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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Seventy-fourth Meeting of the Nutrition Society was held at the Department of Physiology, Downing Street, Cambridge, on Thursday and Friday, 1/2 *July,* 1982 *when the following papers in the form of posters were presented:*

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Computed intakes of essential nutrients by growing rats offered a varied diet. By H. GILLIAN BARR, R. GRAY and K. J. MCCRACKEN, *Agricultural and Food Chemistry Research Division, Department of Agriculture, and The Queen's University of Belfast, Newforge Lane, Belfast BT9 5PX*

It has been established that growing rats exhibit hyperphagia when offered a varied diet (cafeteria-feeding). In some cases this has resulted in 'diet-induced thermogenesis' (Rothwell & Stock, 1979, 1980). Since the concentrations of amino acids, minerals and vitamins are generally lower in the foods offered under a cafeteria-feeding regimen than in the accompanying stock diet the overall diet consumed could be deficient with respect to some essential elements.

A computer program was prepared to calculate various nutrient intakes using food tables (Paul & Southgate, 1978) as a data base. This was applied to the food intake data from the experiment described by McCracken & Barr (1983). The dietary regimens were: stock diet (PRD, Labsure, Poole, Dorset) *ad lib.* (S), stock diet *ad lib.* plus varied diet (VL); stock diet enriched with methionine, minerals and vitamins plus varied diet (VH). Two strains of rat were compared—Sprague–Dawley (SD) (Charles-River, Kent) and Norway Hooded (NH).

The concentrations of selected nutrients (g or mg/MJ ME) are shown in the table, together with the estimated requirements (National Research Council, 1978) for growing rats.

		SD		NH		National Research Council
	S	VL	VH	VL	VH	(1978)
S-amino acids (g)	0·39	0·31	0·38	0·32	0·34	0·38
Calcium (g)	0·47	0·13	0·19	0·09	0·11	0·31
Phosphorus (g)	0·55	0·18	0·23	0·15	0·16	0·25
Thiamin (mg)	0·66	0·19	0·37	0·14	0·20	0·25
Riboflavin (mg)	0·78	0·23	0·45	0·17	0·26	0·19
Pyridoxine (mg)	0·78	0·20	0·41	0·13	0·22	0·38
α-Tocopherol acetate (mg)	5·47	1·56	3·24	1·07	1·78	1·90

The mean ME intakes of stock diet for SDVL, SDVH, NHVL and NHVH were 20, 18, 12 and 8% respectively of total intake. VL and VH were deficient in calcium for both strains of rat. SDVL, NHVL and NHVH intakes of S-amino acids, phosphorus, thiamine, pyridoxine and vitamin E were deficient relative to the National Research Council (1978) requirements.

These computations suggest that more attention should be paid to the intakes of individual nutrients in experiments where hyperphagia is induced using the varied diet regimen.

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No impairment in efficiency of energy utilization in young growing rats kept at 24° and offered a varied diet. By K. J. McCracken and H. Gillian Barr, *Agricultural and Food Chemistry Research Division, Department of Agriculture, and The Queen's University of Belfast, Newforge Lane, Belfast BT9 5PX*

Some reports (Rothwell & Stock, 1979, 1980) indicate that growing rats kept at temperatures below the zone of thermoneutrality and offered a varied diet (cafeteria-feeding) exhibit hyperphagia but that most of the extra energy consumed is liberated as heat. However, Barr & McCracken (1982) found no evidence of 'diet-induced thermogenesis' in growing rats offered a varied diet and kept at 29°. One problem with the varied diet regimen is the uncertainty of nutrient concentration in the diet consumed.

This study was designed to attempt to confirm the reports of 'diet-induced thermogenesis' at temperatures below the thermoneutral zone and to examine the effect of eliminating possible dietary deficiencies using two strains of rat of different growth potential.

Thirty-five Sprague–Dawley (SD) rats (Charles-River, Kent), of initial weight 130 g, and thirty Norway Hooded (NH) rats, of initial weight 90 g, were allocated to one of the following treatments:

1. Stock diet *ad lib.* (PRD, Labsure, Poole, Dorset) (S) (10 SD, 8 NH rats)
2. Varied diet including unsupplemented stock diet (VL) (10 SD, 8 NH rats)
3. Varied diet including stock diet with additional minerals, vitamins and amino acids (VH) (10 SD, 10 NH rats)
4. Slaughtered for initial carcass composition (5 SD, 4 NH rats)

Room temperature was 24°. After a 21-d feeding period, S, VL and VH rats were slaughtered and the carcasses were homogenized and analysed for crude protein (N×6·25), fat and ash. The digestible energy (DE) intakes were determined and metabolizable energy (ME) was calculated as 0·96 DE. Energy retention (ER) was calculated using factors for protein and fat.

The ME intakes of SDS, SDVL, SDVH, NHS, NHVL, NHVH rats respectively were 351, 450, 435, 241, 309 and 303 kJ/d or 1·06, 1·32, 1·26, 1·03, 1·26 and 1·22 MJ/d per kg $W^{0.75}$ ($P<0.001$, SED = 0·023). Carcass gains were (g/d) 8·4, 9·5, 10·6, 4·9, 6·2 and 6·5 ($P<0.001$, SED = 0·26) and the energy contents of the carcass gains were (MJ/kg) 9·5, 14·8, 13·5, 9·8, 16·6 and 16·3 ($P<0.001$, SED = 0·27). Energy retention (MJ/d per kg $W^{0.75}$) was 0·24, 0·41, 0·42, 0·20, 0·42 and 0·43 ($P<0.001$, SED = 0·020). Linear regressions of ER on ME intake using a common intercept of −0·45 MJ/kg $W^{0.75}$ for zero ME intake yielded slopes of 0·65, 0·65, 0·63, 0·69, 0·71 and 0·71 ($P<0.01$).

The results indicate that rats exhibit hyperphagia when offered a varied diet and deposit large amounts of fat and that the efficiency of utilization of energy is unimpaired even though a number of essential nutrients may be marginally deficient (Barr *et al.* 1983).

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Heat production and energy retention of young pigs given low-protein diets. By K. J. McCRACKEN, *Agricultural and Food Chemistry Research Division, Department of Agriculture, and The Queen's University of Belfast, Newforge Lane, Belfast BT9 5PX*

Weight maintenance in young pigs fed low-protein diets *ad lib.* has been attributed to 'diet-induced thermogenesis' (Miller & Payne, 1962; Gurr *et al.* 1980). Some preliminary results are presented which challenge this hypothesis.

