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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Sixty-third Meeting of the Nutrition Society was held at the University of Southampton on Friday, 17 *July,* 1981 *when the following papers were read:*

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Measurement of cell turnover in the gastrointestinal wall of sheep using single injections of [6-^{3}H]thymidine. By J. B. ROWE and S. JAMES, *ICI Pharmaceuticals Division, Mereside, Alderley Park, Macclesfield, Cheshire SK10 4TG*

The incorporation of isotopically labelled thymidine during cell division has been widely used to measure cell production and turnover. Following an intravenous injection of [6-^{3}H]thymidine, the index, fractional incorporation (FI), may be determined as the percentage of the total [^{3}H] radioactivity in the tissue sample which is present in the DNA fraction (Tew & Taylor, 1978). The use of this index to estimate cell turnover accounts for the variability of total [^{3}H] radioactivity that may occur between tissues.

The objectives of the studies reported here were: (1) to determine the variability of FI measurements between samples and between animals, (2) to measure the relative contribution of the gastrointestinal tract to cell turnover in the whole body and (3) to examine the effect of the physical form of the diet on the FI of the rumen epithelial cells. One mCi [6-^{3}H]thymidine was infused into the jugular vein over a 1 h period immediately prior to sampling in all sheep, except that used to measure distribution of thymidine in the whole animal, which received 2 mCi over the same period. Samples of tissue were digested in potassium hydroxide and DNA precipitated using perchloric acid. The precipitate of DNA was washed twice with chilled ethanol and then counted. The supernatant and washings were also counted.

In the first experiment the FI was measured in four samples of rumen epithelium taken from different sites in each of four animals, given 800 g ground alkali-treated straw and 200 g lamb-weaner concentrate/d (see Table). The coefficients of variation for FI of rumen epithelium between animals on the same diet were 5% and between samples taken from the same animal 11%. In the second experiment an estimate of the relative production of cells was made for each tissue by multiplying the FI for the tissue by its weight. On this basis, cell production in the rumen epithelium tissue and the small intestine comprised 3 and 8% respectively of total body production. The sheep was given 1 kg dried beet pulp diet/d. In the third experiment the effect of diet on FI was further examined.

			FI	
Diet	No. of animals	Samples/ animal	Rumen epithelium	SE
Ground straw, pelleted	4	4	42·5	1·1
Finely ground cereal	1	4	42·0	2·9
Dried beet pulp	1	2	50·0	—
Mature grass, chopped	1	4	59·2	2·7

These results suggest that the amount of cell synthesis in the rumen epithelium is affected by the physical form of the diet. However, since this only accounts for approximately 3% of total cell production, it may be of little importance in the over-all N economy of the animal.

Tew, K. D. & Taylor, D. M. (1978). *Eur. J. Cancer*, **14**, 153.

Quantitative changes in the rumen fermentation of sheep associated with feeding monensin. By J. B. ROWE, A. DAVIES and A. W. J. BROOME, *ICI Pharmaceuticals Division, Mereside, Alderley Park, Macclesfield, Cheshire SK10 4TG*

The changes in the molar proportions of rumen volatile fatty acids (VFA) when monensin is given to cattle are well established. The effect of monensin on the rates of end-product formation during rumen fermentation has not been comprehensively quantified. Prange *et al.* (1979) and Van Maanen *et al.* (1979) estimated the effect of monensin on the rate of propionate production in cattle, and Allen & Harrison (1979) measured the effect on acetate production in sheep. In the study reported here, the rates of production of acetate, propionate, butyrate and methane were each measured in seven sheep. Three animals were given a diet containing 33 mg monensin/kg and four animals the same diet without monensin. The diet was fed continuously from a moving belt feeder suspended above the animals. Each animal received 500 g hay and 600 g concentrate/d (consisting of (g/kg concentrate) barley 800, soya bean 170, minerals and vitamins 30) and was acclimatized to the respective diet for six weeks before measurements of rumen fermentation were made. The rates of VFA production and interconversion were measured using separate continuous infusions of [U-^{14}C]acetate, [2-^{14}C]propionate and [1-^{14}C]butyrate, as described previously (Rowe *et al.* 1981). Methane production was measured from a total collection of expired and eructated gas (see Table). The mean concentrations of VFA (mmol/l) in rumen fluid for the control and monensin-treated animals respectively were acetate: 70·9, 78·9; propionate: 24·6, 37·1 and butyrate: 16·3, 9·5. The extent of interconversion of C between acetate and butyrate was reduced in the presence of monensin and it is suggested that this was due to the lower bacterial population density in the monensin-treated animals (22·3 and $9·8\times10^9$/ml rumen fluid for control and monensin treatment respectively) resulting in reduced secondary fermentation.

