Nutritional obesity and body composition

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It will be abundantly evident from other papers in this symposium that obesity is not a common feature in modern farm animals nor should it be inferred from the inclusion of this paper that nutritional obesity is an inevitable consequence of the consumption of animal products. The justification for including this subject rests entirely on the obsession with human obesity in our overnourished western world and the considerable research effort, particularly during the last 15 years, on the development and sequelae of overnutrition, using animal models. However, detailed examination of the voluminous literature reveals that most studies have used the laboratory rat and were undertaken to study food intake or energy metabolism with only a very limited number being primarily designed to examine body composition. The author has therefore been forced to be selective in terms of literature cited and acknowledges difficulty in reaching unifying themes.

It will become clear that the general principles relating to body composition of growing and adult animals which have been elucidated and developed during the last 40 years apply equally well during the development of obesity as during undernutrition. It is therefore fitting to begin this paper with a tribute to those who so painstakingly laid the basis of our understanding of growth and body composition and, in particular, to two research groups from Cambridge, namely the late Sir John Hammond and his colleagues (McMeekan, 1940*a*,*b*,*c*; Palsson & Verges, 1952; Palsson, 1955) and McCance & Widdowson and their colleagues (Spray & Widdowson, 1950; Dickerson & McCance, 1961; Cabak *et al.* 1962; Lister & McCance, 1967; Widdowson, 1968).

Hyperphagia and consequential obesity have been induced in the laboratory rat by offering high-fat diets (Mickelsen *et al.* 1955), by gastric intubation (Cohn *et al.* 1957), by offering concentrated sugar solutions (Muto & Miyahara, 1972) or by offering a varied 'cafeteria' diet (Sclafani & Springer, 1976). Spontaneous obesity was observed in the squirrel monkey (*Saimiri sciureus*) given semi-purified diets (Ausman *et al.* 1981) and obesity was induced in sheep by long-term feeding of pelleted diets (Blaxter, 1976; Blaxter *et al.* 1982). The present paper will primarily examine the extent to which the means of inducing nutritional obesity in the laboratory rat affects chemical and anatomical aspects of body composition but will also take account of species differences where possible. In addition, consideration will be given to (1) the effect of dietary fat composition on the development of obesity and on the composition of body fat, and (2) variations in the energy content of weight gain during hyperalimentation or of weight loss during fasting or restricted energy intake.

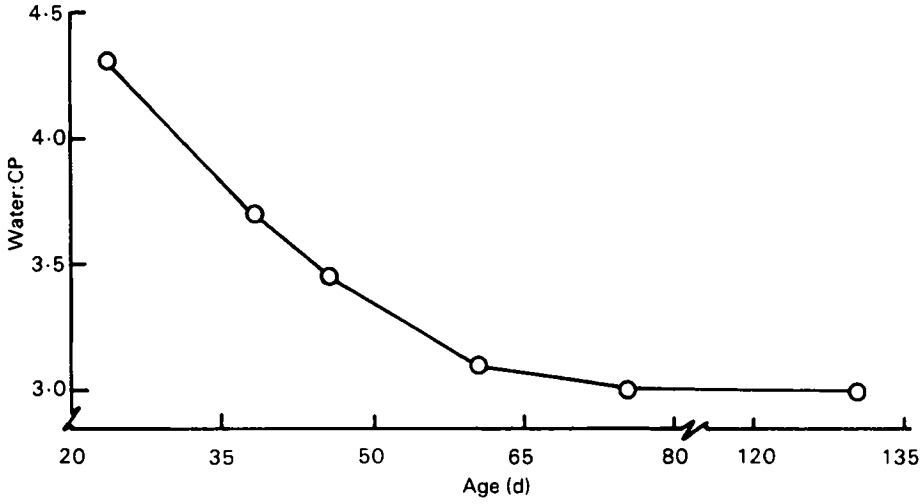

Fig. 1. Total body water:crude protein (nitrogen × 6·25; CP) in Osborne Mendel rats (calculated from the results of Schemmel *et al.* (1969)).

