards milk synthesis rather than towards incorporation into body tissue.

M.A.L. acknowledges receipt of an Agricultural Research Council Studentship.

Baird, G. D., Symonds, H. W. & Ash, R. (1975). *J. agric. Sci., Camb.* 85, 281.
Symonds, H. W. & Baird, G. D. (1973). *Res. Vet. Sci.* 14, 267.

*Present address: Biological Laboratory, The University, Canterbury, Kent CT2 7NJ.

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Protein synthesis in cattle. By G. E. LOBLEY, P. J. REEDS and K. PENNIE,
Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB

Two 10-month old Hereford–Friesian heifers and an 8-year-old dry cow were used to determine the rates of protein synthesis in the main tissues of the body. Their weights were 236, 263 and 628 kg respectively and all were given 2×2 kg of concentrate and 2×1 kg of hay daily. On the day before the experiment catheters were inserted in both external jugular veins. Through one of these a solution containing L-[4,5-^{3}H]leucine and either L-[2,3 sidechain-^{3}H]tyrosine or L-[2,6 ring-^{3}H]tyrosine was infused for 8 h. The rates of infusion were 0·4 μCi/kg per h for leucine and 2 μCi/kg per h for tyrosine. Blood samples (20 ml) were withdrawn at regular intervals from the other catheter. At the end of the infusions the animals were killed with sodium pentobarbitone and immediately pieces were excised of hide, liver, intestine and m. longissimus dorsi and m. vastus lateralis. Blood and tissue samples were treated and analysed as described by Nicholas *et al.* (1977). Fractional synthesis rates (FSR) were computed from both the tissue ($k_{s,i}$) and blood ($k_{s,p}$) free amino acid specific radioactivities (SR). The remainder of the ingesta-free body was dissected and the amount of protein (N×6·25) in the main tissues determined.

After 3 h of infusion the SR of both free leucine and tyrosine in the blood was constant. One of the young animals (263 kg) showed signs of diarrhoea during the experimental period. This heifer had lower FSR for liver, intestine and hide than the other animal of similar age (see Table 1). The cow had lower FSR than the young animals.

Table 1. *FSR×100, $k_{s,i}$ ($k_{s,p}$), in main tissues*

Animal wt (kg)	Muscle	Intestine	Liver	Hide
A–236	1·8 (1·6)	53·2 (28·0)	38·5 (10·8)	7·4 (3·4)
B–263	2·0 (1·8)	47·2 (22·4)	24·2 (9·9)	4·7 (3·2)
C–628	0·9 (0·8)	27·9 (14·2)	14·4 (7·3)	3·9 (1·4)

The products of the protein content and FSR ($k_{s,i}$) for the 4 tissues of each animal were summed ($A_{s,i}$). The amounts were 2140, 1760 and 2800 g/d for animals A, B and C respectively, that is considerably in excess of likely net accretion rates. The four tissues each contributed a reasonably fixed proportion to $A_{s,i}$ in all 3 animals: muscle was 15–21% intestine (including rumen) 48–61%; liver 7–10%; and hide 16–27%. Total protein synthesized in the 4 tissues calculated with $k_{s,p}$ resulted in amounts 54–58% of the corresponding $A_{s,i}$ values. Estimates of protein synthesis obtained from the plasma amino acid flux were different for leucine and tyrosine, the value for leucine being closer to $A_{s,p}$ and for tyrosine closer to $A_{s,i}$. Amino acid oxidation calculated from the excretion of urea and ammonia in the urine was 13%, 13% and 15% respectively of the total leucine flux.

Nicholas, G. A., Lobley, G. E. & Harris, C. I. (1977). *Br. J. Nutr.* 38, 1.

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Fat induced hypertension in rabbits. 1. The effects of fibre on the blood pressure increase induced by coconut oil. By TAKLA GARDEY, P. G. BURSTYN and T. G. TAYLOR, *School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU*

Previous work has shown that fat-enriched diets can elevate the blood pressure of rabbits (Burstyn *et al.* 1972; Burstyn & Firth, 1975). The effects of dietary fibre on blood pressure have been investigated.

Twenty rabbits were weaned onto a diet containing about (g/kg) 30 fat (as naturally occurring fats in barley meal and soya-bean meal, and 1% added corn oil), 200 wheat bran and 100 cellulose (Solkafloc). The arterial blood pressures were measured daily by means of an ear capsule. The animals were maintained on the diet for 5 weeks before the start of the experiment, blood pressures being determined for the final 2 weeks of this control period. The mean blood pressure of each animal in the final week was taken as the control value.

The animals were divided into four groups of five and given modifications of the control diet: group A, no cellulose and 200 g coconut oil/kg; group B, 200 g cellulose and 200 g coconut oil/kg; group C, no cellulose and no added fat; group D, 200 g cellulose/kg and no added fat. Adjustments were made to the protein and carbohydrate contents of the diets to maintain a constant ratio protein: metabolizable energy (1.5 g protein: 100 kJ energy).

Arterial pressure following feeding of diets enriched in fat and/or cellulose, expressed as per cent of control ± standard error

(Statistical significance determined by paired *t* test)

	Period on diet (weeks)							
Group	Control	1	2	3	4	5	6	7
A	100±4	105±4	111±3	119±6	116±3	123±3	125±3	122±3
			$P<0.05$	$P<0.05$	$P<0.01$	$P<0.01$	$P<0.01$	$P<0.01$
B	100±3	98±4	102±3	102±2	105±2	106±3	105±4	108±3
								$P<0.02$
C	100±4	96±4	95±4	102±5	103±5	100±5	103±4	100±12
D	100±2	97±2	98±5	97±6	105±6	104±6	95±2	—

The results confirm that fat-enriched diets (group A) can increase blood pressure in rabbits. The addition of cellulose (group B) can delay and diminish this effect. The added cellulose in the absence of added dietary fat has no effect upon the blood pressure (compare groups C and D).

Burstyn, P. G., Horrobin, D. F. & Muiruri, K. M. (1972). *Br. J. Exp.Path.* 53, 258.
Burstyn, P. G. & Firth, W. R. (1975). *Cardiovascular Res.* 9, 807.

