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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATION

The Three Hundred and Forty-seventh Meeting of the Nutrition Society was held at The Physiological Laboratory, Department of Physiology, Downing Street, Cambridge CB2 3EG on Wednesday 23 July 1980, when the following papers were read:

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The thermogenic response to comfortable temperature extremes in lean and obese subjects. By S. E. BLAZA* and J. S. GARROW, *Clinical Research Centre, Watford Road, Harrow, Middx HA1 3UJ*

It has been suggested that one of the factors responsible for the onset and maintenance of obesity may be a generalized thermogenic defect (Jung *et al.* 1979). That cold exposure produces less marked responses in obese individuals than in their lean counterparts has been confirmed many times since originally observed by Winslow *et al.* 1937. All such experiments have used extreme cold, unlikely to be often experienced by most individuals, as the stimulus, thus ensuring the maximum response. The present work was undertaken in order to establish whether lean or obese subjects differed in their thermogenic response to temperatures which would be tolerated in life.

Five lean and five obese women were studied as inpatients. All subjects were given an 800 kcal (3·36 MJ) diet. On day 2 each subject's 'comfort zone' was determined by cycling the temperature in the direct calorimeter and recording the subject's preference, expressed by a pointer and dial system. Previous experiments had shown this method to give reproducible results.

Five 24 h measurements of heat loss in the direct calorimeter were made: a control measurement at the midzone temperature, 'warm' and 'cool' at the extreme comfort temperatures, and exercise and food, the results of which will be presented elsewhere. A latin square design corrected for the influence of reduced energy intake. The mean RMR before each run was relatively constant.

Table 1 shows the effect of comfortable cool temperature in the two groups. The values given are not a result of variations in deep body temperature or heat balance. In response to a cool environment the lean subjects increased heat loss by 7·7%, while in the obese subjects heat loss decreased by 2·1%. However, this does not mean that obese subjects have a lower energy expenditure at any temperature. In the comfortable temperature it was 155% of the lean subjects, and in the cool environment only 140% of the lean subjects. Thus, a failure of cold-induced thermogenesis alone cannot explain the maintenance of the obese state in these women.

		Comfort		Cool extreme			
Group	W/H^2 (kg/m^2)	RMR (ml O_2/min)	HL (watts)	RMR (ml O_2/min)	HL (watts)	Minus control HL	% over control
Lean	20·4	193	62	198	67	4·8	7·7
Obese	36·0	257	96	264	94	−2·0	−2·1
$\frac{\text{Obese}}{\text{Lean}} \times 100$		133	155	133	140		

Jung, R. T., Shetty, P. S., James, W. P. T., Barrand, M. & Callingham, B. A. (1979). *Nature, Lond.* **279**, 322.
Winslow, Herrington & Gagge (1937). *Am. J. Physiol.* **120**, 1.

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0029–6651/80/3903–347A $00.35

The effect of changes in energy expenditure on energy intake of obese and lean subjects. By MERRIL L. DURRANT, *MRC Clinical Research Centre, Watford Road, Harrow, Middx HA1 3UJ*

Twelve obese (98·8±21·8 kg) and four lean (59·0±8·1 kg) subjects were admitted to a metabolic ward (Garrow *et al.* 1978). The protocol was approved by the Northwick Park Hospital Ethical Committee.

The subjects ate from a refrigerated snack dispensing machine (SID) for 7 d. SID supplied 108 portions of food with an energy content of 12·55 MJ/d. Food was arranged into three meals/d with four different foods/meal. The schedule allowed *ad lib.* feeding conditions with small intermeal intervals for winding on uneaten food. Reloading and checking were done daily.

Subjects were given a set amount of exercise on days 2, 3, 4 or 5, 6, 7. They were required to perform 1000 revolutions on a Monark bicycle ergometer at the highest load they could manage. This varied between 1·5 and 2·0 kponds. Subjects achieved an increase in energy expenditure of 435±105 (range 255–690) kJ/d. A cross over design was used so that half the subjects did exercise on the first 3 d and the remaining subjects did exercise on the last 3 d. Hunger and appetite ratings were made seven times daily on 100 mm linear analogue scales.