Chemical composition of the empty body

If we take the view that glycogen is quantitatively unimportant then we are left with four major components of the body, i.e. protein, ash and water (the lean body mass) and fat. Since water is the major component of the lean body mass any changes in body water can significantly affect body composition. The major factor affecting body water in the growing animal is undoubtedly age or stage of physiological maturity.

It can be calculated from the results of Schemmel *et al.* (1969) that, in the rat, water:crude protein (nitrogen × 6·25; CP) declines from a post-weaning value of about 4·5 to 3·1 by 60 d of age (Fig. 1) and then remains constant. A similar relation is observed in the mouse (Eisen & Leatherwood, 1979; Rucklidge, 1982) whereas the results of Kotarbinska (1969) on pigs indicate a rapid fall in the ratio up to 10 kg followed by a continual decline up to 100 kg. Although the constancy of water:CP is often discussed it is worth noting that considerable variability occurs between individual animals of similar age, weight and nutritional history. For example the range of water:CP in 8-kg pigs (Kotarbinska, 1969) was from 4·04 to 4·80 and in 22-kg pigs the range was from 3·68 to 4·38. A similar degree of variability is found in the sheep of Blaxter *et al.* (1982) and in rats.

The close relation between muscle and bone deposition (Elsley *et al.* 1964) and between CP and ash deposition (Kotarbinska, 1969) is well-established. The ash, though structurally and metabolically vital to the well-being of the animal, is a relatively small proportion of the empty-body-weight (EBW), being approximately 0·2 × CP in the pig (Kotarbinska, 1969) and 0·15 × CP in the rat (Barr, 1984). Calculations based on the results of several studies on rats (Cohn & Joseph, 1969; Schemmel *et al.* 1969; McNiven, 1980; Walks *et al.* 1983), one on sheep (Blaxter

et al. 1982) and one on the squirrel monkey (Ausman *et al.* 1981) lead to the general conclusion that the body water–CP–ash relations are unaffected by the plane of nutrition or method of feeding. Two exceptions are worthy of note. First, Heggeness (1965) reported that rats alternatively fed at maintenance or *ad lib.* in a 3 d cycle contained less protein and had a higher water:CP value (3·5) than *ad lib.*-fed controls (3·1) after 60 d on experiment. The second example relates to the cafeteria-fed rat. Barr (1984) observed a significant reduction in the proportion of ash:fat-free weight and in the weight of carcass calcium in rats given an unsupplemented cafeteria diet, due to the very low Ca content of most of the cafeteria foods offered. A further complication is that the body water:CP may be altered in protein deficiency. However, results are conflicting, perhaps due to the type of diet, level of intake and stage of growth at which the deficient diet was given. For example, McCracken (1968, 1975) found that water:CP was higher in protein-deficient rats than in litter-mates given restricted energy, in contrast to the results of Henry & Toothill (1962). Fuller (1983) observed 'a degree of dehydration of the fat-free body mass' of pigs given a small amount of a high-protein diet but did not comment on those offered low-protein diets. However, the low-protein pigs of McCracken & McAllister (1984) lost body water with no change in body protein or else failed to gain water in proportion to protein deposition (Table 1), suggesting that in the pig the ratio is more related to physiological age than to diet.

The results of Schemmel *et al.* (1969) indicate that in the rat, the rate of protein deposition is rapid up to about 70 d and then declines to almost zero by 200 d. Hence we can identify three phases in the development of the rat: (a) up to 70 d of age characterized by a reduction in water:CP, high CP deposition and relatively low fat deposition, (b) unchanged water:CP, reducing protein deposition and increasing fat deposition, (c) maturity where the major change in composition is due to fat accretion. Having established this baseline in terms of the normal response to a low-fat diet offered *ad lib.*, let us examine the effects of nutritional hyperphagia during the early growth phase (Fig. 2). These results (H. G. Barr & K. J. McCracken, unpublished results) relate to rats given a low-fat stock pelleted diet *ad lib.* (C), a high-fat pelleted diet *ad lib.* (HF) or a cafeteria high-fat diet (CAF) for 21 d, the HF and CAF treatments resulting in 15 and 27% increases in energy intake respectively relative to that of the controls. Because of quantitative