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Fat induced hypertension in rabbits. 2. The effect of feeding diets containing high concentrations of safflower oil and palm oil. By MARION KENNEDY, P. G. BURSTYN and D. R. HUSBANDS*, *School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU*

The effect of fats containing high levels of linoleic acid, safflower oil and high levels of palmitic and oleic acid, palm oil on blood pressure in rabbits was investigated.

Growing New Zealand White rabbits were maintained on a low-fat diet for 2 weeks, during which time blood pressure was measured daily (mean 66 mm Hg). Serum cholesterol (mean 3·54 mmol/l) and serum triglyceride (mean 0·87 mmol/l) were determined after an overnight fast. The rabbits were divided into groups. Four animals (A) were given a diet containing 200 g safflower oil/kg, five animals (B) were given a diet containing 200 g palm oil/kg and five animals (C) were given a diet with 200 g palm oil/kg and 200 g cellulose/kg (Solkafloc). The protein and energy contents were adjusted to allow for these ingredients (Gardey *et al.* 1978). Blood pressure was recorded and serum samples were collected after overnight fasting for triglyceride and cholesterol determinations.

Feeding palm oil (B and C) but not safflower oil (A) elevated the plasma concentration of cholesterol during the first 3 weeks but after 7 weeks plasma cholesterol levels were raised in all groups. Triglycerides fell in all groups but least in that given cellulose (C). All these high-fat diets caused an increase in blood pressure above that recorded on the low-fat diet but the increase was greater with palm oil than with safflower oil and the addition of cellulose with the palm oil lessened the rise. The greatest change in serum lipid values resulted in the greatest change in blood pressure.

Changes in serum cholesterol, serum triglycerides and blood pressure in rabbits given diets containing 200 g safflower oil and 200 g palm oil/kg expressed as a percentage of the pre-experimental values

	Cholesterol		Triglyceride		Blood pressure	
Period on the diet (weeks)	1–3,	7	1–3,	7	3,	6–8
Group A safflower oil	85[a]	123	66[c]	59	108	122[e]
Group B palm oil	133[b]	134	74[c]	47	111	129[f]
Group C palm oil with added cellulose	139[b]	150	93[d]	71	110	121[e]

a differs from b $P<0{\cdot}01$; c differs from d $P<0{\cdot}05$; e differs from f $P<0{\cdot}05$

Gardey, T., Burstyn, P. G. & Taylor, T. G. (1978). *Proc. Nutr. Soc.* **37**, 97A.

*On leave from Department of Chemistry, Biochemistry and Biophysics, Massey University, Palmerston North, New Zealand.

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Growth, gut fill and hormone levels in pair-fed lambs under two daylengths. By WENDY B. BROWN, R. JONES and J. M. FORBES, *Department of Animal Physiology and Nutrition, University of Leeds LS2 9JT*

Long daylength stimulates growth in lambs fed either *ad lib.* or at an individually restricted level on a concentrate-based diet; carcass composition is not affected and weights of carcasses and gut contents are significantly greater at slaughter (El Shahat *et al.* 1974). Plasma prolactin levels are significantly elevated by long daylength (Forbes *et al.* 1975), whereas growth hormone is unaffected.

Three experiments have been carried out under similar conditions but with castrated male lambs pair-fed for 3 months according to the mean weight of animals in 16 and 8 h daylengths. In Expts 1 and 2 the sheep were 5 months of age at the start, while in Expt 3 they were 2·5 months.

	Expt					
	1		2		3	
Daylength (h light)	8	16	8	16	8	16
n	4	6	7	8	6	6
Gut fill at slaughter (kg)	3·8	4·3	4·9	6·0*	5·7	7·7**
Empty body-weight (kg)	46·8	46·5	43·0	44·1	30·0	30·5
Kidney and caul fat (kg)	1·24	0·94*	0·78	0·50	0·38	0·32
Proportion of muscle in three-rib joint	0·40	0·43	0·42	0·48	0·45	0·45
Plasma prolactin in last month (ng/ml)	50·3	71·9*	44·1	100·6**	51·1	133·3***

$^{*}P<0·05$; $^{**}P<0·01$; $^{***}P<0·001$.

The table shows that gut fill at slaughter was much greater in long daylength but that empty body weight was not affected. The carcasses were, however, leaner and tended to be longer and wider. Plasma prolactin was greatly elevated by long daylength, whereas growth hormone was unaffected.

There was thus an effect of daylength on lamb growth even with the same input of food. Most of the effect on live weight gain was in gut fill but the carcasses were slightly larger and significantly leaner.

El Shahat, A. A., Jones, R., Forbes, J. M. & Boaz, T. G. (1974). *Proc. Nutr. Soc.* **33**, 83A.
Forbes, J. M., Driver, P. M., El Shahat, A. A., Boaz, T. G. & Scanes, C. G. (1975). *J. Endocr.* **64**, 549.

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Plasma growth hormone and spontaneous meals in sheep. By P. M. DRIVER and J. M. FORBES, *Department of Animal Physiology and Nutrition, University of Leeds LS2 9JT*

There have been several reports that plasma levels of growth hormone (GH) of sheep are depressed following the consumption of newly offered food (e.g. Bassett, 1974; Driver, 1977). We have investigated whether plasma GH levels are related to spontaneous meals in castrate male lambs.

Two animals were housed in adjoining individual pens and fed *ad lib.* on a concentrate feed whose weight was continuously recorded. On two occasions blood samples were taken, via jugular cannulas, every 20 min for a minimum of 28 h. Ovine GH levels were measured by radioimmunoassay (Driver, 1977).

Graphs of plasma GH against time showed a regular pattern of GH secretion usually in the form of a double peak every 3·5–5 h. A difference between the two animals was apparent, one having low basal GH levels (4–6 ng/ml), but large sharp peaks (45–70 ng/ml), the other a higher basal level (7–15 ng/ml) but lower, broader peaks (40–50 ng/ml). The patterns of GH secretion from the same animal between the two experiments were almost identical. GH levels were high just before but fell when fresh food was offered and feeding took place.