Mean daily energy intake from SID (MJ)

	Total days 2–7	Days 2, 3, 4	Days 5, 6, 7	No exercise	Exercise
Obese	5·00±2·16	5·20	4·81	5·04	4·97
Lean	4·86±1·26	4·55	5·17	4·54	5·18

Mean daily hunger and appetite scores

	Days 2, 3, 4	Days 5, 6, 7	No exercise	Exercise
Obese	388	398	415	362
Lean	456	422	439	440

The results show that lean and obese subjects had similar intakes from SID. These intakes were below levels for weight maintenance and both groups lost weight (mean 1·8±0·9 kg for obese and 0·34±0·59 kg for leans). The obese subjects did not increase energy intake when expenditure was raised whereas lean subjects did (**t* 3·9 $P<0·05$). This increase in intake (649 kJ/d) exceeded the increase in expenditure and implies overcompensation on the part of the normal weight subjects. Hunger and appetite scores did not significantly respond to exercise and do not explain the failure of energy balance regulation in either group.

Garrow, J. S., Durrant, M. L., Mann, S., Stalley, S. F. & Warwick, P. M. (1978). *Int. J. Ob.* **2**, 441.

0029–6651/80/3903-347A $00.35

The effect of changes in preload energy content on energy intake of obese and lean subjects. By MERRIL L. DURRANT, *MRC Clinical Research Centre, Watford Road, Harrow, Middx HA1 3UJ*

Fourteen obese (95·9±20·7 kg) and six lean (67·4±14·4 kg) subjects were admitted to a metabolic ward. The protocol was approved by the Northwick Park Hospital Ethical Committee.

The subjects ate from a refrigerated snack dispensing machine (SID) for 7 d. SID supplied 108 portions of food with an energy content of 12·55 MJ/d. Food was arranged into three meals with four different foods/meal. The schedule allowed *ad lib.* feeding conditions with small intermeal intervals for winding on uneaten food. Reloading and checking were done daily.

Subjects were fed 2·51 MJ preloads (milkshakes, soup or fruit juice) three times daily on days 2–3. On days 4–5 and 6–7 subjects were fed preloads which were indistinguishable but contained 1·26 MJ/d or 3·77 MJ/d (Durrant & Royston, 1979). A cross over design was used so that the sequence was alternated for every subject. Hunger and appetite ratings were made seven times daily on 100 mm linear analogue scales.

Mean daily energy intake from SID (MJ)

	Total days 2–7	Days 2–3	Days 4–5	Days 6–7	1·26 MJ preload	3·77 MJ preload
Obese	5·81±2·02	3·28	3·33	3·24	3·44	3·14
Lean	8·58±1·35	6·65	6·15	5·46	6·17	5·44

Mean daily hunger and appetite scores

	Days 2–7	Days 2–3	Days 4–5	Days 6–7	1·26 MJ preload	3·77 MJ preload
Obese	286±182	294	296	268	282	282
Lean	382±130	417	335	400*	365	366

These results show that lean subjects ate more than obese subjects. Both groups show a decline in intake with time. Both groups shift energy intake in the right direction to compensate for the changes in energy intake but in neither group is this difference statistically significant nor does it match the 2·51 MJ difference in preload energy content. Thus, energy intake is imprecisely regulated by all subjects.

Hunger and appetite scores are higher in the lean than the obese group. Although there was no significant difference in hunger scores between test weeks, the normal weight subjects did express more hunger on the last 2 d of the study (*t 2·5, P=0·055) whereas the obese showed less.

Durrant, M. L. & Royston, P. (1979). *Int. J. Ob.* **3**, 335.