Table 1. *Changes in body-weight, water and protein (kg) of young pigs given restricted quantities of a normal diet (200 R) or given low-protein diets (45 or 20 g CP/kg) ad lib. (45 A or 20 A) for 24 d*

	Initial	200 R	45 A	20 A
Empty body-wt	7·8	8·9	9·9	8·3
Water:CP	4·96	4·34	4·03	4·04
Water gained	—	1·14	0·15	−0·50
CP gained	—	0·38	0·16	0·01

CP, crude protein (nitrogen × 6·25).

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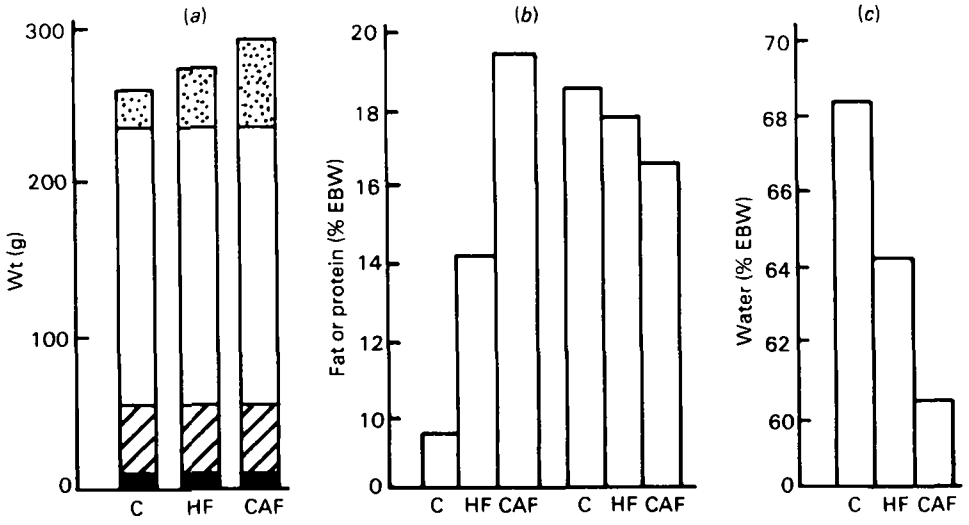

Fig. 2. Body composition of male Sprague-Dawley rats, initial weight 100 g, after 21 d on a low-fat pelleted diet (C), a high-fat pelleted diet (HF) or a varied diet (CAF). (*a*) Weight of ash (■), protein (▨), water (□) and fat (▩). (*b*) Relative amounts of fat or protein (% empty-body-weight (EBW)). (*c*) Relative amounts of water (% EBW).

and qualitative differences in protein intake it is not possible to assert that protein deposition rate is already maximal in the rat fed *ad lib.* on a low-fat chow but it seems likely that this is the case. More importantly the results illustrate the concept advanced by Wilson (1954, 1960) and supported by Elsley *et al.* (1964) that the major effect of nutrition is on the fat content of the body. Hence the still common practice of discussing body composition in terms of proportions of EBW can lead to problems of interpretation when animals of different age, weight or previous nutritional state are compared (Fowler, 1968; Webster, 1986). For example, in the experiment discussed previously (Fig. 2) the effect of reducing water:CP more that offsets the increased fat content of the control rats so that CP:EBW increased from 167 g/kg at 100 g live weight to 192 g/kg at 400 g live weight, whereas the increased fat content with the HF treatment maintained CP:EBW almost constant and the still higher fat content of the CAF group caused a reduction in CP:EBW.

