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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Fourteenth Meeting of the Nutrition Society was held in the Morris Lecture Theatre, Robin Brook Centre, St Bartholomew's Hospital, West Smithfield, London on Monday and Tuesday, 20/21 *May* 1985, *when the following papers were read:*

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The effect of denervation on protein synthesis in brown adipose tissue in cafeteria-fed rats. P. W. EMERY and O. C. FLEET, *Department of Nutrition, Queen Elizabeth College, London W8* and N. J. ROTHWELL and M. J. STOCK, *Department of Physiology, St George's Hospital Medical School, London SW17*

The hyperphagia which results from feeding rats a highly palatable 'cafeteria' diet causes an increase in the mass and protein content of brown adipose tissue, as well as increased thermogenic activity in this tissue. Both these effects may be due to the local action of chronically-stimulated sympathetic nerves, but we have now investigated the extent to which systemic influences such as the altered hormonal environment may also be involved.

Twenty, male Sprague-Dawley rats (Charles Rivers Ltd), weighing 150–160 g, were anaesthetized and the five sympathetic nerves supplying one lobe of interscapular brown adipose tissue (IBAT) were cut; the contralateral lobe was left intact. The rats were then allocated randomly to two groups and fed for 4 d on either a cafeteria diet (Rothwell & Stock, 1982) or a stock diet (PRD, Christopher Hill Ltd), after which the rate of protein synthesis in the two IBAT depots was measured in vivo using the large-dose phenylalanine method (Garlick *et al.* 1980).

	Stock-fed				Cafeteria-fed			
	Intact		Denervated		Intact		Denervated	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
IBAT mass (mg/lobe)	84·9	8·2	89·3	6·0	134·1	7·8	111·6	8·4
IBAT fat (mg/lobe)	23·9	5·7	21·8	2·6	62·8	4·0	50·9	2·0
IBAT protein (mg/lobe)	4·8	0·5	5·2	0·5	7·1	0·7	5·5	0·5
IBAT protein synthesis rate (%/d)	20·7	1·7	22·9	3·3	25·8	2·0	31·3	2·5

The increased mass of both lobes of IBAT in the cafeteria-fed rats was largely due to lipid accumulation; however, there was also an increase in the protein content of the intact lobe (42%, $P<0.05$ *v.* mean stock fed), which was completely prevented by denervation ($P<0.05$). The rate of protein synthesis in IBAT was increased by 26% in the cafeteria-fed rats ($P<0.05$ mean cafeteria *v.* mean stock fed). However, there was no significant difference in protein synthesis rates between the intact and denervated lobes. Thus it appears that protein synthesis in brown adipose tissue increases in response to the altered hormonal environment caused by overfeeding, but protein breakdown in this tissue is suppressed by the action of the sympathetic nervous system to allow a net increase in the amount of metabolically active protein.

Garlick, P. J., McNurlan, M. A. & Preedy, V. R. (1980). *Biochemical Journal* **192**, 719–723.
Rothwell, N. J. & Stock, M. J. (1982). *British Journal of Nutrition* **47**, 461–471.

Effect of hypophysectomy on energy balance and brown adipose tissue thermogenesis in the rat. By NANCY J. ROTHWELL and MICHAEL J. STOCK. *Department of Physiology, St George's Hospital Medical School, Tooting, London, SW17*

Surgical adrenalectomy stimulates thermogenesis and brown adipose tissue (BAT) activity, and inhibits fat deposition in the rat (Marchington *et al.* 1983). These effects may be due to either a fall in corticosterone or a rise in adrenocorticotrophic hormone (ACTH) levels. In the present study we have investigated this problem further by measuring energy balance in hypophysectomized (HYPX) male rats with or without replacement with ACTH (10 μg/rat per d), and their sham-operated, pair-fed controls. Due to pair-feeding, metabolizable energy intakes were similar for all groups, but body-weight and energy gains were lower in HYPX rats over the 19 d experiment (mean (SE)) (body energy gain: control 128 (10), HYPX 23 (28), $P<0.001$, HYPX/ACTH 207 (60) kJ, not significant), and net energetic efficiency was markedly suppressed by hypophysectomy (HYPX 4 (3), control 19·0 (1·8)%, $P<0.001$) but restored by ACTH (20·2 (2·1)%).