The heat production of pigs fed a milk-based starter diet (diet 1, 210 g protein/kg) was measured in a closed-circuit respiration chamber at 29° for at least one 22-h period. Food intake was restricted to 300 g/d and the mean initial heat production was 605 kJ/d per kg $W^{0.75}$. Pigs were allocated to one of four treatments:

T1, diet 1 restricted (140 g/d) T2, diet 2 (60 g protein/kg) *ad lib.*

T3, diet 3 (20 g protein/kg) *ad lib.* T4, starting control.

Heat production was determined every third day over a 21-d period.

The mean metabolizable energy intakes of T1, T2 and T3 were 2·34, 4·76 and 4·40 MJ/d respectively. Weight gains of T1, T2 and T3 were (g/d) 50, 70 and 20 respectively. Heat production declined on all three treatments (Fig. 1). The mean calculated energy retention (ER) was 0·5, 3·1 and 2·7 MJ/d for T1, T2 and T3 respectively. Carcass analysis on the first replicate confirmed the calculated ER and that ER of T2 and T3 was almost entirely due to fat.

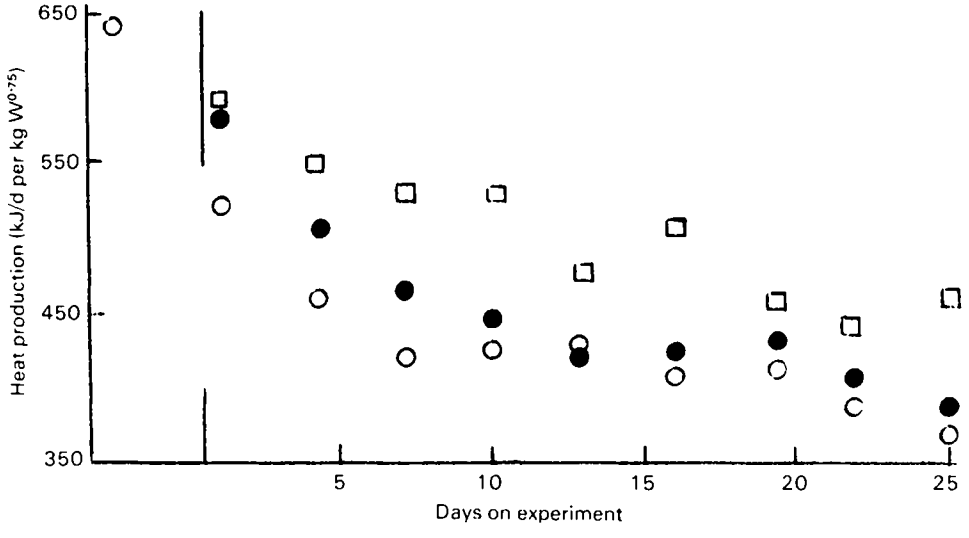

Fig. 1. Mean heat production of pigs (n 3) given restricted amounts (130 g/d) of diet 1 (○), diet 2 *ad lib.* (□) or diet 3 *ad lib.* (●) after initial measurements (n 8) on diet 1.

The results indicate that reducing the growth rate of young pigs either by restriction of energy or protein intake causes a marked reduction in metabolic rate. They do not support reports of increased 'diet-induced thermogenesis' on low-protein diets.

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Gluconeogenesis and fatty acid oxidation in ruminant liver and mammary tissue slices as influenced by methylmalonic and ethylmalonic acids. By K. W. J. WAHLE, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB* and W. D. STEINHOUR and J. M. ELLIOT, *Department of Animal Science, Cornell University, Ithaca, New York* 14853, *USA*

Methylmalonic acid (MMA) inhibits gluconeogenesis from various substrates, including propionic acid, in isolated hepatocytes from rodents (Arinze *et al.* 1979) and inhibits palmitate oxidation in human fibroblasts (Glasgow & Chase, 1976). Propionate is a major precursor of glucose in ruminants and MMA could accumulate in tissues when metabolism of methylmalonyl-CoA (MMCoA) is impaired at the MMCoA-mutase reaction. Palmitate oxidation is considered to be an important source of endogenous acetate in ruminants (Bell, 1980). Accumulation of MMA could thus have a marked detrimental effect on ruminant metabolism.

In sheep liver slices, MMA significantly inhibited glucose production from [2-^{14}C]propionate at 10 mM ($P<0.025$) and 20 mM ($P<0.001$) concentrations and the incorporation of ^{14}C from [*U*-^{14}C]palmitate to glucose at 5 mM ($P<0.025$), 10 mM ($P<0.001$) and 20 mM ($P<0.001$) concentrations. Oxidation of propionate and palmitate was significantly inhibited only at 20 mM-MMA ($P<0.025$ and $P<0.001$ respectively).

In liver and mammary tissue slices from lactating cows, MMA significantly inhibited ($P<0.001$) both gluconeogenesis from propionate (liver) and propionate oxidation at 20 mM concentrations only.

The capacity of liver to oxidize [2-^{14}C]MMA was about 1·5-fold greater than that of mammary tissue, but the capacity of these tissues to oxidize propionate was 7–20-fold greater than that for MMA. Propionate greatly reduced MMA oxidation. Ethylmalonate (EMA), which is excreted in the urine of barley-fed sheep (Lough & Calder, 1976) and which may accumulate in their tissues, also inhibited gluconeogenesis from propionate and propionate oxidation in bovine liver slices at 20 mM concentrations.

Both MMA and EMA inhibit hepatic gluconeogenesis and fatty acid oxidation in slices at concentrations which were relatively high compared with their concentrations in plasma. It is conceivable, however, that these metabolites could accumulate in the cell, particularly in mitochondria, and attain concentrations exceeding those observed in plasma.