It has been inferred from the results in Fig. 2 that, in the rapid growth phase, protein deposition in the rat is maximal on a low-fat diet eaten *ad lib.* Whilst the same criticism of possible limitation of protein intake applies to the results of Schemmel *et al.* (1969), the close agreement between total body protein and age up to around 140 d with both grain and high-fat diets strengthens the view that the rate of deposition of lean body mass cannot be increased in the young rat by increased energy intake. The same appears to hold for the squirrel monkey (Ausman *et al.* 1981).

A reduction in the rate of protein deposition can occur as a result of force-feeding (Cohn *et al.* 1957; Cohn & Joseph, 1960; Barr, 1984) or due to the

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consumption of a low-protein diet, and both situations exacerbate nutritional obesity. The force-feeding effect appears to be due to the method of administration rather that to periodicity of intake (Cohn & Joseph, 1960; Ozelci *et al.* 1977; Schoenborne & Canolty, 1980). There is also an increased efficiency of energy utilization thus resulting in excessive accumulation of fat. This is illustrated by the results of Barr (1984) who achieved similar energy intakes by rats force-fed on a high-fat diet or consuming a cafeteria diet of similar composition. During a 21 d experiment the force-fed group gained only 75% as much protein as the cafeteria group but 1·6 times the weight of fat. Reduced protein deposition will also occur if the diet is deficient in protein and although energy intake may be reduced relative to that of animals fed on a normal diet *ad lib.*, it is possible to have an increase in the energy available for fat deposition (McCracken & McAllister, 1984), again leading to gross alterations in the composition of the body.

Whereas protein deposition in the young rat is unaffected by overfeeding there is clear evidence that accretion of body N occurs in the adult (Schemmel *et al.* 1969; McCracken & McNiven, 1983; Barr, 1984) whether hyperphagia is due to a high-fat diet, force-feeding or cafeteria-feeding. In the study by Schemmel *et al.* (1969) the CP content of 200-d-old rats given a high-fat diet was similar to that of grain-fed animals at both 274 and 423 d but was 13% higher in 330-d-old rats given the high-fat diet. A similar increase (10%) occurred when females were introduced to force-feeding at 10 months of age (McCracken & McNiven, 1983).

Lean body mass increased throughout the life span (5–6 years) of squirrel monkeys exhibiting spontaneous obesity (Ausman *et al.* 1981). Blaxter *et al.* (1982) concluded that the empty-body gain of adult sheep contained 68% fat, 8% CP, 24% water. Analysis of the results of Schemmel *et al.* (1969) for the high-fat rats at body-weights above the 400 d weight of the grain-fed animals, i.e. between 600 and 1043 g for males and 441 and 735 g for females, suggests that the composition of the gain for both sexes was approximately 74% fat, 7% CP, 20% water.

Energy content of body gain

The energy content of the gain for young animals in the rapid growth phase is about 8–10 MJ/kg but the value increases rapidly as the proportion of fat in the gain rises. Thus in the experiment depicted in Fig. 2, the values for the C, HF and CAF animals were respectively 8·1, 10·4 and 12·7 MJ/kg and in force-fed animals over the same age and weight range (Barr, 1984) the value was 17·7 MJ/kg. The energy contents of the gain of sheep (Blaxter *et al.* 1982) and of the obese adult rats of Schemmel *et al.* (1969) are calculated to have been 28·6 and 30·5 MJ/kg respectively. Even higher values (35·3 and 32·3 respectively) were observed in force-fed adult female rats (McCracken & McNiven, 1983) and in cafeteria-fed adult virgin females (Barr, 1984), and there are a number of situations in which the energy content of the gain can be higher than the value for pure triglyceride. For example, the mean value in pigs given a diet containing 46 g CP/kg (McCracken & McAllister, 1984) was 88 MJ/kg and when the dietary CP content was 21 g/kg,

pigs showed increases in fat and energy coupled with weight loss. This phenomenon probably amounts for a major part of the apparent discrepancy in energy balance reported by Miller & Payne (1962). Another example relates to the rat post lactation (Spray, 1950; McCracken & Barr, 1985), where water accumulated during lactation (Spray, 1950; Moore & Brasel, 1984) was replaced by lipid with little or no change in body-weight.

