Severe undernutrition in growing and adult animals

17.* The ultimate results of rehabilitation: Pigs†

By D. LISTER‡ and R. A. McCANCE

Medical Research Council Department of Experimental Medicine, University of Cambridge

(*Received* 13 *October* 1966—*Accepted* 22 *May* 1967)

1. Ten pigs which had been undernourished for a year, and weighed about 5·5 kg at that time, were rehabilitated on an excellent diet. They grew fast, but no faster per kg body-weight than normal pigs, and they stopped growing at the same chronological age. Consequently, they did not attain the same adult size as the normally reared pigs.

2. The rehabilitated animals appeared to be fatter than the control animals, but the proportions of muscle to bone in the carcasses of the males and females were within the range to be expected in normal animals of the same fat-free weight, although the castrates may have been somewhat less muscular on this basis.

3. The weights of the organs per kg of fat-free body weight were within the limits found in normally reared animals of the same fat-free weight.

Interest in the ability of animals to resume growth after periods of starvation or undernutrition has centred mainly on two issues; first, is the composition of the body which may have become both chemically and anatomically abnormal during undernutrition (Widdowson, Dickerson & McCance, 1960; Dickerson & McCance, 1964) once more restored to normal; and secondly, do the animals reach normal adult size? Studies of growth have shown that there are differences in response which depend upon the species of animal, and the severity, duration and time of onset of the period of undernutrition (Wilson & Osborn, 1960; McCance & Widdowson, 1962). Hammond and his co-workers in Cambridge studied the anatomical development of pigs and sheep during and after dietary growth retardation (McMeekan, 1940*a–c*, 1941; Wallace, 1948*a–c*; Pálsson & Verges, 1952), and concluded that, by altering the rate at which animals grow, it was possible to modify the proportions of the tissues and organs in the growing animal and even in the adult after rehabilitation (McMeekan & Hammond, 1940). Since the early work, however, suggestions have been made that some of the differences in the proportions of muscle and bone in animals reared under different dietary treatments were really those appropriate for the particular live weight at which they were killed, taking into account the different amounts of fat in the carcasses (Wilson, 1954*a*, *b*; Tulloh, 1963; Elsley, McDonald & Fowler, 1964). There is also a limited amount of experimental evidence which supports this conclusion (Luitingh, 1962; Butterfield, 1963), but only rarely have animals been kept under observation for a sufficiently long time to be sure that growth has ceased. The purpose

* Paper no. 16: *Br. J. Nutr.* (1966), **20**, 633.

† Reprint requests to 5 Shaftesbury Road, Cambridge.

‡ Present address: A.R.C. Meat Research Institute, Langford, Nr Bristol.

of the present investigation was to assess the performance of pigs which had suffered extreme restrictions of food intake and hence of growth for 1 year, when they were allowed free access to a good diet.

MATERIALS AND METHODS

Ten undernourished pigs, four castrates, three sows and three boars, weighing 5–6 kg when they were 1 year old were rehabilitated to maturity; temporary effects of rehabilitation have been noted in others slaughtered before that time (Mount, Lister & McCance, 1963; Dickerson & McCance, 1964). Animals from the healthy colony of 'Large White' stock provided the controls. They were reared and looked after as described by McCance (1960) and Čabak, Gresham & McCance (1962), but a short summary of the procedure will be given here.

The animals to be undernourished were weaned from the sow at about 10–14 days of age on to a commercial early weaning diet (Amvilac no. 2; Glaxo Research Ltd, Greenford, Middx.). After a few weeks, they were transferred to a diet containing 50% Amvilac no. 2 and 50% of meal mixture containing wheat offals, barley meal, maize meal and fish meal. The final mixture contained about 19% protein, 6% fat and 60% carbohydrate. Each undernourished pig received about 90 g of the mixture per day divided equally between three meals. The amount of this food was increased slightly through the year of undernutrition to allow a little growth to continue. Even so, at the end of the year the pigs weighed only about 5 kg. Each undernourished animal was weighed weekly.