Out of a total of 72 spontaneous meals from 4 experimental runs, 55 were associated with decreasing GH levels following a peak and only 17 with an increase in GH. Furthermore, at least 12 of these 17 meals could be attributed to 'social' feeding when the animal's neighbour was eating following a fall in GH. As GH levels increased during periods of non-eating and fell with eating, the evidence favours the view that ruminant GH is secreted in response to a decrease in the concentration of circulating metabolites. The rapid fall coincident with a meal (probably before absorption) indicates that a neural mechanism (Bassett, 1974) may be involved. The size of each GH peak did not appear to influence meal size or length.

We believe that these results, together with other evidence, strongly suggest a role for GH in the short term control of feeding in sheep, possibly as part of a 'trigger' mechanism, but it is likely that insulin is also involved.

We acknowledge the assistance of Mrs W. B. Brown and Dr J. Falconer.

Bassett, J. M. (1974). *Aust. J. Biol. Sci.* 27, 157.
Driver, P. M. (1977). PhD Thesis, University of Leeds.

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Acetate production by sheep hind–limb muscle. By D. W. PETHICK and D. B. LINDSAY, *Biochemistry Department, ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

Acetate present in ruminant blood is derived both from exogenous (fermentative) and endogenous (tissue) sources (Annison & Armstrong, 1970). Of the body tissues, only the liver has been thoroughly studied with reference to acetate release and it accounts for about 20% of the endogenous production in non-lactating fed or fasted sheep (Bergman & Wolff, 1971). We now report the contribution of skeletal muscle to endogenous acetate produced in the whole animal.

Clun Forest sheep (3 wethers and 3 lactating ewes) received a constant intravenous infusion of [U–^{14}C]acetate for 60 min, and towards the end of the infusion estimations were made of the concentration and radioactivity of acetate in arterial blood and venous blood draining predominantly from muscle (Domanski *et al.* 1974). Blood flow was measured immediately after the infusion. From these values acetate entry rate and muscle acetate exchange were calculated. Experiments were made either 3 h after receiving a maintenance ration of chopped hay of which about 80% had been eaten (fed animals) or 3 d after the last meal (fasted animals). Animals designated lactating had unrestricted feed intake.

A simultaneous uptake and release of acetate was always detected (Table 1). Fasting and lactation resulted in a significant decrease in acetate release from muscle. Since an increase in plasma free fatty acid concentration (Leat, 1974) and thus utilization by muscle is to be expected, there are probably sources of acetate other than incomplete oxidation of long-chain fatty acid. Assuming that the fraction of total acetate entry rate, which is of endogenous origin, is 0·2 in fed and 0·8 in fasted sheep (e.g. Bergman & Wolff, 1971) and that there is about 10 kg skeletal muscle, then this tissue could account for about 60% of endogenous acetate entry in fed and 30% in fasted animals.

Table 1. *Acetate concentration (mM), entry rate (mmol/h) and muscle exchange (nmol/min per g) in fed, fasted and lactating sheep*

State of animals	Arterial acetate	Acetate entry rate	Net muscle uptake	Muscle output
Fed	1·37±0·2	155±41	139·9±41·8	30·2±6·5
Starved	0·15±0·02*	26±5*	7·3±4·1*	10·8±1·7*
Lactating	2·0±0·5	284±72	72·6±8·4	12·3±2·4*

*Significantly different from fed; $P<0·05$.

Annison, E. F. & Armstrong, D. G. (1970). In *Physiology of Digestion and Metabolism in the Ruminant*. p. 422 [A. T. Phillipson, editor].
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Domanski, A., Lindsay, D. B. & Setchell, B. P. (1974). *J. Physiol., Lond.* **242**, 30.
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The effect of protein and carbohydrate in very low energy diets on thyroid hormone levels in overweight adults. By P. G. H. BYFIELD, M. L. DURRANT, D. BIRD, M. LAND, P. ROYSTON, R. L. HIMSWORTH and J. S. GARROW, *Clinical Research Centre, Harrow, Middlesex HA1 3UJ*

Energy restriction has been reported to be associated with a fall in serum 3,5,3′-triiodothyronine (T3) levels (Spaulding *et al.* 1976) and a rise in serum 3,3′,5′-triiodothyronine (reverse T3) levels whilst the precursor of both compounds, thyroxine (T4), maintains constant levels. Dietary carbohydrate has been implemented in these adjustments.

Eight overweight adults were admitted to a metabolic unit for 3 weeks on diets approved by the Northwick Park Hospital Ethical Committee.

During week 1 patients received a total of 3·35 MJ and 6 g protein nitrogen/d. The second and third week diets provided 0·84 MJ from either a protein source (6 g N) or carbohydrate (0·5 g N) daily. Four patients were given the protein diet in week 2 followed by carbohydrate in week 3 (group I) and four patients fed in the opposite sequence (group II). Fasting blood samples were taken on admission and at the end of each test week.

Serum thyroxine levels, which were in the lower half of the normal range, and thyrotrophin levels did not change throughout the 3 weeks. For the combined groups in week 1, T3 fell ($P<0·05$) ($2·26\pm0·29$ to $1·93\pm0·37$ nmol/l) and reverse T3 rose ($P<0·01$) ($0·14\pm0·04$ to $0·22\pm0·04$ nmol/l).

The two crossover weeks were analysed for both sequence and dietary effects. T3 was significantly lower ($P<0·01$) at the end of the protein week (group I week 2, $1·26\pm0·28$ nmol/l; group II week 3, $1·35\pm0·55$ nmol/l) compared with the end of the carbohydrate week (group II week 2, $1·57\pm0·61$ nmol/l; group I week 3, $1·74\pm0·41$ nmol/l). The sequence effect was not significant.