It appears that approximately 20% more ME was available to the animal from rumen fermentation when monensin was given in the diet, principally from an increased propionate production and lower levels of CH_4 loss.

	Control		Monesin	
Parameter	gC/d	MJ/d	gC/d	MJ/d
Useful energy:				
Acetate production*	110	4·01	102	3·72
Propionate production*	12	0·51	49	2·09
Butyrate production*	25	1·14	22	1·00
Total 'useful' energy	—	5·66	—	6·81
Wasteful energy:				
CH_4 loss	4·4	0·323	2·7	0·198
Acetate–butyrate interconversion	72	0·052	50	0·030
Total 'wasteful' energy	—	0·375	—	0·228

*Amount available after accounting for interconversion between VFA.

Allen, J. D. & Harrison, D. G. (1979). *Proc. Nutr. Soc.* **38,** 32A.
Prange, R. W., Davis, C. L. & Clark, J. H. (1978). *J. Anim. Sci.* **46,** 1120.
Rowe, J. B., Davies, A. & Broome, A. W. J. (1981). *Proc. Nutr. Soc.* (In the Press).
Van Maanen, R. W., Herbein, J. H., McGuillard, A. D. & Young, J. W. (1979). *J. Nutr.* **108,** 1002.

Direct utilization of whole soya bean as a human food. By A. PROŃCZUK, *Institute of Human Nutrition, Warsaw Agricultural University (SGGW-AR), 166 Nowoursynowska St., 02–766 Warsaw, Poland*

Increasing interest in cultivation of soya bean, due to its high productivity and good nutritional value, has resulted in a two-fold increase of its world yield during the last ten years. Due to the lack of proper technology for production and utilization of soya bean protein products (isolates, texturates, analogues, etc.) and because of nutritional and economical reasons, it seems that, at least in the countries where legumes have been used for years as human food, direct utilization of whole soya bean should be considered.

Since 1978, when Polish breed soya bean 'Ajma' was introduced for large scale cultivation, research has been directed to find the best, most economical method of processing this soya bean for the purpose of utilizing it in food preparations (Prończuk *et al.* 1981). From the technological and nutritional point of view, the best method of processing was found to be soaking dehydrated soya beans (1 kg soya bean in 3–5 dm^3 of water) for 4 h, cooking it for 60–90 min in fresh water and after removing the excess water, using it for food preparations. This method of processing eliminated 97% of the tripsin inhibitor activity from the beans and lowered the content of soluble carbohydrates, consisting of flatulance factors, by 70–80%. After soaking and cooking, 'Ajma' soya beans contain 34 g soluble carbohydrates/kg dry matter whereas the traditionally processed white beans and peas contain 31 and 50 g/kg DM respectively.

The evaluated nutritive value based on the content of eighteen nutrients and biological value of protein for processed soya beans have been shown to be much better, especially in reference to protein, fat, calcium, phosphorus, magnesium, manganese and riboflavin, than for white beans and peas. The amount of utilizable protein in a cooked soya bean was 242 g/kg DM compared to 112 and 129 g/kg DM for peas and white beans respectively. Utilizing the work out process, it was possible to prepare more than forty different dishes which had a good organoleptic value measured by the five point scale method. Evaluation of consumer acceptability of dishes with soya bean were also favourable. Further large scale feeding studies are needed to get a definite answer as to much soya bean can be used in this way as food; however, the results already obtained are quite promising.

Prończuk, A., Swiderski, F., Roszkowski, W. & Wronowski, S. (1981). *Acta Alimentaria Polonica* (In the Press).

Toxicity of legumes and the assessment of protein quality. By G. B. REAIDI and A. E. BENDER, *Department of Nutrition, Queen Elizabeth College, London W8 7AH*

Guidelines for breeding improved varieties of legumes include the suggestion that toxic factors should be reduced but, apart from pancreatic hypertrophy to detect trypsin inhibitors, no methods have been suggested (Hulse *et al.* 1977).