The thermic response to food (40 kJ meal) was increased by 50% in HYPX rats and BAT mass, protein content and thermogenic activity (assessed from purine nucleotide binding to isolated mitochondria) were all elevated in HYPX rats but were not completely restored by ACTH treatment (BAT GDP-binding (pmol/mg protein): control 37 (3), HYPX 132 (16), $P<0.001$; HYPX/ACTH 60 (8), $P<0.05$). Measurements of adrenal and testicular weights and plasma corticosterone levels (ng/ml: control 119 (23), HYPX 5 (1), $P<0.001$, HYPX/ACTH 133 (13) indicated that hypophysectomy was successful and replacement complete. Aldosterone levels remained normal. Hypophysectomy had a similar effect on thermogenesis and BAT activity to adrenalectomy and this was largely, but not totally, prevented by ACTH replacement.

These results are consistent with an inhibitory effect of corticosterone on thermogenesis, whereas ACTH may be stimulatory.

Marchington, D., Rothwell, N. J., Stock, M. J. & York, D. A. (1983). *Journal of Nutrition* **113**, 1395–1402.

Hypothalamic control of brown adipose tissue in Zucker lean and obese rats. By S. J. HOLT, H. V. WHEAL and D. A. YORK, *School of Biochemical and Physiological Sciences, University of Southampton, Southampton SO9 3TU*

The obesity of the Zucker (*fa/fa*) rat is associated with the loss of brown adipose tissue (BAT) thermogenesis in response to dietary signals although non-shivering thermogenesis is normal (Holt *et al.* 1983). This loss of diet-induced BAT function has been connected with a reduction in the sympathetic stimulation of BAT and the absence of any increases in BAT sympathetic activity in response to food intake (York *et al.* 1985). Adrenalectomy, which prevents the development of obesity, restores both BAT thermogenesis and sympathetic activity to normal in the obese (*fa/fa*) rat.

Since the ventromedial hypothalamic area is known to have an important role in regulating both food intake and energetic efficiency (Bray & York, 1979), experiments have been performed to investigate the effect of electrical stimulation of the ventromedial and other hypothalamic centres on BAT function.

Female lean and obese (*fa/fa*) rats (7 weeks of age) were anaesthetized with urethane, after which core temperature was maintained by a thermostatically controlled heating blanket. A fine bipolar steel electrode was stereotaxically introduced into the appropriate hypothalamic region and the area stimulated for 30 s (50 Hz at 20–30 volts). Location of the electrode was confirmed histologically after each experiment. BAT temperature was continuously recorded by a thermocouple fixed between the lobes of the interscapular pad.

Stimulation of the supraoptic and ventromedial hypothalamic areas both increased BAT temperature. The increase in temperature was prevented either by propranolol (20 mg/kg intraperitoneally) or by bilateral denervation of the interscapular BAT. Stimulation of the ventromedial hypothalamus in obese (*fa/fa*) rats induced a normal increase in BAT temperature (Table). This response was unaffected by adrenalectomy.

Increase in BAT temperature (△°) *after electrical stimulation* (5 *V*) *of the ventromedial hypothalamus of lean and obese* (fa/fa) *rats* (*number of animals in parentheses*)

Lean						Obese					
Control			Adrenalectomized			Control			Adrenalectomized		
Mean	SE		Mean	SE		Mean	SE		Mean	SE	
0·81	0·05	(11)	0·90	0·04	(5)	0·91	0·04	(7)	0·90	0·13	(8)

The results suggest that the efferent sympathetic pathway from the ventromedial hypothalamus to BAT is normal in the obese rat. Failure to increase sympathetic stimulation of BAT after feeding in the *fa/fa* rat may reflect either the inability to monitor or respond to dietary signals, or an inhibition of the sympathetic response.

Bray, G. A. & York, D. A. (1979). *Physiological Reviews* **59**, 719–809.
Holt, S. J., York, D. A. & Fitzsimons, J. T. R. (1983). *Biochemical Journal* **214**, 215–223.
York, D. A., Marchington, D., Holt, S. J. & Allars, J. (1985). *American Journal of Physiology*. (In the Press).