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Can dietary linoleate be replaced by linolenate? By W. M. F. LEAT and CHRISTINE A. NORTHROP, *ARC Institute of Animal Physiology, Babraham, Cambridge CB2 4AT*

It is generally accepted that linoleic acid (C18:2ω6) is the major essential fatty acid, which can satisfy the growth requirement of the rat through two generations (Tinoco *et al.* 1979). To what extent linolenic acid (C18:3ω3) can replace linoleic acid over a similar time scale is uncertain since rats fed linolenate have an impaired parturition making it difficult to obtain second generation animals (Leat & Northrop, 1979). By suitable dietary manipulation second generation rats reared on linolenate can now be obtained.

Fifteen 21-d-old female Wistar rats were reared on a basal diet low in essential fatty acids. Group 1 rats (1–6) remained unsupplemented, group 2 rats (7–9) were supplemented with methyl linoleate and group 3 rats (10–15) with methyl linolenate, both at 0·6 μl/g body-weight/d. A fourth group of rats (16–18) was reared on a commercial diet.

At sixteen weeks of age, the mean weight (±SEM) of the rats were: group 1, 192·7±3·54 (6); group 2, 222·0±3·79 (3); group 3, 219·8±6·3 (6); group 4, 236·7±15·2 (3). The mean weight of the rats in group 1 were significantly lower ($P>0·01$) than those of groups 2 and 4. The rats were mated and the results at parturition were; group 1 (basal diet alone), twelve dead pups born to three dams; group 2 (basal diet + C18:2), one dead and twenty-one live pups born to three dams; group 3a (basal diet + C18:3), three pregnant rats carrying thirty live foetuses had an impaired parturition and were killed. The other three pregnant rats of this group (3b) were dosed with methyl linoleate in place of methyl linolenate from the 17th d of gestation. Twenty-six live pups were born to these three animals; group 4 (commercial), thirty live pups born to three dams.

The second generation rats of groups 2 and 3b were weaned at 28 d and fed the basal diet supplemented (0·3 μl/g body-weight/d) with either methyl linoleate or methyl linolenate. At 17 weeks of age the mean weight of the rats fed linoleate was 213·9 g ± 5·46 (11) which was not significantly different from that of rats fed linolenate (201·2 g ± 4·55 (6).) It is concluded that linolenate can replace linoleate for growth of rats through two generations provided that linoleate is fed during late gestation to allow normal parturition to occur.

Leat, W. M. F. & Northrop, C. A. (1979). *J. Physiol., Lond.* **290**, 37P.
Tinoco, J., Babcock, R., Hincenbergs, I., Medwadowski, B. & Miljanich, P. (1979). *Lipids* **13**, 6.

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Intestinal 1α,25-dihydroxycholecalciferol-dependent calcium absorption inhibited by essential fatty acid restriction. By A. W. M. HAY, *Department of Chemical Pathology, University of Leeds,* E. B. MAWER and F. SUTHERLAND JONES, *Department of Medicine, The Medical School, University of Manchester, Manchester M13,* and A. G. HASSAM, M. A. CRAWFORD and P. A. STEVENS, *Nuffield Laboratories of Comparative Medicine, Zoological Society of London, London NW1 4RY*

The active hormonal form of vitamin D_3 1α,25-dihydroxycholecalciferol (1α,25 $(OH)_2D_3$) is known to stimulate calcium transport in the intestine (Hill *et al.* 1973), however, the biochemical basis of this action is still not clear.

Recently we reported (Hay *et al.* 1980) that Ca transport in vitamin D-deficient rats raised on an essential fatty acid (EFA) deficient diet is not activated by vitamin D supplementation 72 h after dosing. We report here that the same effect can be seen with 1α,25 $(OH)_2D_3$ in 6 h suggesting, therefore, a direct role for essential fatty acids in the mechanism of Ca absorption.

An in vitro Ca transport system (Hill *et al.* 1973) was used to assess the effect of EFA restriction. Weaning inbred Wistar albino strain rats were raised on a semi-synthetic D-deficient EFA-adequate diet. After one week on this diet the animals were subdivided, one group remaining on the diet, the other receiving an EFA-deficient diet by substituting hydrogenated coconut oil for the arachis oil in the EFA-replete diet. Two weeks later, and 6 h prior to the experiment half the animals each received 10 ng of 1α,25 $(OH)_2D_3$ orally. Animals were killed by cerebral fracture and the intestine removed immediately for the gut-sac preparation.