K. W. J. Wahle was the recipient of a NATO Senior Fellowship.

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Activity in vitro of fatty acid synthetase and acetyl-CoA carboxylase from lactating bovine mammary tissue as influenced by methylmalonyl-CoA, coenzyme A and short-chain dicarboxylic acids. By K. W. J. WAHLE, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB* and I. P. WILLIAMSON and A. SMITH, *Department of Biochemistry, University of Aberdeen, Aberdeen AB9 1AS* and J. M. ELLIOT, *Department of Animal Science, Cornell University, Ithaca, New York* 14853, *USA*

Methylmalonyl-CoA (MMCoA), a metabolite of propionate, is a competitive inhibitor of fatty acid synthetase (FAS) from various tissues of ruminants and nonruminants (Scaife *et al.* 1978; Wahle & Paterson, 1979). Under conditions of propionate excess or deficient MMCoA mutase activity, which result in impaired propionate metabolism, methylmalonic acid (MMA) can accumulate in animal tissues and it has been implicated as a possible factor in the etiology of the low milk-fat syndrome in dairy cows fed predominantly on grain diets (Frobish & Davis, 1977). MMA (10 mM) does not, however, impair acetate incorporation into fatty acids by mammary gland slices (Croom *et al.* 1981).

Bovine mammary FAS was inhibited in vitro by 15 and 70% using 40 μM and 60 μM MMCoA respectively; inhibition was competitive with respect to malonyl-CoA (K_i = 11 μM). Similarly, 2·5 μM and 6·25 μM coenzyme A inhibited FAS activity in vitro by 34 and 75% respectively and inhibition was also competitive with respect to malonyl-CoA (K_i = 1·7 μM). The K_m for malonyl-CoA was 29 μM.

MMCoA (50 μM) also inhibited the carboxylation of acetyl-CoA (non-competitively) and propionyl-CoA (competitively, K_i = 21 μM) by 10 and 40% respectively. MMA and related short-chain dicarboxylic acids (320 μM) and also ATP (5 mM) enhanced FAS activity in vitro, although concentrations were high when compared with inhibitory concentrations of MMCoA and CoASH.

Because MMCoA has a low KK_i for FAS, its accumulation in mammary tissue of concentrate-fed cows could thus be associated with metabolic events leading to the low milk-fat syndrome. The role of CoASH remains unclear, but it is tempting to postulate a possible regulatory involvement in fatty acid synthesis and oxidation.

K. W. J. Wahle was a recipient of a NATO Senior Fellowship.

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Structural specificity for vitamin A activity in spermatogenesis in the rat.

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The structural specificity for a retinoid given orally to show vitamin A activity in spermatogenesis in rats is narrower than for its systemic role (Thompson *et al.* 1964).

The structural requirements for the characteristic anti-keratinizing action of vitamin A appear much wider when retinoids are added in organ culture than when given orally, because of the narrow specificity of plasma retinol-binding protein (RBP) required for delivery of vitamin A to target tissues (Pitt, 1978).

In order to test their activity in spermatogenesis uninfluenced by the specificity of plasma RBP, retinoids were injected into the testes of rats maintained on retinoic acid as the sole source of vitamin A, so spermatogenesis had ceased.

As judged histologically and in some cases by the activity of carnitine acetyltransferase (*EC* 2.3.1.7), spermatogenesis was restored by small injections of retinol and of retinyl acetate and retinaldehyde, which can be hydrolyzed or reduced to retinol; this confirmed the results of Ahluwalia & Bieri (1971). Retinoic acid, α-retinyl acetate, 3-dehydroretinol (vitamin A_2), 5,6-epoxyretinaldehyde and 4-oxoretinaldehyde were ineffective, even when two injections, each of 250 μg, were given. Similar injections of retinaldehyde oxime had some regenerative activity, as judged by carnitine acetyltransferase assays.

Giving retinaldehyde oxime orally (15 μg/d for 10 weeks) restored spermatogenesis; no retinol but only retinaldehyde oxime could be found in the testes. Giving 3-dehydroretinol orally also promoted spermatogenesis, but only as a consequence of its conversion by the rat to retinol (Border & Pitt, 1981).

Considering also the work of Tosukhowong & Olson (1978) on 15-methylretinol, we conclude that the specificity of retinoids for support of spermatogenesis in rats is narrow, even when delivered directly into the testis. No compound with a modified β-ionone ring was effective and a hydroxyl group appeared to be necessary at the end of the chain.

These requirements are much more stringent than those for the anti-keratinizing action of the vitamin in the trachea in vitro. This finding can be used to support the hypothesis that the action of vitamin A in spermatogenesis is different from that in the systemic function of the vitamin, implausible though that may seem on *a priori* evolutionary arguments.

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Cellular distribution of vitamin A in rat liver studied in isolated fat-storing and other liver cells. By A. M. DE LEEUW, A. BROUWER, K. J. VAN DEN BERG,* A. BROUWER* and D. L. KNOOK, *Institute for Experimental Gerontology TNO and *Radiobiological Institute TNO, PO Box* 5815, 2280 *HV Rijswijk, The Netherlands* (Introduced by M. I. GURR)

The liver is the main storage site of vitamin A in the mammalian body. In humans 70–80% and in rats even 95% of the total vitamin A is stored in the liver, predominantly in the form of retinyl palmitate. The liver consists of different cell types and the distribution of vitamin A within the liver is still poorly described. We have developed methods for the isolation and purification of the four main liver cell types, viz, parenchymal (PC), Kupffer (KC), endothelial (EC) and fat-storing cells (FSC) from rats which made possible the quantitative determination of the content of vitamin A and retinoids in all major liver cell types.

The average yield of isolated cells/g liver was about 40, 6, 22 and 3×10^6 cells for PC, KC, EC and FSC, respectively. The viability was >85% for PC and >95% for KC, EC and FSC. The purity of the isolated cells was at least 80%. The lipid droplets in isolated FSC showed a strong specific vitamin A fluorescence when exposed to light of 328 nm. The nature and the amounts of vitamin A and its derivatives were studied using a high pressure liquid chromatography technique.

As compared with PC, KC and EC, the FSC were found to contain high concentrations of both retinol and retinyl palmitate. Preliminary results showed the presence of at least 1·5 μg retinol and 15 μg retinyl palmitate/million FSC isolated from 3-month-old rats, or 20 μg retinol and 220 μg retinyl palmitate/mg cell protein. PC, KC and EC contained about 0·01, 1·0 and 0·2 μg retinol and 0·1, 6 and 0 μg retinyl palmitate/mg cell protein respectively. Total liver homogenates prepared from 3-month-old rats contained about 0·4 μg retinol and 3·5 μg retinyl palmitate/mg cell protein. The results indicate that the retinol and retinyl palmitate found in the normal rat liver are concentrated mainly in the FSC.