Blood lactate concentration during one-leg exercise with normal and low muscle glycogen concentration in the non-exercising leg. By ADRIANNE E. HARDMAN, A. HUTBER and C. WILLIAMS, *Department of Physical Education and Sports Science, University of Technology, Loughborough, Leicestershire LE11 3TU*

Blood lactate concentration during exercise is often assumed to reflect the rate of glycolysis in skeletal muscle. However, skeletal muscle also takes up lactate, at a rate which is related to its glycogen content (Essen *et al.* 1975). The purpose of the present study was to examine the influence of the glycogen concentration of non-exercising muscle on blood lactate concentration during exercise.

Six male subjects exercised with one leg for 30 min at 75% of one-leg maximum oxygen uptake ($\dot{V}O_{2\ max}$) on two separate occasions, i.e. with normal glycogen (NG) and low glycogen (LG) concentrations in the contralateral limb. The day before the LG trial, subjects cycled with the contralateral limb for 90 min at 70% of one-leg $\dot{V}O_{2\ max}$ to reduce muscle glycogen concentration in that limb. A low-carbohydrate diet was fed in the 24 h before each trial (13·7 MJ, 157 g carbohydrate) to ensure low liver glycogen concentration before each trial and to restrict glycogen repletion in the muscle of the contralateral limb before the LG trial.

Pre-exercise concentration (mean and SD) of plasma fat metabolites were higher for the LG trial than for the NG trial (free fatty acids, 0·55 (0·11) *v.* 0·41 (0·07) mmol/l, $P<0·05$; glycerol 0·105 (0·046) *v.* 0·074 (0·024) mmol/l, not significant). Blood glucose concentration was significantly lower during the final 5 min of the LG trial than the NG trial (3·99 (0·25) *v.* 4·23 (0·36) mmol/l, $P<0·05$). Blood lactate concentrations were, however, similar for the LG trial (10 min, 4·87 (1·65) mmol/l; 30 min, 4·43 (1·94) mmol/l) and the NG trial (10 min, 4·95 (1·52) mmol/l; 30 min, 4·49 (2·10) mmol/l). These results suggest that the glycogen content of a large non-exercising muscle mass has no measurable influence on the concentration of blood lactate during one-leg exercise.

Essen, B., Pernow, B., Gollnick, P. D. & Saltin, B. (1975). In *Metabolic Adaptation to Prolonged Exercise*, pp. 130–134 [H. Howald and J. R. Poortmans, editors]. Basel: Birkhäuser Verlag.

Quantification of the functional capacity for carbohydrate absorption in man by a simple non-invasive technique. By J. D. O'BRIEN, D. G. THOMPSON, M. IBBOTSON, R. MCCREA, W. R. BURNHAM and E. WALKER, *Department of Gastroenterology, The London Hospital, Whitechapel, London E1*

In normal people, dietary carbohydrate is often incompletely absorbed in the small intestine (Anon., 1983) and, in some individuals, sufficient carbohydrate is fermented by colonic bacteria to produce symptoms indicative of food intolerance, e.g. diarrhoea, bloating and excessive production of gases, including hydrogen.

Although many patients associate the ingestion of specific carbohydrate-containing foods with such symptoms, lack of a suitable clinical technique for measuring absorptive capacity has prevented the identification of which, if any, carbohydrate may be responsible in an individual patient. We report the development of a simple technique which now enables the functional absorptive capacity of the gut for a carbohydrate to be determined and, using this method, have identified the absorptive capacity of the normal intestine for fructose.

After an overnight fast, fourteen normal adult volunteers each consumed, on separate days, a series of mixtures of fructose and glucose dissolved in 250 ml water. The quantity of fructose in the solutions varied from 0 to 50 g. Glucose (virtually completely absorbed in the upper jejunum), was added as necessary to maintain the total quantity of sugar at 50 g, thus eliminating the effects of differing solution osmolalities on gastric emptying and transit.

Serial measurements of exhaled breath hydrogen concentration were made after each meal; a rise indicated the fermentation of unabsorbed sugar in the colon.

No subject showed a rise in breath hydrogen after 50 g glucose, indicating complete absorption. As the proportion of fructose increased, however, a rise was seen in all individuals. The dose of fructose at which this rise first occurred indicated that the individual's functional absorptive capacity for fructose had just been exceeded.