All reverse T3 changes were in the opposite direction to T3 changes. However, the sequence effect was stronger than the dietary; reverse T3 was significantly higher ($P<0·05$) at the end of week 2 (group I week protein, $0·33\pm0·09$ nmol/l; group II week carbohydrate, $0·27\pm0·03$ nmol/l) than at the end of week 3 (group II week protein, $0·24\pm0·04$ nmol/l; group I week carbohydrate, $0·23\pm0·04$ nmol/l). Reverse T3 was higher at the end of the protein week compared with the carbohydrate week but did not reach the 5% significance level, possibly due to small sample size.

These results confirm an effect of energy restriction on levels of circulating T3 and reverse T3 but the direction and magnitude of the effects are related to dietary composition at low levels of energy intake.

Spaulding, S. W., Chopra, I. J., Sherwin, R. S. & Lyall, S. S. (1976). *J. clin. Endocr. Metab.* **42**, 197.

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The quality of protein in the digesta entering the small intestine of sheep.

By I. L. Johnson*, A. Thompson and D. G. Armstrong, *Department of Agricultural Biochemistry, University of Newcastle upon Tyne, Newcastle upon Tyne NE1 7RU*

The quality of microbial protein harvested from the rumen has been shown to exhibit little variation (Bird, 1972). No such studies have been reported on the protein of whole digesta entering the duodenum, yet for the host animal, it is this product whose over-all protein quality is of importance since amino acids available to the body arise from microbial protein, undegraded feed protein and endogenous secretions.

To investigate possible variations in the quality of whole digesta protein, digesta was collected from the proximal duodenum of sheep given isonitrogenous diets comprising equal parts of rolled barley and chopped hay supplemented with soya-bean meal (SBM) or isobutylidene diurea (IBDU). The digesta was freeze-dried, ground and used as the sole protein source in four purified diets for rats. Diets D1 and D2 contained the digesta from sheep given the SBM and IBDU supplemented diets respectively. The third and fourth diets were similar to D1, but with added methionine. These four diets plus 10 g/kg and 70 g/kg albumin diets were fed to 24 male, 6 week old Wistar rats for a 7 d balance trial, to determine biological value (BV), true digestibility of nitrogen (TDN) and net protein utilization (NPU) of the duodenal protein; there was a 7 d preliminary feeding period.

Diet	TDN	BV	NPU
70 g albumin/kg	97·9[a]	99·2[a]	97·1[a]
D1	75·9[b]	51·3[b]	39·4[b]
D2	82·9[c]	60·1[c]	49·8[c]
D1+1·67 g methionine/kg	76·8[b]	74·3[d]	57·1[d]
D1+2·33 g methionine/kg	76·4[b]	71·9[d]	54·9[d]
SEM	0·78	1·27	1·37

Values in each column with different superscripts are significantly different, $P<0·05$.

The organic matter apparently fermented in the rumen of sheep given SBM and IBDU supplemented diets was measured, and, using data from Miller (1973), Broster & Oldham (1977) and Mathers & Miller (1977), it was calculated that the proportion of microbial N in duodenal N was 0·662 in D1 and 0·799 in D2; undegraded feed N as a proportion of the total N was 0·257 in D1 and 0·106 in D2. If vegetable feed N escaping ruminal degradation has a lower BV than microbial N this may explain the higher 'quality' of the digesta from sheep given the IBDU supplement. The results also demonstrate the probability that S-amino acids are limiting in digesta of sheep given the SBM-supplemented diet.

I.L.J. was in receipt of an MLC postgraduate studentship.

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Broster, W. H. & Oldham, J. D. (1977). In *Nutrition and the Climatic Environment* [W. Haresign, H. Swan and D. Lewis, editors]. London: Butterworths.
Miller, E. L. (1973). *Proc. Nutr. Soc.* 32, 79.
Mathers, J. C. & Miller, E. L. (1977). *Proc. Nutr. Soc.* 36, 75A.

*Present address: Department of Applied Biology, Cambridge University, Pembroke Street, Cambridge CB2 3DX.

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Effect of streptozotocin diabetes on protein synthesis in the liver, kidney and intestinal mucosa of young rats. By V. M. PAIN, M. A. McNURLAN, E. C. ALBERTSE, M. J. CLEMENS* and P. J. GARLICK, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT and *Department of Biochemistry, St George's Hospital Medical School, London SW17 0QT*

In a previous report (Pain & Garlick, 1974) we showed that streptozotocin-induced diabetes in rats results in a fall in the rate of protein synthesis in skeletal muscle and heart, but little or no change in the fractional rate of synthesis of intracellular liver proteins. Since protein synthesis was measured by the constant infusion method (Garlick *et al.* 1973) the results for liver exclude consideration of the synthesis of plasma proteins. By measuring incorporation after a massive dose of labelled amino acid for 10 min, reliable rates of protein synthesis can be obtained in rapidly turning over tissues such as intestine (McNurlan *et al.* 1978). With the liver this method estimates total protein production (i.e. intracellular liver proteins+plasma proteins). Furthermore, treatment of the liver homogenate with a specific antibody to rat serum albumin enables us to estimate albumin synthesis in these animals as a proportion of total liver protein synthesis.

Effect of diabetes on fractional rates of protein synthesis in visceral organs

(Mean values with standard errors)

Organ	Change in protein mass (%)	Rate of protein synthesis (%/d)	
		Control	Diabetic
Liver	No change	79·4± 5·2	58·8± 6·7
Intestine (jejunal mucosa)	+23	146·0±14·2	148·0±20·6
Kidney	+14	44·5± 1·6	44·7± 3·0

The results in the table show that total protein synthesis in liver is reduced in diabetic animals. Since synthesis of intracellular liver proteins is not affected, the results suggest that the main effect is on synthesis of plasma proteins. In agreement with this, immunoprecipitation of labelled albumin from the liver shows a selective fall in albumin synthesis, relative to total liver protein synthesis (from 11·6±0·9% in controls to 8·5±0·8% in diabetics). Preliminary results suggest that this effect is associated with a decreased concentration of translatable messenger RNA for albumin in the liver of diabetic rats.

In contrast to the liver which shows no change in protein mass, both the kidney and the jejunal mucosa show an increase in protein mass but there is no change in the rate of protein synthesis.