At the other extreme is the well-documented situation of weight gain associated with energy loss in young animals subjected to restricted energy intake (McCracken, 1975; Fuller, 1983; McCracken & McAllister, 1984) or the situation of stable weight associated with energy loss which can occur in the lactating female (Spray, 1950; Barr, 1984).

These examples clearly illustrate the dangers inherent in estimating changes in total body energy from changes in body-weight.

Development of supply and functional organs

Schemmel *et al.* (1969) concluded that the observed increase in body protein content of hyperphagic rats 'could probably be accounted for by increases in blood volume and weight of such organs as liver, heart, kidney and especially the adipose tissue'. It might be expected that the increased flow of substrate would occasion some increase in organ weights similar to the hypertrophy of liver observed during pregnancy and lactation (Robinson, 1986). Significant increases in liver weight (27%) and liver protein (19%) have been observed in young cafeteria-fed rats offered a low-fat diet (Barr, 1984). However, it would appear that increases in liver size are minimal in the adult (Armitage *et al.* 1983; McCracken & McNiven, 1983) and may be due to accumulation of glycogen, water and fat rather than to any increase in functional capacity. McCracken & McNiven (1983) calculated that only about 10% of the increased protein deposition in the body of force-fed adults could be accounted for by liver protein, with perhaps another 20% in adipose tissue, and concluded that there may be some increase in muscle protein during overfeeding. The composition of adult gain of sheep (Searle *et al.* 1972; Blaxter *et al.* 1982) would tend to support this view.

Development of fat depots

Most of the fat deposited in the body is in discrete organs such as the perirenal fat, subcutaneous fat and the genital fat. The results of Schemmel *et al.* (1970) demonstrate that there are major differences in the rate of development of different fat depots in rats which become obese and that sex differences also occur. For example, the interscapular fat depot, which is less that 1% of EBW in the normal rat, increased to 4% in obese males and to 10% in obese females. By contrast the genital fat, which accounts for approximately 3% of EBW in normal adults, only increased to 4% in males and 6% in females. These differences may be partly explained by the extent of hyperplasia or hypertrophy in individual depots. It has been widely accepted that in the mature animal hypertrophy is entirely responsible for fat deposition (Hood, 1983). In the rat most studies have been conducted on the

epididymal fat pad (Knittle & Hirsch, 1968; Czajka-Narins & Hirsch, 1974; Gruen *et al.* 1980) and there is clear evidence of increases in depot size as a result of hypertrophy. In contrast are the results of Faust *et al.* (1978), Gale *et al.* (1981), Obst *et al.* (1981) and Walks *et al.* (1983) who reported large increases in cell number in retroperitoneal and inguinal fat pads with, in some cases, little or no increase in mean cell size. Clearly this subject is too complicated for any full discussion in the present context but it would seem that there are between-species and between-depot variations in development and that generalizations about the development of fat depots are unjustified.

Effect of dietary fat on composition of body fat

This is an area which has received little attention in studies on the induction of nutritional obesity although there is an extensive literature on the effects of dietary fat on the fat composition of farm and laboratory animals (Hilditch & Pedelty, 1940; Garton & Duncan, 1954; Perkins *et al.* 1961; Leat *et al.* 1964; Christie *et al.* 1972; Wahle & Radcliffe, 1977) and certain aspects of fat synthesis and deposition have recently been reviewed (Freeman, 1983; Hood, 1982, 1983; Leat, 1983; Pearce, 1983; Weekes, 1983). It is well-established that, in the non-ruminant, dietary triglycerides can be absorbed and deposited without any structural rearrangement (Garton & Duncan, 1954), that high levels of dietary fat reduce *de novo* lipogenesis and that diets containing high levels of unsaturated fatty acids, particularly linoleic, give rise to high concentrations of unsaturated fatty acids in depot fat (Wahle & Radcliffe, 1977; Boyd, 1978). It has been suggested that, since medium-chain triglycerides are not deposited in the body and presumably are preferentially used as an energy source, diets containing high levels of medium-chain triglycerides should be less likely to give rise to obesity than those containing high levels of unsaturated fat (Lavau & Hashim, 1978; Turkenkopf *et al.* 1982). However, it is not clear from these studies whether the differences observed were mainly due to differences in metabolizable energy intake or to the high efficiency of deposition of dietary triglyceride containing long-chain unsaturated fatty acids.