Repeated studies indicated that this threshold dose was constant within individuals (10 g) but varied between individuals (range of threshold dose 20–>50 g), depending upon body size. Two subjects showed no hydrogen rise after 50 g, but a rise at 80 g fructose.

In three individuals, intravenous hyoscine butylbromide was given to temporarily inhibit gastrointestinal transit. A rise in threshold dose by at least 10 g occurred, indicating an increase in fructose absorption with increased duration of small intestinal contact.

This study demonstrates that the functional absorptive capacity of the small intestine for a test carbohydrate can be non-invasively determined by breath hydrogen analysis. Because of its simplicity, the method seems particularly suitable for screening for carbohydrate malabsorption in patients with suspected food intolerance.

Anon. (1983). *Gastroenterology* **85**, 769.

The influence of the sex of the individual and of the menstrual cycle on galactose tolerance. By CELIA A. WILLIAMS, ALISON M. OWENS and PAULA HUNT, *Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH*

It has been suggested that galactose tolerance is influenced by the sex of the individual, the stage of the menstrual cycle and by pregnancy; the results are conflicting and are based on the excretion of urinary reducing bodies after galactose ingestion (Rowe, 1924) or lactose (Watkins, 1928).

To investigate the influence of the menstrual cycle on galactose tolerance, eight female volunteers were investigated on four occasions during their menstrual cycle. Six males were investigated at regular weekly intervals. All females were nullipare and had histories of regular 24–31 day menstrual cycles. Basal body temperature and onset of menstruation were recorded. All subjects were considered to be in good health and non-obese, the age range was 18–33 years. After an overnight fast each subject was given 0·5 g galactose/kg body-weight (BW) in 4 ml water/kg BW. Blood samples were taken before the meal and at 15, 30, 60 and 90 min after. Serum was analysed for galactose, glucose and insulin. Urine was collected for 6 h after galactose ingestion, the volume recorded and a portion analysed for galactose.

Results from seven female subjects were classified according to week. The serum response to galactose was found to be consistently greatest during either week 3 or 4. The mean (with SE) serum galactose concentration at 30 min was 1·92 (0·278) mmol/l in week 4 and 0·99 (0·144), 1·30 (0·328) and 1·25 (0·159) mmol/l in weeks 1, 2 and 3 respectively. The mean area under the serum galactose response curve was greatest in week 4. The serum glucose and serum insulin response to galactose and the subsequent urinary galactose excretion were not influenced by the menstrual cycle. There were no differences in the mean serum galactose response to galactose or in the urinary excretion of galactose in the male volunteers during repeated galactose tolerance tests. The mean serum galactose level at 30 min was significantly greater in males when compared with the female response in week 1 ($P<0·01$) but there were no significant sex differences in the areas under the serum galactose or serum insulin response curves. The mean area under the serum glucose response curve to galactose was significantly greater in males than in females during weeks 1, 2 and 3 ($P<0·01$).

These results show that the serum galactose response to galactose is not influenced by the sex of the consumer, although the serum glucose response is significantly greater in males. It is possible that the elevated serum progesterone levels or simultaneously elevated progesterone and oestrogen levels found during the pre-menstrual phase influence galactose tolerance. This effect is not mediated through an altered urinary loss of galactose. Differences in the rate of gastric emptying which occur in the menstrual cycle (Macdonald, 1956) might explain the results reported. It is possible that the hormonal changes in the menstrual cycle can influence the hepatic metabolism of galactose.

Macdonald, I. (1956). *Gastroenterology* **30**, 602–607.
Rowe, A. W. (1924). *Archives of Internal Medicine* **34**, 388–401.
Watkins, O. (1928). *Journal of Biological Chemistry* **80**, 33–66.

The effects of a high unrefined carbohydrate, high fibre, low fat and low sodium dietary regimen in type II diabetics with moderate hypertension. By P. J. PACY, P. M. DODSON, A. J. KUBICKI, R. F. FLETCHER, and K. G. TAYLOR, *Department of Diabetes and Clinical Investigation Unit, Dudley Road Hospital, Birmingham*

We have previously reported the hypotensive response after treatment for 3 months with a high cereal fibre (40 g/d), low fat (25% dietary energy) and low sodium (60–80 mmol/d) diet in type II diabetic subjects with hypertension (Dodson *et al.* 1984). However, the response in patients with moderate hypertension (diastolic blood pressure >105 and <115 mmHg) is not known. We have therefore conducted a 3 month study of such a diet in thirteen patients (age range 51–64 years, ideal body-weight 127·3±11·3%) with hypertension (mean duration 3·8 years) and type II diabetes. Hypertension in these subjects had been poorly controlled for a year before the study (mean systolic 188·7 (SD 13·5) and diastolic blood pressure 111·3 (SD 4·1) mmHg) despite a combination of two or more antihypertensive drugs.