Specific tests for each of the toxins occurring in legumes would be very lengthy so an attempt was made to find whether toxins as a group would be revealed during a routine assay for protein quality.

Protein was evaluated by the standard 10 d NPU method (Bender & Miller, 1953) and by the 28 d PER method (Association of Official Analytical Chemists, 1975). Raw red and white kidney beans, *Phaseolus vulgaris*, proved fatal to rats within a few days when they provided 50% of the protein in a diet containing 100 g protein/kg (Reaidi & Bender, 1981). When cooked they had PER 1·4 and NPU 43 (see Table).

Effect of raw kidney beans (Phaseolus vulgaris) *on protein quality (fed at 100 g protein/kg diet)*

	10 d NPU assay			28 d PER assay		
Diet	Food intake g	Weight change g	NPU	Food intake g	Weight change g	PER
Casein	262	99	65	235	75	2·6
Casein—cooked white beans (7·5:2·5)	226	73	60	235	64	2·2
Casein—raw white beans (7·5:2·5)	118	−4	39	146	22	1·3
Cooked white beans—cooked red beans (5:5)	151	9·5	43	167	25	1·4
Cooked white beans—raw white beans (9:1)	132	−12·5	31	—	—	—
Cooked white beans—raw white beans (8:2)	123	−19	24	—	—	—
Cooked white beans—raw white beans (7·5:2·5)	124	−22	24	102	−5	−0·4

The replacement of 25% of the protein of a casein diet with cooked white beans lowered the NPU from 65 to 60 and PER from 2·6 to 2·2 because of their lower quality. The replacement with raw beans in the same proportion reduced these values to 39 and 1·3 respectively, presumably because of their toxicity. The reduction in food intake itself lowers PER but has no effect on NPU. The effect of toxins was also shown when raw beans were added to a diet of cooked beans. With 10% of the protein supplied from raw beans the NPU decreased from 43 to 31, and with 20%, it decreased further to 24.

It is concluded that the addition of suspected material to a standard diet will reveal the presence of toxins in a 10 d assay period by a reduction in NPU.

Association of Official Analytical Chemists (1975). *Official Methods of Analysis* 12th Ed. Washington DC: Association of Official Analytical Chemists.

Bender, A. E. & Miller, D. S. (1953). *Biochem. J.* **53**, vii.

Hulse, J. H., Rachie, K. O. & Billingsley, L. W. (1977). In *Nutritional standards and methods of evaluation for food legume breeders.* Ottawa, Canada: IDRC.

Reaidi, G. B. & Bender, A. E. (1981). *J. Sci Fd Agric.* (In the Press).

The ascorbic acid content of thirteen varieties of potato. By PATRICK KEMP and THOMAS C. KEMP, *Agricultural Research Council, Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

Potatoes are an important source of ascorbic acid in the British diet but unlike apples which contain 5–30 mg ascorbic acid/100 g according to variety (Paul & Southgate, 1975) are not often sold by varietal name.

Five early varieties (lifted in July 1979, Suttons Foremost, late September), three second earlies (lifted late August) and five maincrop varieties (lifted late September) were stored at 10° until mid-December and afterwards between 8–15°. All except Foremost are National Institute of Agricultural Botany recommended varieties grown and lifted under similar conditions and were supplied by them as selected undamaged tubers matched for size (100 g). All tubers remained firm, free from sprouts and disease. Ascorbic acid content but not dehydroascorbic acid was determined on January 6th, February 16th and March 16th 1980 by the indophenol method as in the Official Methods of Analysis of the AOAC (1980). Pentland Javelin from another source was used to examine the effect of size. The content of 50 g tubers was 9·52 mg/100 g, 400 g tubers 9·34; 100, 200 and 300 g tubers gave intermediate values. Home Guard and King Edward had the highest values for ascorbic acid and all varieties except Desirée lost ascorbic acid over the 10 week experimental period. No correlation could be found between ascorbic acid content and any other characters listed in the NIAB classified list 1980/81.

(Between tuber variation ±5%)

		Ascorbic acid mg/100 g fresh weight		
	Variety	January 6th	March 16th After 10 weeks	%
Early	Suttons' Foremost	11·6	8·8	76
	Home Guard	18·1	14·1	78
	Maris Bard	11·9	10·2	86
	Pentland Javelin	10·9	9·9	91
	Ulster Sceptre	10·2	7·6	75
Second	Maris Peer	10·7	8·6	80
	Wiljo	9·6	9·2	96
	Red Craigs Royal	9·4	9·1	97
Maincrop	Desirée*	*10·2	*10·4	*100
	King Edward	15·6	12·8	82
	Maris Piper	13·2	11·1	84
	Pentland Crown	12·9	9·3	72
	Pentland Dell	10·9	8·9	82

*Desirée gave a red coloured filtrate which obscured the end-point.