Vitamin A studies in the Maya Indian population of Belize. By P. M. MATHIAS*, *Tropical Metabolism Research Unit, University of the West Indies, Jamaica* and D. HAMILTON, *Department of Nutrition, University of North Carolina, USA* and W. K. SIMMONS, *Caribbean Food and Nutrition Institute, Kingston, Jamaica,* and P. S. E. G. HARLAND, *Department of Medicine, University of the West Indies, Jamaica*

These studies were part of a survey of the health and nutritional status of the Maya Indian population of Belize funded by PAHO. The sample included children aged 5 (n=22), 9 (n=43) and 14 (n=19) years-old from three Maya villages. Dietary data were collected from ten families by 24-h recall. Serum vitamin A and carotene were measured in all children and in seven of the families which comprised eleven adults (aged 23–42, mean 30·5 years) and twenty-five children (aged 3–12, mean 6·8 years).

		Children						Families			
		Vitamin A (μg/l)		Carotene (mg/l)				Vitamin A (μg/l)		Carotene (mg/l)	
Age	No. of subjects	Mean	SE	Mean	SE	Group	No. of subjects	Mean	SE	Mean	SE
5	22	302	24	0·89	0·08	Adults	11	463	46	0·77	0·11
9	43	304	13	1·01	0·11	Children	25	276	22**	0·58	0·05**
14	19	385	24*	1·08	0·13						
Total	84	321	11	1·00	0·08						

*Statistically different from 5 and 9 years-olds (P<0·05).
**Statistically different from adults (P<0·05).

The dietary studies showed that vitamin A intake was low in all families, ranging from 6 to 65% (mean, 27%) of the recommended daily intake. Yellow corn was the major source (>75%) of vitamin A, as carotenoids. This inadequate intake was not confirmed by the biochemical data (Table). Only 11% of all children had 'low' vitamin A values of <200 μg/l, and none had 'deficient' levels of <100 μg/l. However, 22% of the 5-year-olds and 28% of children in the family study had low serum vitamin A and would be considered as populations at risk of vitamin A deficiency by PAHO. In contrast, there were no low values in the 14-year-old and adult groups, whose mean vitamin A levels were significantly higher than those of the 9-(P<0·01) and 5-(P<0·05) year-olds and family children (P<0·0005) respectively. Mean serum carotene was similar in all age groups, although children in the families had a lower level than adults (P<0·05). There was a significant correlation between serum vitamin A and carotene in all children (r 0·41, P<0·001), including those in the families (r 0·45, P<0·05) but not in adults. These results imply that vitamin A status in children may be more dependent on their carotenoid intakes and that their liver stores are lower than in adults. Overall it appears that vitamin A intake of the Maya is adequate on a long-term basis, with the possible exception of the younger children.

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Factors influencing plasma levels of vitamin A and retinol-binding protein during the early phase of recovery of children with severe malnutrition. By P. M. MATHIAS*, *Tropical Metabolism Research Unit, University of the West Indies, Jamaica*

In a previous communication (Mathias, 1982) zinc was shown to influence plasma vitamin A and retinol-binding protein (RBP) in malnourished children in the later stages of their treatment. This report, part of the same study, looks at factors affecting vitamin A and RBP in the early phase of recovery. Experimental details are as before (Mathias, 1982). The table shows admission and maintenance values for vitamin A and RBP in the different malnourished groups.

				Vitamin A (μg/l)				RBP (μg/ml)			
		Days treated		Admission		Maintenance		Admission		Maintenance	
Group	No. of subjects	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Kwashiorkor	8	15	2	248	62^{a}	350	59^{b}	32·6	$7·4^{a}$	44·4	$5·3^{b}$
Mar-Kwash	6	10	3	141	36^{a}	602	61^{c}	17·3	$3·0^{a}$	57·2	$6·5^{b}$
Marasmus	5	9	2	150	47^{a}	444	35^{b}	24·3	$4·8^{a}$	50·6	$9·1^{b}$
Under-nourished	3	7	1	230	108^{abc}	516	128^{abc}	23·1	$6·9^{a}$	40·5	$6·8^{ab}$
Total	22	10	1	194	30^{a}	462	38^{bc}	25·3	$3·3^{a}$	48·8	$3·5^{b}$

Values with the same units in the same column or row that do not have common superscripts are significantly different ($P<0·05$).

On admission there was no difference in vitamin A and RBP between the groups. The maintenance diet supplied 585 μg vitamin A, 0·6 g protein/kg body-weight and 400 kJ/kg body-weight per d (a protein–energy ratio of 2·5%). Vitamin A and RBP increased in all groups, with an overall highly significant ($P<0·0005$) increase in both values, by the end of maintenance. Those children ($n=6$) with 'deficient' plasma vitamin A levels of <100 μg/l had highly significant ($P<0·005$) increases in vitamin A (49±10 to 482±60 μg/l) and RBP (12·6±1·8 to 48·4±7·4 μg/ml) in a mean of 8±2 d. No child displayed clinical vitamin A deficiency.

The results show that contrary to previous findings (Arroyave, 1969; Reddy *et al.* 1979) neither a high protein diet nor a massive vitamin A dose is required to stimulate a rise in vitamin A and RBP in plasma of malnourished children. The balance between dietary protein and energy, rather than a specific effect of protein, is probably the important variable which influences liver RBP sysnthesis, and hence the mobilization of liver vitamin A stores, during the early phase of recovery. The results also indicate that liver vitamin A reserves were adequate in children with 'deficient' plasma vitamin A levels. Plasma levels of RBP alone did not indicate any differences in protein status between the malnourished groups.

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The effect of two retinoids on the response of an inbred mouse strain to the bladder carcinogen *N*-butyl-*N*-(4-hydroxybutyl)nitrosamine (BBN). By R. M. HICKS, J. A. TURTON, C. N. TOMLINSON, J. GWYNNE and K. NANDRA, *Department of Cell Pathology, School of Pathology, Middlesex Hospital Medical School, Riding House Street, London W1P 7LD* (Introduced by M. I. GURR)

The potential of two synthetic retinoids, namely *N*-(tetrazol-5-yl)-retinamide (TR) and *N*-(4-hydroxyphenyl)-retinamide (HPR), to inhibit the development of bladder cancer in the B6D2F1 mouse has been investigated. Following exposure to the bladder carcinogen BBN, this particular mouse strain develops carcinoma *in situ* which progresses rapidly to a very aggressive, flat, invasive, poorly differentiated transitional cell carcinoma of the bladder.