Eleven patients completed the study with no change in drug therapy throughout and two patients were withdrawn owing to an elevation in blood pressure after 1 month of dietary treatment. Drug therapy was omitted on the mornings of the initial and 3 month visits.

After 3 months, the eleven patients were divided into two groups by a separate 'blind' observer using a compliance scoring system. For each of urine sodium excretion, weight, glycosylated haemoglobin, fasting serum cholesterol, and triglyceride levels that decreased an arbitary score of −1 was given, while +1 was given for each that increased. Patients were considered compliers if their total score was zero or positive. The blood pressure response was not included. Those compliant to the regimen comprised group A ($n7$) and those who were not (group B, $n4$) acted as controls. It was considered unethical to recruit a non-treated control group.

Group A demonstrated a significant reduction in mean (and SD) systolic (190·4 (18) to 166·6 (22·4) mmHg, $P<0·02$) and diastolic blood pressures (113·1 (3·7) to 103·3 (9·1) mmHg; $P<0·01$), body-weight (78·5 (5·5) to 74·3 (6·8) kg; $P<0·02$), daily urinary Na output (210·3 (79·9) to 120·3 (56·1) mmol; $P<0·02$) and low density lipoprotein–cholesterol (3·4 (0·8) to 2·2 (0·7); $P<0·02$) after 3 months. A reduction of mean glycosylated haemoglobin was also noted of 2·2%. In contrast, group B demonstrated no significant changes and, in particular, blood pressure remained constant (systolic 204·5 (27·9) to 198·8 (34·8) mmHg and diastolic 111·0 (2·2) to 110·3 (8·9) mmHg).

We conclude that there may be a hypotensive response to this dietary regimen but that this modified diet, at best, could be considered only as an adjunct to conventional antihypertensive drug therapy in patients with moderate hypertension.

Dodson, P. M., Pacy, P. J., Bal, P., Fletcher, R. F. & Taylor, K. G. (1984). *Diabetologia* **27**, 522–526.

One year follow-up treatment of hypertension in type II diabetic subjects with a high cereal fibre, low fat and low sodium dietary regimen. By P. M. DODSON, P. J. PACY, M. G. WEBBERLEY, R. F. FLETCHER and K. G. TAYLOR, *Department of Diabetes and Clinical Investigation Unit, Dudley Road Hospital, Birmingham*

We have previously reported the hypotensive response of a high cereal fibre (40 g/d), low fat (25% dietary energy) and low sodium (60–80 mmol/d) diet in a 3 month controlled trial in mildly hypertensive diabetic subjects (Dodson *et al.* 1984). However, it is not known whether this response is maintained in the long term. We now report the effects of this dietary regimen, after 1 year in fifty hypertensive diabetic subjects (mean age 55·8±8·1 years, twenty-five male and twenty-five female, ideal body-weight 133·2±22%).

Fourteen patients (group A) did not achieve normotension after 3 months and were commenced on antihypertensive drug therapy, and two patients did not take part in the follow-up. The remaining thirty-four patients (group B) demonstrated significant reductions (see Table) in mean systolic and diastolic blood pressures, body-weight, urinary Na output, glycosylated haemoglobin and serum triglyceride, after both 3 months and 1 year without change in drug therapy. Group A, however, demonstrated no significant reduction after 3 months in mean (and SD) urinary Na output (195·8 (85·3) to 165·7 (82·9) mmol/d), body-weight (81·5 (16·8) to 80·2 (15·9) kg) and serum triglyceride levels (1·83 (1·19) to 1·72 (1·05) mmol/l), but a weakly significant reduction in mean glycosylated haemoglobin (11·6 (3·9) to 9·9 (3·2)%, $P<0.05$), suggesting poor compliance.