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Isolation of intestinal villus and crypt cells suitable for 'in vitro' studies.

By JACQUI BADCOCK and A. M. TOMKINS, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1 7HT*

The intestinal mucosa is a heterogeneous collection of epithelial cells with differing functions and it is important to obtain epithelial cells from specific sites on the villus, in order to study nutritional effects on the small intestine. Previous cyrostatic techniques (Imondi *et al.* 1969) have obtained villus and crypt fractions, but such methods do not permit in vitro studies.

A technique has been established (modified from Weiser, 1973) to prepare nine fractions of isolated epithelial cells. Cell populations were characterized according to their enzyme patterns (Badcock & Tomkins, 1978); villus tip cells had high activities of absorptive enzymes e.g. sucrase, and crypt cells had high activities of enzymes associated with DNA synthesis e.g. thymidine kinase. Light microscopy demonstrated intact morphology of the isolated cells and viability was assessed by several parameters. Results from measurements of oxygen consumption, carbon dioxide production, ratios of Na^+ and K^+ in intracellular and extracellular fluids and trypan blue exclusion, showed that viability was maintained sufficiently long for in vitro studies. Incorporation of tritiated thymidine into DNA of crypt cell preparations was linearly related to time, showing that this technique obtained cells suitable for studies of DNA synthesis.

The details of the method will be discussed.

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Protein synthesis and amino acid oxidation in growing pigs. By P. J. REEDS, M. F. FULLER, G. E. LOBLEY, A. CADENHEAD and J. D. MCDONALD, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Ten female pigs of 34 kg were given a conventional diet with nitrogen 30 g/kg (apparent digestibility 0·86) and metabolizable energy 12·5 MJ/kg. It was given once hourly to two animals at a rate (per $kg^{0.75}$) of 90 g/d (L) and to six at a rate of 130 g/d (H). A further two animals received the diet at the H rate in two equal portions at 10.00 and 16.00 hours. A solution of L-[1-^{14}C]leucine was infused at a constant rate (approximately 8 μCi/h) into the aorta and blood was sampled from a catheter in the aorta 130 mm proximal to the point of infusion. For 3 d, including the day of the infusion, measurements were made of O_2 consumption, CO_2 production and of urinary N and urea. Radioactivity in the expired air was measured continuously with an ionisation chamber. Two of the pigs fed hourly at the H rate received simultaneous infusions of L-[1-^{14}C]leucine via the aorta and L-[4,5-^{3}H]leucine via the hepatic portal vein.

The specific radioactivity (SR) of free leucine in the blood of the animals which were fed once hourly attained a constant value after 3–4 h of infusion; the SR of CO_2 became constant about 30 min later than blood leucine. In the animals which received both hepatic portal ^{3}H and aortal ^{14}C infusions the ratio ^{3}H:^{14}C in the free leucine of blood was not significantly different from the ratio in the infusate. This suggests that free leucine of dietary and endogenous origin mixed completely.

The flux (*Q*) of leucine was estimated from the mean SR of blood leucine in a minimum of four samples taken after 4–6 h of infusion.

Feeding scale	Protein synthesized (S:g N/d)	Fraction of label lost in CO_2 (f_0)	Amino acid oxidation (g N/d)	
			O_L	O_U
H	88	0·16	18	17
H	79	0·12	17	16
H*	71	0·22	20	20
H*	76	0·17	16	18
L	36	0·26	14	13
L	52	0·28	12	13

*These animals failed to eat their hourly meals completely during the infusions.

It is assumed that at plateau of leucine SR (1) the flux of leucine represents the sum of the leucine incorporated into protein and oxidised to CO_2, (2) the fraction of the infused dose excreted as CO_2 (f_0) is the fraction of Q which is oxidized, (3) the SR of the precursor for the oxidation of leucine is the same as the SR of blood leucine. Calculations of total amino acid oxidation (g N/d) by $Q.f_0 \div$average leucine content of protein (O_L) and from urea excretion (O_U) were similar (Table 1) suggesting that assumption (3) is acceptable.

In the two animals which were fed twice daily the SR of blood leucine failed to remain constant in the post-prandial period, giving estimates of Q which rose from 2·04 g N/h at 09.45 hours to 10·6 g N/h at 15.30 hours and of O_L which rose from 0·2 g N/h to 1·6 g N/h over the same period.

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Glucose and urea kinetics in cows in early lactation. By I. Bruckental, J. D. Oldham and J. D. Sutton, *National Institute for Research in Dairying, Shinfield, Reading Berks. RG2 9AT*

The aims of the reported experiments were to study changes in glucose and urea kinetics during the first 9 weeks of lactation and to test the use of urea production as a measure of protein catabolism in order to quantify (a) the maximum potential conversion of protein carbon to glucose carbon and (b) the efficiency of utilization of metabolizable protein (EPU; Oldham, 1978) for milk protein production.

Four Friesian cows were used. They were given, (/d), 3 kg hay and 9 kg concentrates (g/kg, barley 930, groundnut meal 50, minerals 20) for the first 2 weeks of lactation then 4 kg hay and 12 kg concentrates. Concentrates were delivered hourly by continuous feeders and the hay was given in four portions daily. Simultaneous single intravenous injections of [6-^{3}H]glucose (1·5 mCi) and [^{14}C]urea (1 mCi) were given in the 2nd, 4th and 9th weeks *post-partum*. Jugular venous blood samples were then withdrawn from an indwelling catheter for up to 15 h after injection. Milk yield at the time measurements were made was 25–34 kg/d. The cows lost 40–75 kg live weight.

Decline in urea specific activity did not follow first-order kinetics for the first 2 h after injection. This was interpreted as rapid distribution of [^{14}C]urea in a space approximately twice expected blood volume followed by slower distribution in a water space of about 60% body-weight (range 53–74%). Of the injected ^{14}C, 10% (range 3–19%) was recovered in milk and 22% (range 11–43%) in urine so that 44–82% (mean 68%) of urea was degraded, presumably in the digestive tract.