Association of Official Analytical Chemists (1980). *Official Methods of Analysis*, p. 746. [William Horwitz, editor]. Washington DC: AOAC.

Paul, A. A. & Southgate, D. A. T. (1975). In *McCance & Widdowson's The Composition of Foods* [A. A. Paul and D. A. T. Southgate, editors]. London: H.M. Stationery Office.

Changes in brown adipose tissue mitochondria during cafeteria feeding in the rat. By SARAH L. BROOKS, NANCY J. ROTHWELL and M. J. STOCK, *Department of Physiology, St. George's Hospital Medical School, Tooting, London SW17 0RE*

We have demonstrated that the high levels of diet-induced thermogenesis (DIT) seen in hyperphagic cafeteria fed rats resembles non-shivering thermogenesis, and is due to sympathetic activation of brown adipose tissue (BAT) (Rothwell & Stock, 1981). Nicholls (1976) has proposed that thermogenesis in BAT results from a proton conductance pathway in the inner mitochondrial membrane which allows uncoupled respiration and the activity of this pathway can be estimated from the binding of guanosine diphosphate (GDP) to BAT mitochondria. In the present experiments we have studied the progressive changes in GDP binding over 30 d of cafeteria feeding and in response to acute noradrenaline (NA) injection.

Ninety-six male, Sprague-Dawley rats (Charles River, UK), aged 35 d, were divided into eight groups and allowed free access to water and a pelleted stock diet (PRD, Christopher Hill Gp. Ltd) while half the animals also received a choice of highly palatable food items for 30 d (cafeteria group). After 3, 15 and 30 d of cafeteria feeding and 10 d after withdrawal of the cafeteria diet, six control and six cafeteria fed rats were injected with NA (25 μg/100 g body-weight s.c.) and killed 1 h later, together with six saline-injected rats from each dietary treatment.

Hypertrophy of BAT mass occurred in all cafeteria rats and a doubling of mitochondrial GDP binding was seen in cafeteria fed rats on day 3 (control 170±41, cafeteria 356±92, pmol GDP/mg mitochondrial protein; $P<0.05$, mean values ± SEM), day 15 (control 230±25, cafeteria 527±65; $P<0.001$) and day 30 (control 265±25, cafeteria 384±20; $P<0.001$) and total GDP binding in the interscapular depot was increased by 3–4 fold. Injection of NA, 1 h prior to sacrifice, caused 180 and 430% increases in GDP binding in control (484±145 pmol GDP/mg P; $P<0.05$) and cafeteria fed rats (1506±150; $P<0.001$) respectively, compared to untreated groups, and this may be due to an unmasking of binding sites already present in the mitochondrial membrane. Linear Scatchard plots obtained from control and cafeteria fed animals at 15 d indicated a single class of binding sites with the same affinity (0.22 μM GDP) for all animals, although the maximum number of binding sites was markedly elevated in NA-treated and cafeteria fed animals. Returning cafeteria rats to the stock diet for 10 d resulted in a decline in GDP binding (control 200±27, cafeteria 271±44 pmol GDP/mgP; not significant) although interscapular BAT mass, and GDP binding after NA treatment (475±152) remained significantly higher than controls (238±48; $P<0.01$).

These results provide further evidence for the involvement of the BAT mitochondrial proton conductance pathway in the diet-induced thermogenesis exhibited by cafeteria fed rats.

Nicholls, D. G. (1976). *Eur. J. Biochem.* **62**, 223.
Rothwell, N. J. & Stock, M. J. (1981). *Pflugers Archiv.* (In the Press).