Biochemical and clinical changes in group B

(Values are means with their standard deviations for thirty-four subjects)

Time	Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)		Body-weight (kg)		Urinary Na (mmol/d)		Urinary Na:K		Glycosylated haemoglobin (%)		Fasting serum triglyceride (mmol/l)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
0	178·4	19·5	95·6	9·6	75·1	11·6	203	76·7	3·0	1·13	12·0	3·25	1·64	0·79
3 months	158·5***	18·4	83·9***	9·9	72·0***	10·6	126·7***	59·5	1·7***	0·69	9·7***	2·9	1·49	0·64
1 year	161·0***	21·6	85·9***	9·3	72·6**	9·96	141***	56	2·05***	0·7	10·5**	3·9	1·42*	0·54

Significantly different from mean value at time 0 (paired *t* test): *$P<0.05$, **$P<0.01$, ***$P<0.001$.

We conclude that a significant hypotensive response accompanied by improvement in diabetic control, can be maintained in the long term in a fair proportion of patients (0·68) on this dietary regimen.

Dodson, P. M., Pacy, P. J., Bal, P., Fletcher, R. F. & Taylor, K. G. (1984). *Diabetologia* **27**, 522–526.

Response of human faecal flora of a volunteer to a change in diet. By G. M. WYATT and C. E. BAYLISS (Introduced by D. A. T. SOUTHGATE), *AFRC Food Research Institute, Norwich, NR4 7UA*

Effects of changes in diet on human faecal flora have not been readily demonstrated (Moore *et al.* 1981). Substrates available for fermentation in the large intestine are mainly polysaccharide materials resistant to digestive enzymes ('dietary fibre') and include the plant gums widely used in food processing. The work of McLean Ross *et al.* (1983) showed increased breath hydrogen after ingestion of gum arabic, suggesting microbial involvement in the degradation of the gum.

In a preliminary study reported here, an adult female volunteer ate 10 g gum arabic daily for 21 d in addition to a normal mixed western diet. Using a most probable number method, the proportion of the total viable anaerobic bacterial flora able to degrade gum arabic was determined in faecal samples collected before, during and after gum arabic ingestion. This proportion increased during ingestion and returned to the previous level after gum arabic was removed from the diet, contrasting with a previous study using guar gum where the total count of viable bacteria increased but the proportion of guar-gum fermenters remained constant (Bayliss & Houston, 1985).

Anaerobic bacterial counts in faeces (viable bacteria/g dry weight)

Period	No. of samples	Total count mean	GA fermenters mean	Proportion fermenting GA
Before ingestion (days 0–7)	4	$4{\cdot}3\times10^{11}$	$1{\cdot}6\times10^{10}$	$0{\cdot}065^{a,b}$
During ingestion (days 10–13)	2	$2{\cdot}6\times10^{11}$	$4{\cdot}8\times10^{10}$	$0{\cdot}183^{a,c}$
During ingestion (days 17–27)	4	$6{\cdot}3\times10^{11}$	$3{\cdot}5\times10^{11}$	$0{\cdot}536^{c}$
After ingestion (days 71–74)	2	$3{\cdot}6\times10^{11}$	$1{\cdot}8\times10^{10}$	$0{\cdot}051^{b}$

[a,b,c]Values with different superscript letters were significantly different ($P=0{\cdot}05$).
GA, gum arabic.

We were unable to detect undegraded gum arabic in faecal samples, suggesting that even with the lowest level of gum-arabic fermenters present, the residence time in the large intestine was sufficient to degrade the polymer.

The results show a clear change in the faecal flora of one individual following a change in diet, probably indicating an adaptive response of the colonic flora to the presence of a new substrate.

Bayliss, C. E. & Houston, A. P. (1985). *Food Microbiology.* (In the Press).
McLean Ross, A. H., Eastwood, M. A., Anderson, J. R. & Anderson, D. M. W. (1983). *American Journal of Clinical Nutrition* **37**, 368–375.
Moore, W. E. C., Cato, E. P., Good, I. J. & Holdeman, L. V. (1981). In *Banbury Report* 7: Gastrointestinal Cancer, pp. 11–19 [W. R. Bruce, P. Correa, M. Lipkin, S. R. Tannenbaum and W. D. Wilkins, editors]. New York: Cold Spring Harbor Laboratory.