If it is assumed that urea was formed solely as a result of protein catabolism, and that 55 g glucose can be formed from 100 g protein then the maximum possible contribution of protein to measured glucose flux (about 3000 g/d) was 24% (range 10–24%). To refine this calculation we have used Nolan's (1975) estimate, in sheep, that only 35% of urea production was due to protein catabolism (i.e. protein catabolised=urea N (g/d)$\times$6·25$\times$0·35) and we have further assumed that no more than 20% of oxidized protein-C passes through the glucose pool (Lindsay, 1976). On this basis it can be estimated that protein contributed no more than 2% of measured glucose flux.

We define EPU as milk protein output$\div$(milk protein output+protein catabolized). Values for EPU were in the range 58–81% and in the 9th week after calving were generally lower than in the 4th week.

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Manipulation of female growth and efficiency of food utilization. 1. The effects of perinatal testosterone propionate, ovariectomy at 21 d and long-acting testosterone esters from 36 to 38 d of age. By B. N. PERRY, *ARC, Meat Research Institute, Bristol* and ANNA MCCRACKEN, *University of Bath*

Manipulation of females to achieve performance characteristics of entire males would have important implications for meat production. Some results with rats as the experimental model which identify basic endocrine mechanisms responsible for sex differences in performance are reported here.

Sixteen litters, each containing six pups, were paired according to parental weights to eliminate potential differences in size expectation. Eight litters were androgenised (T) within 60 h of birth with 2·5 mg testosterone propionate/rat. Eight litters were untreated (UT). From T and UT litters trios of females were selected. From each trio two females were ovariectomised (OVX) at 21 d of age. One received 50 µl long-acting testosterone esters (TP) (Durateston, Intervet) from 36–38 d every 8–10 d. Rats were fed the same level of intake (g/kg$^{0·75}$ per d) to reduce treatment effects on appetite but not to prevent differences in growth emerging. Body compositions were examined at 91 d of age.

Table 1 shows that perinatal testosterone (T) increased subsequent growth and improved food utilization (FCR). OVX also increased growth and improved FCR. The effects of T and OVX were comparable and additive. However, TP treatment after prior OVX did not further improve performance. T–OVX females used food as efficiently as untreated control males but did not grow as rapidly. There were no differences between groups or sexes in body composition large enough to account for the differences in growth rate or efficiency of food use.

Table 1. *The effects of perinatal testosterone (T), ovariectomy (OVX) and long-acting testosterone esters (TP) on the growth and food utilization of female rats*

	Mean 29 d live weight (g)	Mean 29–91 d live weight gain (g)	Mean FCR 21–91 d (g food/g live weight gain)	Lipid (g/100 g ingestafree empty-body-wt)
Females:				
U	53·8[1]	147·6[1 a]	5·5[2 b]	11·4[1 b]
UT OVX TP	53·8	168·5[b]	5·1[b]	9·9[a]
UT OVX	52·8	183·7[b]	4·7[ab]	11·6[b]
T	51·1	180·4[b]	4·8[ab]	11·4[ab]
T OVX TP	51·3	187·9[b]	4·6[ab]	11·1[ab]
T OVX	53·2[1]	209·1[2 c]	4·3[1 a]	12·2[1 b]
Males:	58·7[1]	232·7[3]	4·2[1]	12·3[1]

[1 2 3] ♂ and ♀ groups bearing different superscripts significantly different ($P<0·001$)
[a, b, c,] ♀ groups bearing different superscripts significantly different ($P<0·05$)

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Manipulation of female growth and efficiency of food utilization. 2. The effects of auto-immunity to luteinizing hormone releasing hormone (LHRH) on growth and food utilization. By B. N. PERRY, *ARC, Meat Research Institute, Bristol* and ANNA MCCRACKEN, *University of Bath*

The similar but distinct effects of androgenization (T) and ovariectomy (OVX) on the growth and efficiency of food utilization of female rats (Perry & McCracken, 1978) raise important questions concerning the endocrine mechanisms by which these two treatments affect performance. Auto-immunization against LHRH was employed to investigate a non-surgical approach to oestrogen removal and to see if LHRH contributed to the mechanism whereby androgenization increased growth and food utilization. Therefore, littermate male (naturally androgenized) and female rats were immunized at 21 d intradermaly with LHRH conjugated to bovine serum albumin. Female rats only received a booster injection at 48 d.

The growth and efficiency of food use by immunized females was markedly improved but there was little effect on these parameters in males (see Table).

Serum oestrogen and testosterone levels were not determined. But vaginal opening was blocked in 3/4 immunized females through to slaughter (>110 d), although this normally occurs no more than 3 weeks following the age of primary immunization. In immunized males testicular development was minimal. These testes returned to an undescended abdominal position and weighed less than 0·2×control testes.

Other studies with comparable females fed *ad lib.* have shown that LHRH auto-immunization increased growth and food utilization to an extent comparable to that of OVX but not more. Because T–OVX females grow more than those which are OVX only (Perry & McCracken, 1978; Perry & Furr, 1978) and LHRH auto-immunization does not markedly affect naturally androgenized male growth (see also Fraser *et al.* 1974) the implication is that LHRH is not central to the mechanism whereby androgenization influences growth and food utilization. Male growth and efficiency appears largely determined by the perinatal influence of testosterone, unless requirements for testosterone can be met by the low circulating levels observed in LHRH auto-immunized males (Fraser *et al.* 1974).

The effects of auto-immunity to LHRH on growth and food utilization (FCR)

(Mean values with standard errors)

	Live weight at		FCR 21–91 d, *ad lib.* fed (g food/g live weight gain)
	21 d	91 d	
Females:			
Immunized (*n* 4)	47·3±0·7	289·5±7·5	5·7±0·2
Untreated (*n* 3)	46·0±1·4	214·7±9·2	7·6±0·2
Males:			
Immunized (*n* 4)	49·6±1·1	334·5±6·2	5·6±0·2
Untreated (*n* 3)	49·3±0·9	348·7±1·9	5·3±0·2

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Manipulation of female growth and efficiency of food utilization. 3. Comparison of the effects of perinatal testosterone and oestradiol on the subsequent growth of female rats. By B. N. PERRY, *ARC, Meat Research Institute, Bristol* and B. J. A. FURR, *ICI Pharmaceuticals Division*

The process by which androgen masculinizes neural development of female rats is thought to involve aromatization (McEwen, 1976). If so, our studies (Perry & McCracken, 1978) suggest that perinatally oestrogen is essential, whereas later the opposite appertains. Identification of cellular endocrine mechanisms which satisfy this concept is difficult. Therefore, the effect of perinatal testosterone propionate (TP) (2·5 mg/rat) or oestradiol benzoate (OB) (250 µg/rat) treatment, within 72 h of birth, on the growth of female rats fed *ad lib.* was studied.