145 Female B6D2F1 mice were dosed by stomach tube with 3 mg BBN/week for 10 weeks. They were then placed on a placebo- or retinoid-containing diet for a further 19 weeks, killed and the bladders removed and examined histologically.

In the group which had received the placebo diet, 21% had hyperplastic urothelia and 66% had bladder cancer ranging from carcinoma *in situ* to solid, infiltrating, poorly differentiated transitional cell carcinomas which invaded the full thickness of the bladder wall. Animals maintained on a diet containing 1 mM-TR were similar to the placebo-fed group in their tumour response to the BBN, with a 20% incidence of urothelial hyperplasia and a 62% incidence of bladder cancer. Comparison of the histology demonstrated there to be little or no improvement in the grade of the cancers in these TR-treated animals. By contrast, those animals pretreated with BBN and then fed 1 mM-HPR had a 49% incidence of hyperplasia but only a 25% incidence of bladder cancer. Furthermore, the urothelia in these animals were strikingly better differentiated than those in the placebo-fed or TR-treated groups; for example, only one animal with carcinoma *in situ* was found in the HPR group, by comparison with thirteen in the placebo-fed and seventeen in the TR-fed groups.

These results show that following a fully carcinogenic treatment with 30 mg BBN, feeding HPR is effective over a 5-month period in reducing the incidence and severity of neoplastic disease in the mouse bladder. By contrast, TR had less effect on the neoplastic response of the urothelium to BBN. These findings suggest that retinoids may prove to be organ-site specific and effective only in their particular target organs. The results with HPR suggest also that this, or other organ-specific retinoids, may ultimately prove to be useful in controlling carcinoma *in situ* and invasive cancer of the bladder in man, unlike most of the currently available treatment modalities which are successful only with slow growing exophytic, relatively benign tumours.

Skeletal development as an index of retinoid toxicity. By J. A. TURTON, R. M. HICKS and J. GWYNNE, *Department of Cell Pathology, School of Pathology, Middlesex Hospital Medical School, Riding House Street, London W1P 7LD* and R. HUNT, L. PALMER and P. B. MEDAWAR, *Transplantation Biology Section, Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ* and C. M. HAWKEY, *Wellcome Laboratories of Comparative Medicine, Zoological Society of London, Regent's Park, London NW1 4RY* (Introduced by M. I. GURR)

In a study demonstrating that aspirin protected mice from retinoid toxicity, Harrison (1977) used mortality and bone fractures as end-points of retinoid intoxication. Using Harrison's report as a base-line, the present investigations were undertaken to develop a more sensitive method of quantifying retinoid toxicity.

Retinoids were added to diets to give concentrations from 0·25 mM to 2·0 mM/kg diet and fed to female F344 rats or B6D2F1 mice. At post-mortem, blood samples were taken and measurements of the body and organs were made.

In rats fed 1 mM- or 2 mM-*N*-(ethyl)-retinamide (NER) for 109 weeks, or 0·8 mM- or 1·6 mM-13-*cis*-retinoic acid (CRA) for 74 weeks, there was a dose-related reduction in body-weight. After 49 weeks feeding with 2 mM-NER, relative weights of the stomach, heart, kidney and brain were increased, and the spleen was decreased. Significant reductions were seen in haemoglobin, packed cell volume and fibrinogen while reticulocytes were increased. White cell values, including monocytes, were not affected.

In rats fed 0·8 mM- or 1·6 mM-CRA for 78 weeks, femoral periosteal diameters were 2·46 mm and 2·22 mm, respectively, and 2·77 mm in control animals. In sectioned femora the endosteal diameters measured 1·60 mm in controls and 1·33 mm and 1·13 mm in rats fed 0·8 mM- and 1·6 mM-CRA respectively. Cortical bone thickness was not significantly affected and measured 0·58 mm in control rats and 0·54 mm in both CRA fed groups. Relative femur weight was reduced in a dose-related fashion in CRA-fed animals but femur length was unaffected. The reduction in periosteal diameter thus reflected a reduction in medullary cavity diameter and demonstrated that significant cortical bone remodelling had taken place in retinoid-fed rats.

Weanling rats were fed 0·4 mM-, 0·8 mM- and 1·6 mM-CRA or 0·25 mM-, 0·5 mM- and 1·0 mM-vitamin A acetate (VAA) for 7 weeks. With increasing levels of each compound there was a related reduction in body-weight and relative femur weight. A trend of reduction with increasing dose level was also seen in femur length and periosteal and endosteal diameters. There were no clear trends in relative weights of the spleen, thymus or lymph nodes. Similar studies have been carried out with B6D2F1 mice. For example, with VAA at 0·25 mM, 0·5 mM and 0·75 mM concentrations, increasing levels caused dose-related reductions in body-weight, femur length and periosteal diameter, but relative weights of the lymph nodes and spleen were increased.

It is concluded that measurements of femoral periosteal and endosteal diameters are a simple, quick and accurate quantitative index of retinoid toxicity in vivo.

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Immunopotentiation by retinoids in rats and mice. By R. HUNT, L. PALMER and P. B. MEDAWAR, *Transplantation Biology Section, Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ* and J. TURTON, J. GWYNNE and R. M. HICKS, *Department of Cell Pathology, School of Pathology, The Middlesex Hospital Medical School, Riding House Street, London W1P 7LD* (Introduced by M. I. GURR)

Immunopotentiation by vitamin A is well-known (Dresser, 1968; Floersheim & Bollag, 1972) and many retinoids have vitamin A activity (Bollag, 1979). In this study, five retinoids were tested for their influence on the survival time of skin allografts and on the sizes of peripheral lymph nodes, spleens and thymus.

The retinoids were mixed into the normal diet of the animals at a concentration of 0·25 mmol/kg or multiples of this dose. Vitamin A acetate, 13-*cis*-retinoic acid and β-carotene are available in the form of stable, gelatinized beadlets. *N*-Ethyl retinamide and hydroxy *N*-ethyl retinamide were dissolved in glyceryl tricaprylate prior to mixing into the powdered diet.

Female F344 rats were used and the effect of the retinoids was most clearly seen in older animals which had been treated for more than 1 year. The rejection of Lewis female skin by female F344 rats is a strong reaction, across a major histocompatibility barrier, but with age the mean survival time increases allowing differences between treatments to be seen. The differences in the older rats was not significant but showed a dose-related response. In younger rats there was no difference seen in any of the retinoid treatments on skin allograft rejection. The rats also showed changes in their lymphoid organ weights but only those fed 13-*cis*-retinoic acid were significantly different.