Variability in the energy cost of growth in rats: effect of dietary protein concentration and plane of nutrition. By PENNY COYER, J. P. W. RIVERS, D. J. MILLWARD, *Nutrition Research Unit, Department of Human Nutrition, London School of Hygiene & Tropical Medicine, 4 St Pancras Way, London NW1 2PE*

It is generally accepted that the energy cost of protein deposition (k_p) exceeds that of fat deposition (k_f) and these costs are frequently regarded as constants. Published estimates nevertheless vary widely. Since reductions in dietary protein content affect the composition of tissue gain and may be associated with dietary-induced thermogenesis (Rothwell *et al.* 1983), alterations in growth costs might be anticipated but studies have yielded inconsistent results. Most studies have failed to demonstrate statistically significant changes in the overall cost of energy deposition (k_e) with reductions in dietary protein content; k_p has been found to be either elevated (Campbell & Dunkin, 1983) or unchanged (Close *et al.* 1983). We have estimated both k_e and k_p in rats fed on decreasing amounts of two diets of differing protein content.

Weight-matched groups of male Sprague-Dawley rats (103·5±1·6 g initial weight) were given diets containing 22% (A) or 9% (B) of gross energy as protein fed at approximately 25, 50, 75 or 100% of *ad lib.* intake for 7 d. Metabolizable energy intake (MEI) was estimated from measured energy intakes of individual rats. Energy (E) and protein (P) gains were determined as the difference between the energy and protein contents of an initial group of rats and rats killed after 7 d. Fat (F) gain was estimated as the difference between total energy gain and energy gain as protein.

Regression analysis showed ME (kJ/kg body-weight $(W)^{0.75}$ per d) to be linearly related to E, with k_e increased on the 9% protein diet ($P<0.001$) and maintenance unchanged:

Diet A: MEI = 1·28 (SE 0·04) E + 561·8 (SE 8·9), r 0·99 (n 19)
Diet B: MEI = 1·7 (SE 0·09) E + 563·6 (SE 17·9), r 0·97 (n 18)

The relation between MEI, P, and F (kJ/$W^{0.75}$ per d) was found to be curvilinear: when k_f was held constant at 1·25 kJ/kJ the residual energy intake could be related to P in a quadratic manner (i.e. at low MEI the apparent efficiency of protein gain was increased):

Diet A: MEI–1·25F = 1·15 (SE 0·14) P + 0·005 (SE 0·0017) P^2 + 507 (SE 14·7), R 0.99
Diet B: MEI–1·25F = 2·6 (SE 0·27) P + 0·02 (SE 0·005) P^2 + 513·8 (SE 6), R 0·95

These results demonstrate that the costs of growth vary with dietary manipulation. The apparent value of k_p increases with a reduction in dietary protein content and decreases at low energy intakes.

Campbell, R. G. & Dunkin, A. C. (1983). *British Journal of Nutrition* **49**, 221–230.
Close, W. H., Berschauer, F. & Heavens, R. P. (1983). *British Journal of Nutrition* **49**, 255–269.
Rothwell, N. J., Stock, M. J. & Tyzbir, R. S. (1983). *Metabolism* **32**, 257.

The efficiency of utilization of lysine by weanling rats. By SARAH C. BOLTON and E. L. MILLER, *Department of Applied Biology, Pembroke Street, Cambridge*

The efficiency of utilization of a limiting amino acid by weanling rats has been investigated.

A basal, lysine-deficient diet containing (g/kg) wheat gluten 65, zein 67, sesame seed meal 132, maize starch 192·2, maize oil 25, lard 25, cellulose 50, DL-methionine 5·0, L-histidine 2·5, L-tryptophan 1·0, L-threonine 3·0, L-isoleucine 4·0, L-valine 5·0, mineral and vitamin premixes 46·05, sucrose 342·45, chromic oxide premix 9·0, glutamic acid 20·8 was formulated. Isonitrogenous replacement of glutamic acid with lysine gave six diets with 2·7–13·3 g lysine/kg dry matter (DM).