The effects of TP and OB were very different (Fig. 1). OB treatment at our dose causes a persistent dioestrus syndrome (Brown-Grant, 1974). Thus masculinization of growth appears TP specific.

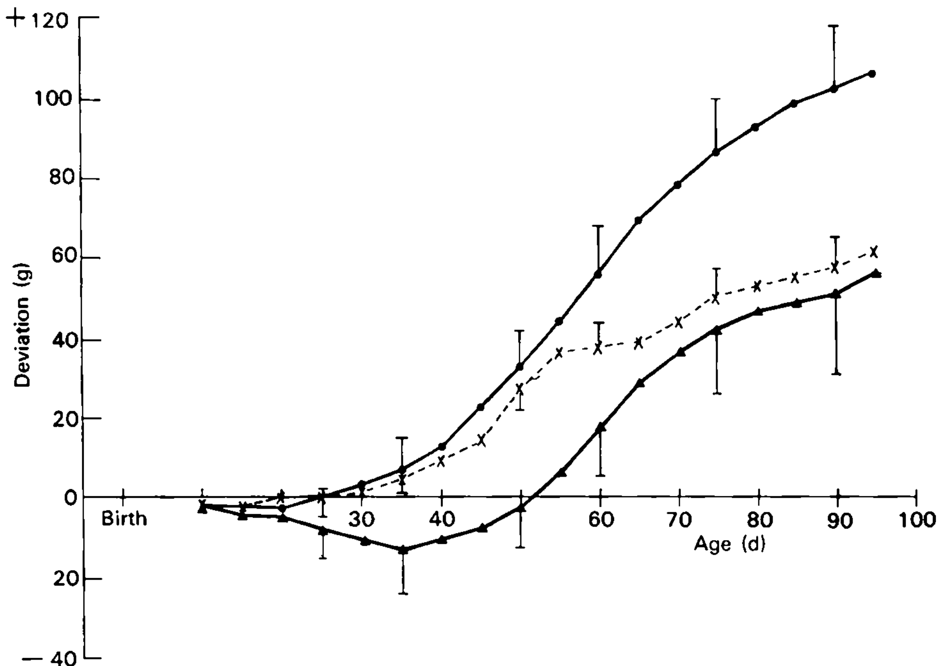

Fig. 1. The deviations in live weight with age of littermate OVX (X–X, *n* 7), TP–OVX (●–●, *n* 12) and OB–OVX (▲–▲, *n* 13) female rats from those of untreated littermate females. Standard deviations at representative ages are shown by vertical bars. Ovariectomy at 21 d.

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Influence of pre- and perinatal malnutrition on sulphated glycosaminoglycans in rat brain. By C. J. BRANFORD WHITE, J. A. APPLEBY and ALISON HARRIS (Introduced by JANET THORN), *Department of Biology, Oxford Polytechnic, Headington, Oxford OX3 0BP*

Pre- and perinatal malnutrition of rats was obtained by reducing mother's intake by 50% during lactation and pregnancy. After 40 d animals were injected with $Na_2{}^{35}SO_4$ and killed 18 h later. After delipidation, 20% less lipid-free brain material was detected in tissue from the experimental animals. Tissue was hydrolysed by treatment with papain and equivalent amounts of ^{35}S-labelled material was found in both the experimental and controls. Papain hydrolysates of the experimental and controls were then chromatographed on DEAE-cellulose using a linear salt gradient. Two major ^{35}S and hexuronic acid fractions were eluted at 0·45 M-NaCl (Fraction A_1) and 0·6 M-NaCl (Fraction A_2). Fraction A_1 was found to be sensitive to chondroitinase ABC and contained galactosamine. Fraction A_2 was undergraded by this enzyme and only glucosamine was detected on analysis. On treatment of this fraction with nitrous acid, 50% of the hexosamine was converted to the anhydromannose derivative indicating that there was an equal proportion of *N*- and *O*-sulphated glucosamine residues. From total sulphate estimation, 25% of Fraction A_2 was disulphated. Glycosaminoglycans were further identified by cellulose acetate electrophoresis in which Fractions A_1 and A_2 migrated with chondroitin sulphate and heparan sulphate standards respectively. From the hexuronic acid values 2·5 times more sulphate was detected in the experimental animals. The concentration of heparan sulphate was the same in both tissues.

This report appears to be the first study of glycosaminoglycans in rats which were ill-fed during pregnancy and lactation. During brain development sulphated glycosaminoglycans are known to reach a maximum after 7 d and then decline to an optimum level reached 30 d after birth (Bachhawat *et al.* 1972). The presence of increased levels of chondroitin sulphate may be linked to some defect in the maturation of brain tissue as restriction of food during pre- and perinatal stages of growth bring about marked and permanent changes in the central nervous system (Dobbing & Smart, 1974).

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Effects of pre- and perinatal undernutrition on muscle fibre and function in fast and slow rat muscles. By K. F. HOWELLS, T. C. JORDAN and DIANA R. MATHEWS (Introduced by JANET THORN), *Neurobiology Laboratory, Biology Department, Oxford Polytechnic, Headington, Oxford OX3 0BP*

Pre- and perinatal undernutrition of rats was effected by means of limiting the mothers' food intake by 50% during pregnancy and lactation. Male offspring were killed at the age of 36 weeks. Body and muscle weights showed marked decreases in the experimental animals, though the deficits found in slow muscles were not statistically significant.