Composition of weight loss

There are large variations in the composition of weight loss which appear to relate to species, strain, sex, stage of growth, initial degree of obesity and degree of restriction imposed. Searle *et al.* (1972) concluded that the composition of weight gain of adult sheep was similar to that of weight loss observed by Panaretto (1964) and Graham (1967) when animals were losing weight slowly. Thus it would appear that 1 kg weight loss under these circumstances is composed of approximately 650 g fat, 90 g protein and 240 g water and the energy content is 26 MJ/kg. Vander Tuig *et al.* (1980) did not observe any significant effect of energy source (fat *v.* carbohydrate *v.* protein) on the composition of weight loss of obese and lean mice. The weight loss of the obese strain contained a similar proportion of fat (345 *v.* 351 g/kg) but a lower proportion of protein (50 *v.* 122 g/kg) and the energy

Table 2. *Composition of weight loss of adult, female Hooded Lister rats during fasting or restricted intake of a high-protein diet*

	Normal		After force-feeding	
	Fasted	Restricted	Fasted	Restricted
Fat (g/kg)	563	761	733	847
Protein (g/kg)	178	94	73	36
Energy (MJ/kg)	26·4	32·1	30·9	34·6

contents of the weight loss were 14·6 and 16·6 MJ/kg for obese and lean mice respectively. In our laboratory (K. J. McCracken and M. McNiven, unpublished results), weight loss during restricted feeding of a high-protein diet was of a different composition from that observed during total fasting and the composition of weight loss was also different after force-feeding (Table 2). The energy content of the weight loss in these studies with female rats was much higher than that observed (Babirak *et al.* 1974; Walks *et al.* 1983) in male rats during fasting where the values were 23·5 and 18·8 MJ/kg respectively. One of the most bizarre examples of change in body composition is that reported by Cohn & Joseph (1969) where male rats, which had been made obese by force-feeding for 2–3 months, voluntarily consumed low energy intakes when given free access to a laboratory chow. During a 34 d period they lost weight whilst depositing protein and water with the result that their final body composition approached that of normal animals. The energy content of the weight loss under these circumstances was 108 MJ/kg, i.e. almost three times the energy value for triglyceride.

REFERENCES

Armitage, G., Hervey, G. R., Rolls, B. J., Rowe, E. A. & Tobin, G. (1983). *Journal of Physiology* **342**, 229–251.

Ausman, L. M., Rasmussen, K. M. & Gallina, D. L. (1981). *American Journal of Physiology* **241**, R316–R321.

Babirak, S. P., Dowell, R. T. & Oscai, L. B. (1974). *Journal of Nutrition* **104**, 452–457.

Barr, H. G. (1984). Factors influencing food intake and energy balance in the laboratory rat. PhD Thesis, The Queen's University of Belfast.

Blaxter, K. L. (1976). *European Association for Animal Production Publication* no. 19, 129–132.

Blaxter, K. L., Fowler, V. R. & Gill, J. C. (1982). *Journal of Agricultural Science, Cambridge* **98**, 405–420.

Boyd, J. (1978). Drug and dietary manipulation of fat and protein deposition in the growing rat and pig. PhD Thesis, The Queen's University of Belfast.