Thirty-six male weanling rats were fasted overnight, weighed and randomly assigned, five to each diet and six for slaughter. The slaughter group were killed, the contents of the gastrointestinal tract removed, and a regression equation relating fasted weight and chemical composition derived to estimate the initial composition of the remaining rats. Diets were offered *ad lib.* and food intake measured. Animals were weighed on the 14th day after fasting overnight, offered food for 2 h and killed 2 h later. Terminal ileal digesta were collected and other gut contents discarded. Carcasses were minced and analysed for lysine. Ileal samples were pooled to one per diet and analysed for chromic oxide and lysine. Lysine secreted in the small intestine was estimated from ileal digesta collected from rats offered a protein-free diet for 6 d; this value (0·40 g/kg DM intake) was used to calculate the true digestibility of lysine.

Diet no. . . .	1	2	3	4	5	6
Lysine in diet, (g/kg DM)	2·7	3·9	6·3	8·6	10·4	13·3
DM intake (g/d)	6·87	11·30	14·51	14·88	13·96	15·34
Weight gain (g/d)	0·82	3·02	5·33	7·11	6·49	7·47
Lysine absorbed (mg/d)	15·9	41·3	83·7	125·2	142·6	198·8
Carcass lysine retention (mg/d)	7·1	30·3	62·8	93·9	90·7	94·6
Endogenous lysine (mg/d)	2·7	4·5	5·8	6·0	5·6	6·1

Diets 1 and 3 are limiting in lysine, the requirement for maximum growth being met by diet 4. A linear relationship

$$y = -0{\cdot}024 + 0{\cdot}825x, \text{ RSD}, 0{\cdot}105, r^2\ 0{\cdot}96$$

was observed between lysine retention (carcass lysine plus lysine secreted in the small intestine) and absorbed lysine for diets 1 to 4. Absorbed lysine was thus used with an efficiency of 82·5 (SE 3·96)%. There was no indication of any deviation from linearity as adequacy of lysine supply was approached.

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Chemical labelling of dietary protein by transformation of lysine to homoarginine: a new technique to follow intestinal digestion and absorption. By H. HAGEMEISTER, *Institut für Physiologie und Biochemie der Ernährung, Bundesanstalt für Milchforschung, Postfach* 6069, *D*-2300, *Kiel, Federal Republic of Germany,* and H. ERBERSDOBLER, *Institut für Humanernährung und Lebensmittelkunde der Universität Kiel, Federal Republic of Germany*

Precaecal digestibility of proteins is calculated from the flow of nitrogen at the ileum. The endogenous N is very difficult to estimate. In this paper a new technique is proposed which allows endogenous and exogenous protein to be distinguished. This is achieved by transforming lysine side-chains in the dietary protein into homoarginine (Mauron & Bujard, 1963) and monitoring the homoarginine content of the digesta. Because homoarginine is not used for protein synthesis it does not appear in endogenous protein. An additional advantage is that homoarginine, contrary to other chemical derivatizations of amino acid side-chains (Carpenter, 1973), can be transformed to lysine by arginase (*EC* 3.5.3.1; Prior *et al.* 1975) thus preventing lysine deficiency.

Göttingen minipigs fitted with T-shaped cannulas at the ileum and fasted overnight, were given a semi-synthetic diet with 50% of the protein supplied as 'guanidinated' casein and 50% as native casein. Of the former, 95% lysine was transformed into homoarginine. Digesta were sampled for 33 h after the morning meal and were analysed for N and homoarginine.

Apparent precaecal digestibility of protein and homoarginine (HA) of casein in two minipigs

	Intake			Appearance at ileum		Apparent precaecal digestibility (%)	
Animal	Dry matter (g/d)	Protein (g/d)	HA (mmol/d)	Protein (g/d)	HA (mmol/d)	Protein	HA
1	191	19·9	4·93	5·16	0·093	74·1	98·1
2				2·22	0·020	88·8	99·6
1	180	38·4	9·86	7·10	0·089	81·5	99·1
2				6·45	0·148	83·2	98·5
1	382	39·7	9·86	3·67	0·087	90·8	99·1
2				5·34	0·227	86·5	97·7

In contrast to the 74–91% apparent precaecal digestibility of protein (Kjeldahl), 98–99% of the homoarginine disappeared up to the ileum (Table). It can be concluded that about 90% of the ileal N is of endogenous origin when casein is fed.

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