The anterior tibialis (ANTIB) and soleus (SOL) muscles were prepared for histochemical demonstration of Type I, IIA, and IIB muscle fibre types using myosin ATPase and succinic dehydrogenase activity. Mean muscle fibre area determinations showed greater decreases in ANTIB than SOL, SOL muscle fibre areas being relatively unaffected by the undernutrition regimen. Changes in the proportions present of the muscle fibre types were seen in ANTIB and to a smaller extent in SOL. In the former muscle some decreases in the area of certain fibre types were associated with increases in the percentage present, possibly showing a tendency to maintain the physiological cross-section area of the muscle. As far as could be ascertained, no evidence of reductions in muscle fibre number was seen. Preliminary studies on young (35 d) experimental muscles suggest that the slow muscle (SOL) is affected by early undernutrition but has a much greater capacity for postnatal recovery.

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Calcium uptake in the intestinal tract of germ-free and conventional chicks. By MAUREEN F. PALMER and B. A. ROLLS, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Since the intestines of germ-free (GF) animals are thinner than those of their conventional (CV) counterparts it might be expected that differences would be evident in the digestive physiology and function of the gut.

We compared absorption of calcium in eight pairs of GF and CV chicks aged 4 weeks. Chicks of the Institute flock were fasted overnight but with water freely available, before being allowed to feed for 1 h. Each chick was then intubated into the proventriculus and a dose of 0·1 μCi ^{45}Ca in 40 mM-$CaCl_2$ was administered. Two birds were killed by intravenous sodium pentobarbitone injection at 5, 10, 15, 20, 25, 30, 45 and 60 min after dosing. The gut was rapidly tied off into sections and removed, whereupon each segment was washed out with 0·1 M-NaCl. Blood samples and the right tibiotarsus were removed. Washings, gut tissue, serum and bone were processed and their ^{45}Ca content was estimated by liquid scintillation counting using an internal standard.

Intestinal transit was appreciably more rapid in the GF chicks. Chicks from both environments demonstrated a double absorption peak, tissue uptake being extremely rapid within 5 min, falling up to 20 min and rising after 30 min. The Ca uptake of the gut tissue was rather higher in CV birds than in their GF counterparts, particularly in the initial stages. Although the amount taken up was similar in the duodenum (at 30 min, GF 884×10^{-6} μCi; CV 877×10^{-6} μCi), the difference in total uptake was mainly due to a much greater uptake in the ileum in CV birds (at 10 min, GF 2510×10^{-6} μCi; CV 4860×10^{-6} μCi). The accumulation in bone was slightly greater in the GF chicks (at 30 min, GF 180×10^{-6} μCi; CV 161×10^{-6} μCi) (cf. Edwards & Boyd, 1963) whereas the serum Ca remained constant and was similar in the two groups.

Several hypotheses could be advanced to explain these differences. The GF gut being thinner, especially in the ileal region, may be capable of taking up less calcium, which would explain both the GF–CV differences and the similarity in the duodenum, which is thicker and where the difference between GF and CV birds is less marked. Alternatively, the thinner GF gut may transport Ca more rapidly so that less remains in the gut tissue. Once absorbed, the Ca may be secreted at a different rate in the two environments. There may be differences in Ca-binding protein in the GF and CV chicks, or in the rate at which it is synthesized in response to Ca uptake (cf. Spencer *et al.* 1978).

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Effect of total starvation on the number and size of fibres in different skeletal muscles from the young of four species. By K. O. KIM and P. V. J. HEGARTY, *Department of Food Science and Nutrition, University of Minnesota, St Paul, MN 55108, USA*

Many of the symptoms of protein-energy malnutrition can be reproduced in animal models (Kirsch *et al.* 1968). However, there is no information on morphological changes in different muscles when an extensive reduction in body-weight occurs due to a total withdrawal of food.

The body-weight of young rats (68 g), rabbits (1400 g), guinea-pigs (208 g) and hamsters (69 g) was reduced to 42, 765, 137 and 45 g, respectively, by a total withdrawal of food. The weight of the biceps brachii and plantaris was reduced significantly ($P<0.01$) in all four species. The weight of the soleus decreased in the starved hamster ($P<0.001$), but not in the other species. The extent of the weight loss in different muscles varied between the species. For example, the percentage weight loss for the soleus, plantaris and biceps brachii in the rabbit was 8, 54 and 52% respectively. Weight loss in the same three muscles from the hamster was 30, 24 and 29% respectively.

A technique, recently developed in our laboratory, using the Coulter Counter was used to simultaneously measure fibre diameter and total fibre number in formalin-fixed muscles.

Table 1. *Muscle fibre number and diameter (μm) in control and starved animals*

(Number of muscles in control and starved groups in parentheses)

			Soleus		Plantaris		Biceps brachii	
Species	Treatment		No.	Diameter	No.	Diameter	No.	Diameter
Rat	Control	(6)	3110	25·5	5756	20·8	8741	23·0
			**	NS	**	*	**	***
	Starved	(6)	2255	24·2	4193	19·4	6496	20·0
Rabbit	Control	(4)	8964	46·3	30300	41·7	22354	43·1
			NS	NS	NS	**	NS	***
	Starved	(3)	8918	50·2	32892	25·3	20878	26·6
Guinea-pig	Control	(6)	3674	23·2	8146	24·4	12664	24·9
			NS	NS	NS	*	NS	***
	Starved	(6)	3667	23·7	7002	20·2	13006	17·6
Hamster	Control	(6)	1352	35·5	1531	32·2	4900	30·3
			NS	**	NS	***	*	NS
	Starved	(6)	1386	29·5	1959	27·2	3694	28·8

NS, not significant; * $P<0.05$; ** $P<0.01$; *** $P<0.001$.

Cross-sections of muscles in which fibre number was significantly reduced during starvation (e.g. the plantaris and biceps brachii muscles in the rat) did not exhibit the extensive muscle degeneration observed in infantile protein malnutrition (Montgomery, 1962), or in vitamin E-deficiency in the rabbit (Chan & Hegarty, 1977).

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