The results for Ca transport expressed as the value of ^{45}Ca serosal:mucosal are shown in the Table (mean±SD). The value is low at 2·31 and 2·0 in the D-deficient animals on the EFA-adequate and deficient diets respectively. Dosing with 1α,25 $(OH)_2D_3$ increased this ratio to 4·0 in the EFA-adequate group but no such rise was observed in the EFA-restricted group, the ratio remaining at 2·67. The difference in the value of the ^{45}Ca ratio between the two 1α,25 $(OH)_2D_3$ dosed groups is significant ($P<0·05$).

	EFA-adequate (Arachis oil)	EFA-deficient (Coconut oil)
Vitamin-D deficient	2·31±0·64 (*n* 7)	2·0±0·56 (*n* 6)
1α,25 $(OH)_2D_3$ treated	4·0±1·34 (*n* 7)	2·67±0·69 (*n* 7)

EFA-restriction clearly has an inhibitory effect on 1α,25 $(OH)_2D_3$ mediated Ca transport across the gut and suggests that essential fatty acids play an important role in the transport mechanism.

Hay, A. W. M., Hassam, A. G., Crawford, M. A., Stevens, P. A., Mawer, E. B. & Sutherland Jones, F. (1980). *Lipids* (In the Press).
Hill, L. F., Lumb, G. A., Mawer, E. B. & Stanbury, S. W. (1973). *Clin. Sci.* **44**, 335.

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The origin of urinary nitrate. By B. BARTHOLOMEW, A. BUTT, C. CAYGILL and M. HILL, *Bacterial Metabolism Laboratory, Central Public Health Laboratory, Colindale, NW9*

It has been postulated that there is a relationship between nitrate intake and gastric cancer incidence, and we are investigating the contribution of drinking water nitrate to the total nitrate exposure.

Bartholomew *et al.* (1979) described nitrate balance studies which indicated that 60–70% of an oral challenge dose of nitrate was excreted in the urine within 24 h. In these studies a basal urinary excretion level was achieved by consuming a 'nitrate-free' diet (less than 5 mg detectable nitrate). This basal level did not reach zero within 5 d of the experiment (as shown by Tannenbaum *et al.* 1978) but did not show the wide fluctuations reported by them. Consumption of the 'nitrate-free' diet even for 21 d gave similar results, the basal levels being 12, 24 and 38 mg respectively in three volunteers (compared with 80–100 mg on a normal diet). This nitrate needs to be accounted for and Tannenbaum (1979) has postulated heterotrophic nitrification.

We have assayed nitrate and nitrite in the ileostomy fluid of twenty-three patients given total colectomy for ulcerative colitis at least 5 years previously; the results showed that, in contrast to Tannenbaum's results (a) both NO_3 and NO_2 were present, (b) the maximum level was only 2 mg/d total NO_3+NO_2. Assay of faecal samples indicated the presence of less than 1 mg/d (NO_3+NO_2) on high or low protein diets.

The present results are consistent with previous observations (Hawksworth & Hill, 1971) that dietary nitrate is efficiently absorbed from the upper small intestine and excreted in the urine since on an intake of 80–100 mg NO_3/d only 2% reaches the terminal ileum and only 1% the faeces. There is no evidence here of heterotrophic nitrification by bacteria in the terminal ileum. We still need to account for the urinary nitrate in persons on the 'nitrate-free' diet. We cannot rule out the possibility that this nitrate was in fact dietary nitrate as this is notoriously difficult to measure (Witter *et al.* 1979).

These studies were supported by the Cancer Research Campaign and the Department of the Environment.