Acute and chronic effects of sucrose and glucose on thermogenic responses and weight gain in the rat. By NANCY J. ROTHWELL, M. ELIZABETH SAVILLE and M. J. STOCK, *Department of Physiology, St. George's Hospital Medical School, Tooting, London SW17 0RE*

Previous studies (Sharief & Macdonald, 1979) on the acute effects of carbohydrates on oxygen consumption ($\dot{V}O_2$) in the rat revealed a greater thermic response to sucrose than glucose. However, these experiments were performed on fasted animals given very small quantities of carbohydrate (2·6–4·3 kJ/rat) under diethyl ether anaesthesia. We have made similar measurements on normally fed animals given larger quantities of sucrose and glucose and have avoided the use of anaesthetics. The chronic effects of these carbohydrates on weight gain and thermogenic capacity were also investigated.

Gastric intubation with sucrose or glucose (40 kJ diluted in 4 ml of water) produced almost identical increases in $\dot{V}O_2$ (% increase; glucose 18·6±2·1, sucrose 18·9±4·8; mean±SEM, *n* 8) in adult, male Sprague-Dawley rats. The time course of the thermic response was also identical, and intubation with 4 ml saline had no effect on $\dot{V}O_2$.

In a separate experiment, eighteen male rats (average weight 180 g) were maintained on a pelleted stock diet (PRD) with either water, 10% glucose or 10% sucrose solutions to drink for a period of 15 d.

Total metabolizable energy intake (kJ/d) was similar for glucose (300±20) and control (260+5) rats, but was significantly elevated in the sucrose fed group (320±6; $P<0·01$). The fractions of total intake derived from sucrose and glucose were 0·45 and 0·34 respectively. However, sucrose fed rats gained significantly less weight, in spite of their hyperphagia, than either control or glucose fed animals (final body-weight (g) control 266±5, glucose 262±2, sucrose, 254±2; $P<0·05$) although the intake/unit gain was reduced in all animals allowed sugar solutions (g gain/MJ eaten, control 26·1±1·0, glucose 21·6±0·3; $P<0·05$, sucrose 18·5±0·5; $P<0·001$). Resting $\dot{V}O_2$ was similar in control and glucose fed animals but was elevated in those receiving sucrose. Both groups given sugar solutions exhibited significantly greater thermogenic responses to noradrenaline injections. Hypertrophy of interscapular brown adipose tissue (IBAT) occurred in both sucrose and glucose fed rats (mass of IBAT (mg) control 176±10, glucose 299±44; $P<0·05$, sucrose 325±13; $P<0·001$).

These results indicate that increasing voluntary energy intake in the rat by presenting glucose or sucrose solutions to drink does not necessarily increase the rate of weight gain, and this response may be due to an enhanced thermogenic capacity and hypertrophy of BAT. The acute effects of glucose and sucrose on $\dot{V}O_2$ were similar, and therefore the differential effects of chronic treatment on weight gain and $\dot{V}O_2$ may be related to either the greater energy intake or the greater proportion of intake derived from the sugar solution in sucrose fed rats.

Sharief, N. & Macdonald, I. (1979). *Proc. Nutr. Soc.* **38**, 83A.

Methane production in lambs fed high- and low-roughage diets. By M. J. L. CLAPP (introduced by J. B. ROWE), *ICI Ltd, Pharmaceuticals Division, Alderley Park, Macclesfield, Cheshire SK10 4TJ*

Groups of fifteen Welsh cross lambs were fed on either a high roughage ((g/kg diet) 500 NaOH treated straw, 250 sugar beet, 100 maize, 80 fish meal, 50 molasses, 20 minerals) or a low roughage ((g/kg diet) 600 barley, 280 maize, 50 sugar beet, 50 molasses, 20 minerals) diet during a 10 week growth trial. Continuous collection of expired and eructed gas was made over 24 h for each lamb using open circuit respiration hoods (Tamminga, 1976) during weeks 1–3 and again during weeks 7–9, and analysed for methane and hydrogen concentrations. The relationships between methane production (Y, l/d) and digestible energy intake (X, MJ/d) were determined by regression analysis for animals fed on each diet:

high-roughage diet: $Y = 12{\cdot}04+1{\cdot}026\ (\pm 0{\cdot}331)X$
low-roughage diet: $Y = 9{\cdot}085+0{\cdot}015\ (\pm 0{\cdot}146)X$.