Cabak, V., Gresham, G. A. & McCance, R. A. (1962). *British Journal of Nutrition* **16**, 635–640.

Christie, W. W., McEwan-Jenkinson, D. & Moore, J. H. (1972). *Journal of the Science of Food and Agriculture* **23**, 1125–1129.

Cohn, C. & Joseph, D. (1960). *Metabolism* **9**, 492–500.

Cohn, C. & Joseph, D. (1969). *Journal of Nutrition* **96**, 94–100.

Cohn, C., Joseph, D. & Schrago, E. (1957). *Metabolism* **1**, 381–387.

Czajka-Narins, D. M. & Hirsch, J. (1974). *Biology of the Neonate* **25**, 176–185.

Dickerson, J. W. T. & McCance, R. A. (1961). *British Journal of Nutrition* **15**, 567–576.

Eisen, E. J. & Leatherwood, J. M. (1979). *Journal of Animal Science* **48**, 810–821.

Elsley, F. W. H., McDonald, I. & Fowler, V. R. (1964). *Animal Production* **6**, 141–154.
Faust, I. M., Johnson, P. R. & Stern, J. S. (1978). *American Journal of Physiology* **235**, E279–E286.
Fowler, V. R. (1968). In *Growth and Development of Mammals*, pp. 195–211 [G. A. Lodge and G. E. Lamming, editors]. London: Butterworths.
Freeman, C. P. (1983). *Proceedings of the Nutrition Society* **42**, 351–359.
Fuller, M. F. (1983). *Journal of Nutrition* **113**, 15–20.
Gale, S. K., Van Itallie, T. B. & Faust, I. M. (1981). *Metabolism* **30**, 105–110.
Garton, G. A. & Duncan, W. R. H. (1954). *Biochemical Journal* **57**, 120–125.
Graham, N. McC. (1967). *Australian Journal of Agricultural Research* **18**, 127–136.
Gruen, R., Kava, R. & Greenwood, M. R. C. (1980). *Metabolism* **29**, 246–253.
Heggeness, F. W. (1965). *Journal of Nutrition* **86**, 265–270.
Henry, K. M. & Toothill, J. (1962). *British Journal of Nutrition* **16**, 125–133.
Hilditch, T. P. & Pedelty, W. H. (1940). *Biochemical Journal* **34**, 971–979.
Hood, R. L. (1982). *Federation Proceedings* **41**, 2555–2561.
Hood, R. L. (1983). *Proceedings of the Nutrition Society* **42**, 303–314.
Knittle, J. L. & Hirsch, J. (1968). *Journal of Clinical Investigation* **47**, 2081–2098.
Kotarbinska, M. (1969). *Instytut Zootechniki Wydawnictwa Wlasne* no. 238, p. 67.
Lavau, M. M. & Hashim, S. A. (1978). *Journal of Nutrition* **108**, 613–620.
Leat, W. M. F. (1983). *Proceedings of the Nutrition Society* **42**, 333–342.
Leat, W. M. F., Cuthbertson, A., Howard, A. N. & Gresham, G. A. (1964). *Journal of Agricultural Science, Cambridge* **63**, 311–317.
Lister, D. & McCance, R. A. (1967). *British Journal of Nutrition* **21**, 787–799.
McCracken, K. J. (1968). Energy metabolism of young rats subjected to a deficiency of calories or of protein. PhD Thesis, University of Cambridge.
McCracken, K. J. (1975). *British Journal of Nutrition* **33**, 277–289.
McCracken, K. J. & Barr, H. G. (1985). *Proceedings of the Nutrition Society* **44**, 106A.
McCracken, K. J. & McAllister, A. (1984). *British Journal of Nutrition* **51**, 225–234.
McCracken, K. J. & McNiven, M. (1983). *British Journal of Nutrition* **49**, 193–202.
McMeekan, C. P. (1940*a*). *Journal of Agricultural Science, Cambridge* **30**, 276–344.
McMeekan, C. P. (1940*b*). *Journal of Agricultural Science, Cambridge* **30**, 387–438.