Bartholomew, B., Caygill, C., Darbar, R. & Hill, M. J. (1979). *Proc. Nutr. Soc.* **38**, 124A.
Hawksworth, G. & Hill, M. J. (1971). *Br. J. Cancer* **25**, 520.
Tannenbaum, S. (1979). In *Naturally Occuring Carcinogens — Mutagens and Modulators of Carcinogenesis* [E. Miller, J. Miller, I. Hirono, T. Sugimura and S. Takayama, editors]. Baltimore: University Park Press.
Tannenbaum, S., Fett, D., Young, D., Land, P. & Bruce, W. (1978). *Science, N.Y.* **200**, 1487.
Witter, J., Gatley, S. & Balish, E. (1979). *Science, N.Y.* **205**, 1335.

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The effects of meal-feeding in lean and genetically obese mice. By S. A. JAGOT and G. P. WEBB, *Department of Paramedical Sciences, North-East London Polytechnic, Romford Road, London E15 4LZ* and J. W. T. DICKERSON, *Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH*

Meal-feeding or gorging is a well known, if little used, method of increasing adiposity in rodents; meal-fed animals are fatter than continuously-fed controls even if they consume the same amount of food (Miller, 1979).

Twelve lean and twelve genetically-obese (*ob/ob*) 6–7 month old male mice of the C57 BL/6 strain were housed in groups of four and food intake and body-weight monitored during the control period when the mice were fed a commercial pellet diet (FFG(M)-Dixons) *ad lib*. The animals then had access to food for a 3 h period daily; food intake and body-weight changes were measured. The *ob/ob* mice ate significantly more ($P<0.001$) than the lean controls during the control period, however, when food availability was restricted the *ob/ob* food intake was significantly lower ($P<0.001$) than that of the lean controls. Meal-feeding reduced food intake initially in all groups; the food intake of the lean mice returned to control levels within one week of the start of meal-feeding whereas the obese mice were still eating only half of their control intake 16 d after meal-feeding had started. During 16 d of meal-feeding *ob/ob* mice lost weight (7.8 ± 0.54 g) whilst the lean mice gained weight (3.9 ± 0.41 g), both changes were highly significant ($P<0.001$).

We have obtained similar results to those of the *ob/ob* mice with outbred mice made obese with bipiperidyl mustard. When fed only on alternate days lean mice showed a compensatory hyperphagia of 65–80% whereas the *ob/ob* intake increased by less than 20%.

Hollfield & Parson (1958) used meal-feeding to reduce body-weight in obese mice, however, they do not report any differences in the response of lean and obese mice to meal-feeding, indeed both their lean and obese animals lost weight when fed ground pellets for 3 h/d. Our results are similar to those of Panksepp (1974) who reported that rats made obese be ventromedial hypothalamic lesioning did not increase their food consumption and stabilize body-weight when fed for 1 h daily. Changes in total food intake can be due to changes in the efficiency and in the duration of feeding (Morrison, 1977). In our experiments maximum duration of feeding in obese mice resulted in reduced efficiency of feeding.

Hollfield, G. & Parson, W. (1958). *Metabolism* **7**, 179.
Miller, D. S. (1979). In *Animal Models of Obesity*, [M. F. W. Festing, editor]. London: Macmillan.
Morrison, S. D. (1977). *Fedn Proc. Fedn Am. Socs exp. Biol.* **36**, 139.
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Are commercial stock diets suitable for toxicological research? By A. WISE, *Medical Research Council Laboratory Animals Centre, Carshalton, Surrey*

Stock diets for laboratory animals have been formulated using the same criteria of growth and breeding performance, and incorporating the same unrefined feedstuffs, as for farm animals. They are not based on any consideration of the experimental model needed by the toxicologist. It is probable that many differences found between human and laboratory animal responses to oral toxins are related to the diet, but have been attributed to species variation. Few toxicologists appear concerned about dietary variables because they mistakenly believe that stock diets have been standardized (Wise, 1980). In addition, many experiments have demonstrated that the extent of a toxic effect depends on whether the animals have been fed on a stock diet or a semi-synthetic diet. Ershoff (1974) and Kritchevsky (1977) have suggested that most of these results are due to the binding activities of the dietary fibre (DF) in the stock diet.