When these retinoids were tested in female hybrid mice ($C57B1/6 \times DBA_2F1$) a very sensitive system of allograft rejection could be used since they are able to recognize the H–Y antigen when grafted with skin from male $C57B1/6 \times DBA_2F1$ mice. With all the retinoids the rejection times were faster than control- or placebo-fed animals, with the exception of β-carotene fed at a dose of 0·5 mmol/kg.

The hypertrophy of peripheral lymph nodes was significant in all the retinoids at all doses but enlargement of spleen and thymus varied with the retinoid and dose used.

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Uptake and retention of vitamin B_{12} and its analogues in neonatal piglets. By NADIA M. F. TRUGO and J. E. FORD, *National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

Sucking piglets in the farrowing pen consume faeces and contaminated litter (Sansom & Gleed, 1981) which are rich in natural analogues of vitamin B_{12} but with no vitamin activity for higher animals. Thus, sows' faeces containing 1·7 μg of 'B_{12}' activity/g, as measured with *E. coli*, mainly comprised cobinamide (factor B) and 2-methyladenylcobamide (factor A). The cobalamins (vitamin B_{12}) contributed only about 0·2 μg/g towards this total (N. M. F. Trugo, unpublished). Kolhouse & Allen (1977) suggested that the uptake and distribution of B_{12} analogues to the body tissues is prevented by vitamin B_{12}-binding proteins (B_{12}-BP) present in the intestine and in the blood plasma. In the piglet the B_{12}-BP are not present in the intestine during the first 2 or 3 weeks of life, and B_{12}-BP present in sows' milk has been pictured as 'bridging the gap' (Ford *et al.* 1975).

The influence of sows' milk on uptake and retention of vitamin B_{12} and two of its analogues (factors A and B) was investigated. Sixteen piglets were weaned at 2 d after parturition and placed in metabolism cages. Four piglets were assigned to each of four treatments. All groups received a formula feed, supplemented in groups 1 and 2 with a purified concentrate of the B_{12} analogues isolated from sows' faeces by solvent partition procedures, and in groups 2 and 4 with sows' milk. Two piglets from each group were killed after 6 d on the experimental diets, and the remaining two after 12 d. Urine, plasma, bile, liver, kidneys, spleen, heart and pancreas were analysed for the presence of vitamin B_{12} and the analogues using differential microbiological assay techniques.

Factor A and factor B were detected in the liver, plasma, urine and bile. Excretion was mainly via the bile. The content in liver was very small in relation to the amounts ingested, suggesting inefficient uptake or poor retention. We found no evidence that the sows' milk B_{12}-BP influenced uptake or retention of the analogues, nor that ingestion of the analogues affected the liver levels of vitamin B_{12}.

N. M. F. Trugo acknowledges the support of CAPES (Brazil) and of the Committee of Vice-Chancellors and Principals of the Universities of the United Kingdom.

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A dietary survey in the Orkney Islands. By NICOLA L. BULL and GLORIA A. SMART, *Ministry of Agriculture, Fisheries and Food, Great Westminster House, Horseferry Road, London SW1P 2AE* and HELEN JUDSON, *Pierowall, Westray, Orkney*

The diets of 43 randomly selected adults (27 women, 16 men) living on the remote Isle of Westray were studied during the summer of 1979. Their ages ranged from 21 to 69 years. Each participant completed a dietary diary for 4 full weeks, recording all food and drink, whether eaten at or away from home. We know of no other recent quantitative dietary survey in the Scottish Islands. The weights of items consumed were estimated from the descriptions, in household measures, provided by each participant. Application of the values selected by Paul & Southgate (1978) to the amounts of food and drink recorded by each of the participants yielded estimates of the intakes of all major nutrients and dietary fibre. The pattern of the diet, in terms of major foods and food groups, was also evaluated as was the apparent nutritional adequacy of the diet. The results will be discussed in full but only selected nutrients are shown in the Table.

		Average intake (per person/d)		Scotland National Food Survey
		Men	Women	
Energy	kJ	11320	8690	9420
	kcal	2704	2076	2250
Protein	g	91·9	77·6	73·6
Fat	g	129	103	103
Carbohydrate	g	313	223	275
Iron	mg	13·1	11·2	11·3
Vitamin D	μg	4·54	3·81	2·47
Dietary fibre	g	16·5	15·0	19·7
Vitamin C	mg	52	49	48
Zinc	mg	11·6	10·4	9·2

The results were not markedly different from those recorded in Scotland by the National Food Survey in 1979 (Ministry of Agriculture, Fisheries and Food, 1981). More fish and shell fish were eaten, however, resulting in higher mean intakes of vitamin D, while the popularity, among active men in particular, of cakes, biscuits and sandwiches contributed to their relatively high fat and carbohydrate intakes. The men also showed a wide range of consumption of alcoholic drinks; the mean alcohol intake by men who drank at all during the study was 25·8 g compared with 2·1 g by women. The full results will be published elsewhere (Bull *et al.* 1982).

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Effect of moderate food restriction in pregnancy on the growth and development of the mammary gland. By D. J. NAISMITH and S. M. ROBINSON, *Department of Nutrition, Queen Elizabeth College, Campden Hill, London W8 7AH*

In a study of breast feeding (14 weeks) in twenty-two normal primiparous women, Naismith & Ritchie (1975) found two subjects only who experienced lactational failure. They differed from the others in one respect only; weight gains during pregnancy were extremely low (4·5 and 5·0 kg). Hytten & Leitch (1971) reported a correlation between performance in lactation and growth in size of the breast during pregnancy. It was suggested, therefore, that failure in lactation might arise from an inadequate food intake during pregnancy (Naismith, 1980).

To test this hypothesis, nine littermate pairs of adult rats (200 g) were given a diet containing 200 g casein/kg for 7 d and were then mated. One of each pair was allowed to feed *ad lib.* while the other was held at the pre-pregnancy level of consumption. The rats were killed on day 22 of pregnancy. Their pups were dissected and weighed. Their mammary glands were weighed and analysed for protein and DNA and their carcasses were analysed for fat protein. Total food restriction amounted to 25%. The results are shown in the table.