McMeekan, C. P. (1940*c*). *Journal of Agricultural Science, Cambridge* **30**, 511–569.
McNiven, M. (1980). The effect of body fatness on the maintenance energy requirements of adult rats. PhD Thesis, Swedish University of Agricultural Sciences, Uppsala.
Mickelsen, O., Takahaski, S. & Craig, C. (1955). *Journal of Nutrition* **57**, 541–554.
Miller, D. S. & Payne, P. R. (1962). *Journal of Nutrition* **78**, 255–262.
Moore, B. J. & Brasel, J. O. (1984). *Journal of Nutrition* **114**, 1560–1565.
Muto, S. & Miyahara, C. (1972). *British Journal of Nutrition* **28**, 327–337.
Obst, B. E., Schemmel, R. A., Czajka-Narins, D. & Merkel, R. (1981). *American Journal of Physiology* **240**, E47–E53.
Ozelci, A., Romsos, D. R. & Leveille, G. A. (1977). *Journal of Nutrition* **107**, 1768–1774.
Palsson, H. (1955). In *Progress in the Physiology of Farm Animals*, pp. 430–452 [J. Hammond, editor]. London: Butterworths.
Palsson, H. & Verges, J. B. (1952). *Journal of Agricultural Science, Cambridge* **42**, 93–149.
Panaretto, B. A. (1964). *Australian Journal of Agricultural Research* **15**, 771–787.
Pearce, J. (1983). *Proceedings of the Nutrition Society* **42**, 263–272.
Perkins, E. G., Endres, J. G. & Kummerow, F. A. (1961). *Journal of Nutrition* **73**, 291–298.
Robinson, J. J. (1986). *Proceedings of the Nutrition Society* **45**, 71–80.
Rucklidge, G. J. (1982). *British Journal of Nutrition* **48**, 341–351.
Schemmel, R., Mickelsen, O. & Mostosky, U. (1970). *Anatomical Record* **106**, 437–446.
Schemmel, R., Mickelsen, O. & Tolgay, Z. (1969). *American Journal of Physiology* **216**, 373–379.
Schoenborne, B. M. & Canolty, N. L. (1980). *Proceedings of the Society for Experimental Biology and Medicine* **163**, 10–14.
Sclafani, A. & Springer, P. (1976). *Physiology and Behaviour* **17**, 461–471.
Searle, T. W., Graham, N. McC. & O'Callaghan, M. (1972). *Journal of Agricultural Science, Cambridge* **79**, 371–382.
Spray, C. M. (1950). *British Journal of Nutrition* **4**, 354–360.

Spray, C. M. & Widdowson, E. M. (1950). *British Journal of Nutrition* **4**, 332–353.
Turkenkopf, I. J., Maggio, C. A. & Greenwood, M. R. C. (1982). *Journal of Nutrition* **112**, 1254–1263.
Vander Tuig, J. G., Romsos, D. R. & Leveille, G. A. (1980). *International Journal of Obesity* **4**, 79–85.
Wahle, K. W. J. & Radcliffe, J. D. (1977). *Lipids* **12**, 135–139.
Walks, M., Lavau, M., Presta, E., Yang, M.-U. & Bjorntorp, P. (1983). *American Journal of Clinical Nutrition* **37**, 387–395.
Webster, A. J. F. (1986). *Proceedings of the Nutrition Society* **45**, 1–9.
Weekes, T. E. C. (1983). *Proceedings of the Nutrition Society* **42**, 129–136.
Widdowson, E. M. (1968). In *Growth and Development of Mammals*, pp. 224–233 [G. A. Lodge and G. E. Lamming, editors]. London: Butterworths.
Wilson, P. (1954). *Journal of Agricultural Science, Cambridge* **44**, 67–85.
Wilson, P. N. (1960). *Journal of Agricultural Science, Cambridge* **54**, 105–130.

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