This compares with $Y = 2{\cdot}89+1{\cdot}75\ (\pm 0{\cdot}157)X$ quoted by Murray *et al.* (1978) for sheep fed on lucerne. The slope of the regression line for low roughage diet was significantly ($P<0{\cdot}05$) different from the others indicating that a single predictive equation for methane production may not be appropriate to all diets. Blaxter & Clapperton (1965) suggested an equation $C_M = 1{\cdot}30+0{\cdot}112D+L(2{\cdot}37-0{\cdot}05D)$ relating methane production (C_M in kCal/100 kCal gross energy intake) with digestibility of dietary energy (D) and level of feeding relative to maintenance (L). Using this equation and the maintenance energy requirements estimated by Thomson *et al.* (1979), the predicted methane production for lambs fed high- and low-roughage diets exceeded those measured by 12·5 (±3·8) and 168·5 (±25·6)% respectively.

The relationship between energy intake and methane production appear to be suitable for predictive purposes when feeding high-roughage diets. However, methane production may be overestimated when feeding diets containing very low levels of fibre. This is not due to hydrogen production and it seems likely that the low rumen pH and high propionate concentrations are contributory factors.

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Thomson, D. T., Fenlon, J. S. & Cammell, S. B. (1979). *Br. J. Nutr.* **41,** 223.

Preliminary studies on the nutritive values of winged beans. By C. JEYAKUMAR HENRY, A. MOSTAFA and J. P. W. RIVERS, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*

The winged bean (*Psophocorpus tetragonolobus*) is an indigenous foodstuff in parts of south-east Asia. The seeds, which are dried for storage, are given a prolonged soaking and boiling before consumption. This procedure both softens the tough seed coat making them edible and inactivates the anti-nutritional factors present (Jaffe & Korte, 1976). However, the prolonged cooking time of about 3–4 h is expensive on fuel. We have therefore examined the effect of various soaking and germination procedures on soaking time (time required to attain 80% increase in weight) and cooking time (time required to soften 90% of the seeds). In addition, sufficient material was available to make some preliminary studies on the effects of dehulling and of germination upon the nutritive value of the protein.

Soaking mixture	Soaking temperature (°C)	Soaking time (h)	Cooking time (min)
Water	20	26	220
	40	21	200
1% Sodium chloride	20	28	240
	40	19	200
0·75% Sodium bicarbonate	20	18	180
	40	12	140
Germinated beans	20	26	110

The Table shows that soaking and cooking times were reduced by soaking at higher temperatures. Allowing the beans to germinate was much more effective than soaking; only soaking in a solution of $NaHCO_3$ at 40° gave comparable cooking times. Germination of seeds for 72 h also improved the nutritive value of the beans. Beans were incorporated into a semi-synthetic diet to give 120 g protein/kg diet and net protein utilization (NPU) measured by the method of Miller & Bender (1955). When germinated, cooked beans were used as the protein source, the NPU was $56 \pm 0{\cdot}98$. With water-soaked cooked whole beans a value of $46 \pm 0{\cdot}7$ was obtained, although this could be increased to $54 \pm 1{\cdot}6$ if the beans were dehulled. Chattopadhyay & Banerjee (1953), working with various legumes, reported an increase in nutritive value on germination while Jaya *et al.* (1975) observed no significant difference on germination. The present study, however, showed an increase in NPU on germination and dehulling of winged beans. It is suggested that the tough seed coat may be responsible for lower NPU and long cooking time. Germination may not only be removing the seed coat but also reducing the levels of anti-nutritional factors.

We thank Professor A. Duncan, University of Florida, for the sample of seeds. C.J.H. acknowledges the financial support of the Nestlé Foundation and the ORS fee support scheme.

Chattopadhyay, H. & Banerjee, S. (1953). *Ind. J. Med. Res.* **41**, 185.
Jaffe, M. G. & Korte, R. (1976). *Nutr. Rep. Int.* **14**, 449.
Jaya, T. V., Krishnamurthy, K. S. & Venkatraman, L. V. (1975). *Nutr. Rep. Int.* **12**, 175.
Miller, D. S. & Bender, A. E. (1955). *Br. J. Nutr.* **9**, 382.

Evaluation of an in vitro immunological test for predicting the suitability of soya-bean products for feeding to preruminant calves. By J. W. SISSONS and R. H. SMITH, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT* and A. NYRUP, *Aarhus Oliefabrik A/S, Aarhus C, Denmark*

Digestive disorders shown by calves given liquid feeds containing certain soya-bean products were tentatively ascribed to a gastrointestinal allergy (Sissons & Smith, 1976), and evidence adduced that glycinin and β-conglycinin components of soya-bean protein were mainly responsible (Kilshaw & Sissons, 1979). Findings that soya-bean concentrate, prepared by alcohol extraction of soya-bean meal, neither caused digestive disorders nor contained much immunologically active glycinin or β-conglycinin whilst heated soya-bean flour and soya-bean protein isolate both contained large amounts of these substances and provoked gastrointestinal disorders, supported this view. Studies were made to test whether an in vitro immunological assay would predict the deleterious qualities of differently treated soya-bean products.