DF has been measured in thirty-five stock diets by well-established detergent methods (Robertson, 1978) and the pectin component by ammonium oxalate extraction, hydrolysis, carbazole reaction. DF ranged considerably, especially for the fourteen rat and mouse diets (from 83 to 224 g/kg dry). Ranges for diets of other species were (with numbers of diets analysed): guinea pigs 275–343 g/kg (4), rabbits 280–349 g/kg (6), primates 135–171 g/kg (6), dogs 139–191 g/kg (3), and cats 56–139 g/kg (2). There were large variations in the proportion of DF in each fraction: pectin 3–18%, hemicellulose 35–66%, cellulose 18–45%, and lignin 7–21%.

Human nutritionists are concerned by the low DF in refined diets (Cummings, 1978), while the levels in laboratory animal diets are high and variable, thus making extrapolation from animals to man even more difficult than it would otherwise be. It is suggested that low-fibre experimental diet formulations would probably be more suitable for toxicological investigations that are relevant to man, especially as they would make the animal more sensitive to toxic effects by permitting greater absorption of test substances that bind to the dietary fibre in stock diets.

Cummings, J. H. (1978). *Am. J. clin. Nutr.* **31**, S21.
Ershoff, B. H. (1974). *Am. J. clin. Nutr.* **27**, 1395.
Kritchevsky, D. (1977). *Fedn Proc. Fedn Am. Socs exp. Biol.* **36**, 1692.
Robertson, J. B. (1978). *Topics in dietary fibre research*, p. 1, [G. A. Spiller and R. J. Amen, editors]. New York: Plenum Press.
Wise, A. (1980). *Nature, Lond.* **284**, 592.

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Milk clotting and acid secretory capacity of the stomach of young pigs reared solely by the sow or weaned at 21 d of age. By P. D. CRANWELL, J. J. SHAUGHNESSY and R. E. SMITH, *School of Agriculture, La Trobe University, Bundoora, Victoria* 3083, *Australia*

The capacity of the stomach to secrete HCl and milk-clotting enzymes was measured in pigs 3 to 44 d of age. The milk-fed group was reared solely by the sow whereas the creep-fed group was reared solely by the sow for 21 d and was then entirely dependent on solid food (27% crude protein; 2·67% bentonite).

Eight pigs, four from each litter, were prepared with gastric fistulas. Samples of gastric contents (10 g approximately) were collected at 30 min intervals for 3 h following sucking or feeding. One hour following removal of the residual gastric contents by flushing with physiological saline, gastric secretion was stimulated by an intramuscular injection of betazoline-HCl (Histalog; Eli Lilley, Indiana, USA) at 3 mg/kg HCl and milk-clotting activity (Foltmann, 1970) were measured in gastric juice which was collected for 15 min intervals during a further 2 to 3 h. Maximum acid and milk-clotting enzyme output following betazoline-HCl injection were calculated and the results are summarized in the Table.

Age (d)		3–11		12–19		23–30		35–44	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Acid output (mmol H^+/h)	MF	$^{a}2·1$	0·6	$^{bf}3·9$	0·6	$^{cd}8·1$	1·8	$^{e}14·7$	1·4
	CF	$^{a}1·7$	0·3	$^{ab}3·8$	0·6	$^{bd}6·5$	1·5	$^{df}7·1$	1·8
Acid output/unit liveweight (mmol H^+/h per kg)	MF	0·6	0·1	0·7	0·1	0·9	0·2	1·0	0·2
	CF	0·6	0·1	0·8	0·1	1·0	0·2	0·6	0·1
Clotting activity (CU/h)	MF	$^{a}0$		$^{b}18$	4	$^{c}50$	16	$^{d}213$	34
	CF	$^{a}0$		$^{b}18$	5	$^{c}62$	16	$^{d}134$	17
Clotting activity/unit liveweight (CU/h/kg)	MF	$^{a}0$		$^{b}3·5$	0·6	$^{bc}6·5$	2·2	$^{e}16·5$	1·7
	CF	$^{a}0$		$^{b}3·6$	0·8	$^{cd}8·8$	2·0	$^{de}12·3$	1·2
Residual clotting activity in gastric contents (CU/g)	MF	$^{ab}0·9$	0·2	$^{a}0·6$	0·1	$^{ab}0·8$	0·1	$^{c}1·7$	0·3
	CF	$^{abc}1·7$	0·8	$^{ab}0·8$	0·1	$^{b}1·0$	0·1	$^{c}2·2$	0·2