	Pups		Mammary gland							
	Weight (g)		Weight (g)		Protein (g)		DNA (mg)		Protein:DNA	
Food intake	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Ad lib.	5·14	0·10	21·64	1·45	1·53	0·16	42·2	3·1	37·2	3·2
Restricted	5·13	0·06	12·56•••	0·76	1·03••	0·07	34·8•	2·3	30·0•	1·8

•$P<0·05$, ••$P<0·02$, •••$P<0·001$.

Neither litter size nor mean pup weight were affected by the moderate food restriction during pregnancy, but the weight of the mammary gland was reduced by 42%. Values for DNA and protein indicated a significant reduction in the cellularity of the gland and also in hypertrophic growth. The carcasses of the dams on restricted food intake contained 39% less fat and 7% less protein, suggesting that energy intake rather than protein intake had been the main determinant of mammary gland growth. This was confirmed in an experiment of similar design, using littermate triplets, in which a second 'restricted' group was given a diet containing 267 g casein/kg. The additional protein consumed did not increase the weight of the mammary gland nor its content of protein and DNA.

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Energy intake of women during the first week post partum. By MARGARET J. WHICHELOW and META GREENFIELD, *Office of the Regius Professor of Physic, University of Cambridge, School of Clinical Medicine, New Addenbrookes Hospital, Cambridge CB2 2QQ.*

The food intakes of 71 women delivering at the Mill Road Maternity Hospital, Cambridge, were assessed throughout the first week post partum. The energy content of the meals supplied by the hospital kitchen was calculated from the weights of the portions served. The items of the diet taken by the patients, the amounts left on the plate and items of food from outside the hospital which were eaten were recorded, at the time they were consumed, in diet diaries. Most patients having a normal delivery and a healthy infant are discharged after 48 h. The energy intakes of such mothers at home was assessed using the same diet diaries and food models to assess portion sizes.

The energy intake supplied by the hospital diet was 9862±1093 kJ/d (2356±261 kcals/d). The mean daily energy intakes with standard deviation of mothers delivering vaginally were: breast feeding, 7 d in hospital 8711±1139 kJ (2081±272 kcal) (n=21) and home after 48 h 8987±1804 kJ (2147±431 kcal) (n=18); and bottle feeding, 7 d in hospital 8602±1603 kJ (2055±383 kcal) (n=7) and home after 48 h 7606±1482 kJ (1817±354 kcal) (n=4). The daily intakes of women delivering by caesarian section tended to be lower: elective caesarian section, breast feeding 8711±484 kJ (1601±408 kcal) (n=5) and bottle feeding 5844 (1396 kcal) (n=2); emergency caesarian section, breast feeding 6216±1880 kJ (1485±449 kcal) (n=8) and bottle feeding 4814±1285 kJ (1150±307 kcal) (n=6). This was due to the low energy value of the intravenous fluids given during the first 2–3 d after operation, before the gradual introduction of oral liquids and then solid food.

The results indicate that the food intakes during the early post partum period were not related to choice of infant feeding, adequacy of lactation, maternal body-weight change or maternal mood. Transient postnatal depression ('baby blues') was, however, related to subjective feelings of decreased appetite (x^2=10·59, P=<0·01).

Although the energy content of the hospital diet appeared adequate for the needs of most patients many items were unpopular, and all the patients ate food brought in from outside. This varied from 3 to 45% of the mothers intake with an average of 11%.

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Effects of heat treatment of milk on its allergenicity and nutritional quality. By L. M. J. HEPPELL, J. E. FORD and P. J. KILSHAW, *Nutrition Department, National Institute for Research in Dairying, Shinfield, Reading RG2 9AT*

In experiments with guinea pigs, McLaughlan *et al.* (1981) found that heat treatment of cows' milk reduced its allergenicity. They suggested that quite severe heat treatment of infant formulas containing cows' milk proteins might be beneficial to the recipient infants. We have conducted similar experiments with skimmed cows' milk and now report the effects of heat treatment on the allergenicity of the milk and on several labile nutrients.

Pint (0·568 l) bottles of fresh, skimmed cows' milk were subjected to varied heat treatments, graded in severity from steaming for 30 min to autoclaving at 121° for 20 min. β-Lactoglobulin, α-lactalbumin and α-casein were than measured, using enzyme immunoassays. Steaming reduced the content of undenatured β-lactoglobulin and α-lactalbumin by more than 95%, whereas casein levels were not reduced even after treatment at 121°.

Groups of five guinea pigs were given unheated or heated milk (500 ml/group per d) for 2 weeks. Twelve days later the animals were tested for systemic anaphylaxis by intravenous injection of 1 ml of the milk preparation that they had been drinking. Serum samples were taken before challenge and tested for I_gG_1 antibody to the individual milk proteins by passive cutaneous anaphylaxis (PCA).

		% PCA +ve to		
Heat treatment	% Anaphylaxis	α-lactalbumin	β-lactoglobulin	α-casein
None	100	60	100	100
Steamed, 30 min	100	0	0	80
115°, 30 min	100	0	0	20
121°, 20 min	80	0	0	20

The capacity of β-lactoglobulin and α-lactalbumin to sensitize the guinea pigs was destroyed by heat treatment, whereas that of casein was slightly reduced.

Severe heat treatment (121°, 20 min) destroyed all the vitamin B_{12}, about 60% of the thiamin and vitamin B_6, and 70% of the asorbic acid. Surprisingly, loss of folate was greater after steaming for 30 min (44%) than after autoclaving at 121° for 20 min (30%). Autoclaving at 121° caused extensive Maillard browning and consequent loss of available lysine (24%), and introduced an unwanted high level of lactulose (170 mg/100 ml).

We conclude that even after severe heat treatment, infant formulas containing cows' milk proteins may remain immunogenic in the human infant. However, such treatment would significantly impair the nutritional quality.

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Torpor in genetically obese mice. By S. A. JAGOT, M. E. JAKOBSON and G. P. WEBB, *Departments of Biology and Paramedical Sciences, North East London Polytechnic, Romford Road, London E15 4LZ*

Mice are generally considered to be homeothermic and this assumption is implicit in their use as animal models in energy balance studies. The defective thermoregulation of ob/ob mice is considered to be exceptional and obviously contributes to the obesity.