Products N, R, S and T, were prepared respectively by extracting fat-free soya-bean flour with 96, 65, 65 and 55% (v/v) ethanol at 60, 60, 78 and 78°C. Four cannulated calves were sensitized by giving them a series of test feeds containing heated soya-bean flour until they showed gastrointestinal disorders to further feeds of flour. They were then given feeds containing steamed products, N, R, S and T in two successive 4×4 latin square experiments. Mean values for small intestine transit time, ileal flow rates during 3 and 21 h collection periods and nitrogen absorption to the ileum (Sissons & Smith, 1976) are given in the Table.

Product	Transit time (h)	Ileal flow rate (g/h) 3 h	Ileal flow rate (g/h) 21 h	Net N absorption (proportion of intake)
N	2·11	467	174	0·18
R	2·77	154	74	0·69
S	2·69	249	103	0·74
T	3·02	132	76	0·66
SED (df)	0·21(11)	56(11)	11(7)	0·06(7)
5% LSD	0·46	124	27	0·13

Haemagglutination inhibition assay (Kilshaw & Sissons, 1979) of saline extracts of products N, R, S and T respectively gave titre values ($\log_2$ reciprocal of highest dilution of extract giving agglutination inhibition) of 12, 5, 3 and 2 for glycinin and 13, 3, 3, and 2 for β-conglycinin. After steaming only product N gave measurable titres; 10 and 11 for glycinin and β-conglycinin respectively.

The results support the idea that haemagglutination inhibition assays indicate whether or not a soya-bean product given in a liquid feed will cause gastrointestinal disorders in calves. They also show the importance of controlling temperature and concentration of ethanol in treating soya-bean products to remove deleterious factors.

Kilshaw, P. K. & Sissons, J. W. (1979). *Res. Vet. Sci.* **27**, 366.
Sissons, J. W. & Smith, R. H. (1976). *Br. J. Nutr.* **33**, 421.

Artificially reared well-fed and underfed rats: measures of body and organ growth in adulthood. By J. L. SMART, D. N. STEPHENS and H. B. KATZ, *Department of Child Health, The Medical School, Manchester M13 9PT*

Rats were reared without their mothers from postnatal day 4 (Smart *et al.* 1981). They were fed a milk substitute by intermittent infusion through a gastric cannula until day 21. Underfed pups (ARU) received 44% of the milk substitute fed to well-fed pups (ARC) between days 4 and 21, and continued to be undernourished till day 25 when they were restored to *ad lib.* feeding. Littermates of the artificially reared young were reared by their mothers (MR). All rats were subjected to a battery of behavioural tests between 17 and 26 weeks (results to be reported elsewhere) and killed at 32 weeks. There were eleven ARC, ten ARU and ten MR rats.

Age (d)	Measure	ARC % difference compared with MR	*P*	ARU % difference compared with ARC	*P*
25	Body-weight	+1	NS	−41	<0·001
224	Body-weight	−5	NS	−9	<0·05
	Body length	−5	<0·001	−1	NS
	Tibia length	−2	NS	−4	<0·05
	Gastrocnemius muscle weight	−17	<0·001	+3	NS
	Epididymal fat pad weight	+34	<0·05	−39	<0·01
	Brain weight	−9	<0·001	−4	NS
	Heart weight	−3	NS	−5	NS
	Kidney weight	−4	NS	−6	<0·05
	Adrenal weight	−14	<0·001	−2	NS
	Liver weight	−6	NS	−6	NS
	Stomach weight	−7	NS	−5	NS
	Intestine length	−2	NS	−4	NS

The previously underfed rats, as expected, showed a number of deficits compared with ARC animals. They were of lower body-weight, were leaner, and had deficits in kidney weight and tibia length, but not in nose-rump length. Although ARC rats achieved similar growth in weight to MR rats both during artificial rearing and subsequently (Smart *et al.* 1981), they were significantly shorter but fatter animals at 32 weeks, and they also had smaller brains, adrenals and gastrocnemius muscles. The most likely cause of these differences would appear to be the composition of the milk substitute fed to the ARC rats, which was relatively low in protein and high in carbohydrate compared with rats' milk (Messer *et al.* 1969).