a, b, c, d, e, f, values in each pair of horizontal rows with different superscript letters differed significantly ($P<0·05$).
MF, milk-fed; CF, creep-fed.

At the termination of the experiment the creep-fed pigs had significantly more stomach tissue per unit liveweight than milk-fed pigs (9·4±0·6 *v.* 6·5±0·5 g/kg; $P<0·01$).

Although residual milk-clotting activity was detected in gastric contents from pigs at 3 d of age, betazoline-HCl did not stimulate milk-clotting enzyme secretion in all pigs until 16 d of age. Milk-clotting activity in gastric juice from pigs up to 2 weeks of age is due mainly to chymosin and after this time to pepsin (Foltmann & Axelsen, 1980). Our results would suggest that chymosin and pepsin secretion are controlled by different mechanisms since it has been demonstrated that betazoline-HCl is a potent stimulus for pepsin secretion (Cranwell, 1977).

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Foltmann, B. (1970). *Meth. Enzym.* **19**, 421.
Foltmann, B. & Axelsen, N. H. (1980). *FEBS Proc.* **60**, 271.

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The effect of dietary protein source on fat induced hypertension in rabbits.

By F. W. VAS DIAS, P. G. BURSTYN and T. G. TAYLOR, *School of Biochemical and Physiological Sciences, University of Southampton SO9 3TU*

There is as yet no satisfactory explanation for the difference in blood pressure between vegetarians and omnivores. Studies in this department have shown that fat exerts a hypertensive effect in rabbits which is antagonized by dietary fibre (Husbands & Burstyn, 1980). A hypotensive effect of dietary fibre has also been observed in man (Wright, Burstyn & Gibney, 1979). The purpose of the present study was to investigate the effect of the protein source on the fat induced hypertension in rabbits.

Sixteen weanling NZ White rabbits were divided into four equal groups and offered one of four diets *ad lib.* for five weeks. The dietary variables were protein source (soya-bean meal *v.* fish meal; 24% protein energy) and fat source (palm oil *v.* corn oil; 50% fat energy). The salt content of all diets were made up to 5·5 g/kg. Arterial blood pressures were measured four times weekly under standard conditions and by the same operator using the ear capsule technique of Grant & Rothschild (1934).

The results were analysed by regression analysis of arterial pressure (AP; mm Hg) on time (T in weeks). There was a highly significant ($P<0.001$) linear increase in arterial pressure with time confirming previous observations. Constants from the linear regression equations of AP on T for the protein and fat sources are given below.

Corn oil	(n 8) $AP = 0.83T + 64.4; S_b + 0.42, P<0.01$
Palm oil	(n 8) $AP = 1.30T + 62.8; S_b + 0.46, P<0.01$
Soya meal	(n 8) $AP = 1.04T + 65.9; S_b + 0.36, P<0.01$
Fish meal	(n 8) $AP = 1.09T + 61.3; S_b + 0.37, P<0.01$

There were no significant differences in regression coefficients due to either protein or fat source although the rate of increase of arterial pressure tended to be greater with palm oil than corn oil. There was no evidence of a protein fat source interaction. These results indicate that fat induced hypertension in rabbits is independent of protein source and do not provide support for the suggestion that the lower blood pressure of vegetarians is due to their dependence on vegetable protein.

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Grant, R. T. & Rothschild, P. (1934). *J. Physiol. Lond.* **81**, 265.
Wright, A., Burstyn, P. G. & Gibney, M. J. (1979). *Br. Med. J.* **2**, 1541.

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