Recent work (Hudson & Scott, 1979; Webb *et al.* 1980, 1982) has shown that when fasted or fed on restricted rations mice can exhibit torpor (reversible adaptive hypothermia). Colonic temperatures of 31° are taken to indicate torpor and temperatures of 25° are common. Webb *et al.* (1980, 1982) have also shown that C57Bl/6 ob/ob mice are (i) more liable to fasting-induced torpor than lean mice, (ii) sometimes found in torpor when food is present and (iii) that weight reduction by meal-feeding improves their resistance to cooling.

Daily monitoring of early morning temperatures in five *ad lib.* fed C57Bl/6 ob/ob mice for 7 weeks showed a recorded (i.e. minimum) 20% incidence of 'morning torpor' and in 4% of observations mice were still in torpor 1·5 h later despite the handling stimulus. No incidence of 'morning torpor' was found in a lean control group.

A group of four C57Bl/6 ob/ob mice placed on a meal-feeding regimen for weight reduction showed a high recorded incidence of 'morning torpor' (70%) during the first week of meal-feeding (cf 20% incidence in meal-fed controls) but this recorded incidence declined to zero as meal-feeding (and weight reduction) was continued for 7 weeks and the pre- and post-feeding temperatures also rose to approach those of the control group. These changes may reflect an absence of torpor or a conditioned arousal in meal fed ob/ob mice prior to the expected morning meal period.

Webb *et al.* (1982) suggested that C57Bl/6 mice seemed particularly liable to enter torpor and that the presence of the ob/ob genes on this background exaggerated the tendency. Preliminary results from mice with the ob/ob genotype transferred to a diversified genetic background has shown essentially similar results to those for C57Bl/6 ob/ob mice. Thus entry into torpor when fed *ad lib.* would seem to be a feature of the ob gene and not totally dependent upon a particular genetic background for its expression.

These results have important implications for the interpretation of results using ob/ob mice and they may even question the suitability of some murine models for energy balance and obesity studies.

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The effect of ovariectomy on adiposity. By P. M. HARRIS and D. F. MACHIN, *Applied Biochemistry Division, DSIR, Palmerston North, New Zealand*

It has been suggested that the rapid weight gain resulting from ovariectomy of the female rat is due to an increase in carcass fat content rather than a proportional increase in all body components (Leshner & Collier, 1973). Wade & Gray (1979) suggested this increased adiposity could result directly from the removal of the influence of oestradiol on lipoprotein lipase (LPL) synthesis, thus altering availability of free fatty acids for triglyceride storage to the adipose tissue.

Fourteen Wistar rats, 6 weeks of age, were divided into two groups of equal weight. One group was ovariectomized (6ovX) and one group left intact (6I). Both groups were fed a stock diet *ad lib.* and killed 5 weeks after surgery. Four fat sites were completely dissected and assayed for levels of LPL (Nilsson-Ehle & Schotz, 1976). The carcasses were analysed for body fat.

	Body-weight/g 1 week after surgery		Body-weight/g 5 weeks after surgery		% Fat in carcass 5 weeks after surgery	
Group	Mean	SEM	Mean	SEM	Mean	SEM
6I	90·7	3·7	162·2	5·5	21·1	1·2
6ovX	98·0	4·6	186·1	7·1	19·1	1·8

	Weight of site as % body-weight 5 weeks after surgery				LPL activity (i.u./site) 5 weeks after surgery			
	Group 6I		Group 6ovX		Group 6I		Group 6ovX	
Site sampled	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Abdominal	1·16	0·13	1·21	0·17	19·5	3·4	22·8	1·8
Scapular	0·58	0·04	0·53	0·06	13·9	1·2	16·2	2·1
Perirenal	0·33	0·06	0·38	0·07	7·1	1·8	11·2	1·7
Paramatrial	0·55	0·08	0·40	0·08	10·5	2·5	10·8	3·2

Ovariectomy resulted in rapid weight gain in this strain of rat but did not influence body composition. Individual fat sites did not vary in weight or LPL activity between treatments. This contrasts with earlier work using the Sprague–Dawley strain and it is possible that the present results are due to strain variation. However, these results show that any increase in body-weight resulting from ovariectomy is not necessarily an increase in the proportion of body fat.

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Requirements for maintenance of the growing brushtail possum (*Trichosurus vulpecula*). By P. M. HARRIS and D. W. DELLOW, *Applied Biochemistry Division, DSIR, Palmerston North, New Zealand*

In New Zealand the feral brushtail possum is regarded as a fur producer of some economic significance ($NZ 23 000 000 p.a. 1980). This has led to interest in farming possums and the need for a suitable diet which will permit rapid growth with minimal fat deposition. Dawson & Hulbert (1970) determined the standard metabolic rate (SMR) of the adult possum to be approximately 30% lower than that of eutharians. The maintenance nitrogen requirement of the adult possum (Wellard & Hume, 1981) is also lower than that of eutharian hind-gut fermenters such as the rabbit.

Juvenile feral possums (0·6–1·4 kg) were trapped and adapted to individual caging and a pelleted ration. In three experiments, three groups of six possums were fed three diets differing in nitrogen (2·2–4·0% N; Trial 1), energy (10·9–13·4 kJ digestible energy (DE)/g; Trial 2) or fibre (9–23% acid-detergent fibre; Trial 3). The diets were offered *ad lib.* for an equilibration period of 14 d and then a 10 d collection period. Maintenance protein requirement was determined to be 475 mg N/kg $W^{0.75}$ and maintenance energy requirement as 370 kJ DE/kg $W^{0.75}$. The SMR was obtained throughout the growth period by measuring oxygen consumption under fasting and resting conditions using an open circuit technique. The SMR of five Chinchilla Rex rabbits maintained on one of the experimental diets was measured over the same growth range.

	Approximate adult wt (kg)	Body-wt measured (kg)	Respiratory quotient		Oxygen consumption (l/h per kg $W^{0.75}$)	
			Mean	SE	Mean	SE
Brushtail possum	2·5–4·0	0·9–2·3	0·87	0·02	0·42	0·01
Rabbit	2·8–3·2	0·9–2·1	0·73	0·02	0·75	0·02

Maintenance energy, measured by the respiratory techniques, indicated that the maintenance requirement of the growing possum (on a metabolic body-weight basis) was 44% lower than that of the growing rabbit. However, the maintenance nitrogen requirement of the possum was only 6% lower than that recorded for the rabbit (Cork & Harrop, 1977). This suggests that the lower maintenance energy requirement is a reflexion of differences in either heat production requirement or in utilization of available food energy.

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