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Production and consumption of legumes in Poland. By W. SEKUŁA, *Institute of Food and Nutrition, Warsaw, Poland*

The position of grain legumes in Polish agriculture has been gradually declining since the end of the 1950s. While in 1950 their total production (both for food and for fodder) amounted to almost 600 000 tonnes, it decreased to some 200 000 tonnes in 1979. The diminishing importance of production of this crop was the result of two opposite trends in the cultivated area and in the average yields: there was namely a sharp reduction in the area under legumes as they were steadily replaced by other crops and rather a slight improvement in the yields. In 1950, legumes occupied 3·5% of total arable land in Poland and only 1·2% in 1979. This unfavourable situation resulted mostly from changes concerning grain legumes grown for fodder. Whereas at the beginning of the fifties their share in total production of legumes was 80%, in 1979 it fell to approximately 50%.

Production of grain legumes used as human food (most widely cultivated in Poland are; peas, beans and broad beans of which peas holds the dominant position) decreased between 1950 and 1960, fluctuated during most years of the two next decades and only in 1978 and 1979 registered a sharp increase. It was first of all a consequence of much better yields although an increased area contributed to it too. An explanation of this growing importance of grain legumes cultivated for food lies in the higher prices paid to farmers.

	1950	1970	1978	1979
Area (1000 ha)				
Total grain legumes	564	285	182	172
Human food	105	44	51	64
Animal food	459	241	132	108
Production (1000 tonnes)				
Total grain legumes	567	362	252	229
Human food	111	64	101	108
Animal food	456	298	151	121
Yields (kg/ha)				
Human food	1060	1460	1990	1700
Animal food	1080	1230	1150	1120

Like in many countries, consumption of grain legumes in Poland decreases as they are steadily replaced by other food groups. According to the results of household budget surveys an average consumption of legumes was 1 kg/person in 1979. Their role in the diet was greater in rural households than in urban ones. In this first group, consumption was 1·5 kg/person and only 0·8 kg/person in the latter.

Inhibition of the hypothalamic oedema induced by gold thioglucose and bipiperidyl mustard. By S. A. JAGOT, P. C. FREEMAN, M. E. JAKOBSON, P. D. ROGERS and G. P. WEBB, *Departments of Paramedical Science and Biology, North East London Polytechnic, Romford Road, London E15 4LZ*

Gold thioglucose (GTG) has been said to induce obesity by lesioning a gluco-receptive satiety area in the ventromedial hypothalamus (Debons *et al.* 1977). However, bipiperidyl mustard (BPM), a chemically unrelated compound, induces a similar lesion and Caffyn (1972) has suggested that both compounds act by exerting an acute inflammatory effect on the blood vessels of the hypothalamus leading to necrosis of ventromedial neurones.

We have used the leakage of intravenously injected Evan's blue dye as a simple semi-quantitative method for investigating the inflammatory response to the two compounds, GTG and BPM. We confirmed the earlier finding of Caffyn (1972) that, within a few hours of administration, both compounds induce a specific increase in the permeability of hypothalamic blood vessels. Exposure to a necrotizing dose of either compound 14 d previously markedly reduced ($P<0.001$ in all cases) the increase in vascular permeability due to subsequent injection of either compound. The increase in vascular permeability due to GTG was abolished when it was given to mice pre-treated with sodium salicylate. In a parallel study salicylate was also shown to prevent GTG-induced obesity. In addition, either a tenth of the necrotizing dose of GTG, or a necrotizing dose of GTG together with sodium salicylate prevented the oedema due to a necrotizing dose of GTG given 18 h later but not to a necrotizing dose of BPM. A small priming dose of BPM was ineffective in blocking oedema due to the subsequent administration of either compounds.

These observations are consistent with the suggestion that GTG and BPM act initially on different receptor sites within the hypothalamus although ultimately inducing their effects via a final common pathway. These results do not support the idea that GTG lesions are due to non specific toxic effects on cerebral blood vessels where the blood–brain barrier is deficient (Perry & Liebelt, 1960), but rather they suggest that GTG binds strongly with receptors in hypothalamic neurones, provoking an inflammatory response if the dose is sufficient.

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