At the end of the year the amount of food was increased so that after 1–2 weeks of rehabilitation the pigs were allowed free access to the same diet with which they were undernourished. A little later, the Amvilac no. 2 was taken from the mixture. During the early period of rehabilitation the pigs were weighed weekly. Later they were weighed monthly and finally every 2–3 months.

The well-nourished controls were reared in the conventional manner; i.e. young pigs which had been allowed access to 'creep' feed (Amvilac no. 2) from 3 weeks of age were weaned from the sow at 6–8 weeks. They were then fed according to body-weight on the meal mixture described above, receiving a maximum of 4 lb/day as adults. In later life the controls were fed the same quantities of a commercial cubed diet (Spillers Ltd) which had roughly the same composition as the meal mixture.

A similar weighing procedure was adopted for the control pigs as for the rehabilitating pigs, more frequent weighings being done when the growth rates were particularly rapid.

All the pigs that have been undernourished since 1961 have also had precautionary supplements of 3 mg riboflavine, 25 mg nicotinic acid and a tablet of Aneurine Co (Roche Products Ltd, Welwyn Garden City, Herts.) per pig/week but this has not been stated before.

When there had been no significant increase in live weight for several months, the rehabilitated pigs were slaughtered, bled and eviscerated in the commercial manner. This was also the slaughter policy adopted for the control pigs. The carcasses of the rehabilitated and control pigs were measured and seven rehabilitated and three controls were dissected into their constituent fat, muscle, bone and 'remainder' (mainly skin) by the methods described by Cuthbertson & Pomeroy (1962). Measurements were made of the long bones as illustrated in Fig. 1 and X-ray photographs were taken of the same bones. The eye muscle (longissimus dorsi) was measured as described by the National Pig Progeny Testing Board (1959), and its cross-sectional area was estimated with a planimeter. The amounts of water, fat and nitrogen in selected muscles were determined by the methods described by Dickerson & Widdowson (1960). The numbers of animals used in each experiment are given with the results.

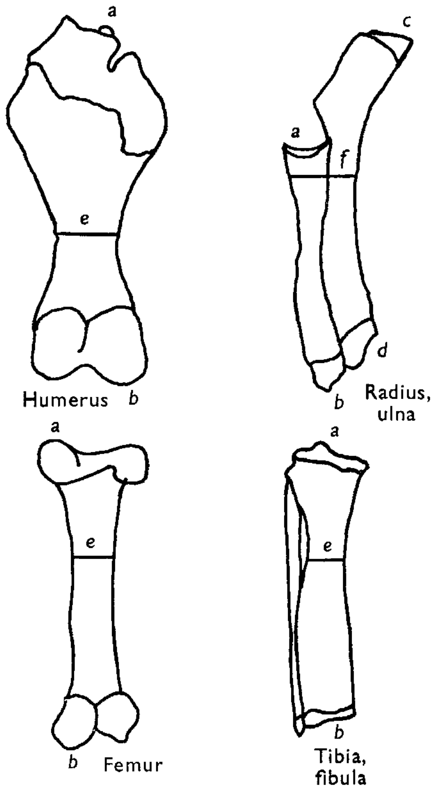


Fig. 1. Diagrams of the long bones of the pigs to show how the measurements of their dimensions were made. *a–b*, *c–d*, the greatest lengths; *e*, the narrowest widths; *f*, the thickness of radius + ulna.

RESULTS

At the end of 1 year's undernutrition, the experimental pigs had the appearance shown in Pl. 1. Such pigs have poor muscular tone and seldom stand on their toes as normal animals do. This may become exaggerated for a time during rehabilitation, and even 'bandy legs' have temporarily been observed in such animals (McCance, 1964), although usually the pigs quickly take on a relatively normal appearance. The leg weakness may be a variant of that seen in rehabilitated poultry (Lister, Cowen &

McCance, 1966), and may be a lesion similar to that which causes so much concern in rapidly growing boars. One animal which developed a permanent limp was found at slaughter to have a distorted femur (Pl. 2*a*) which may have been an extreme result of the temporary strains suffered by the limbs of animals after 9–19 weeks rehabilitation (Pratt & McCance, 1964).

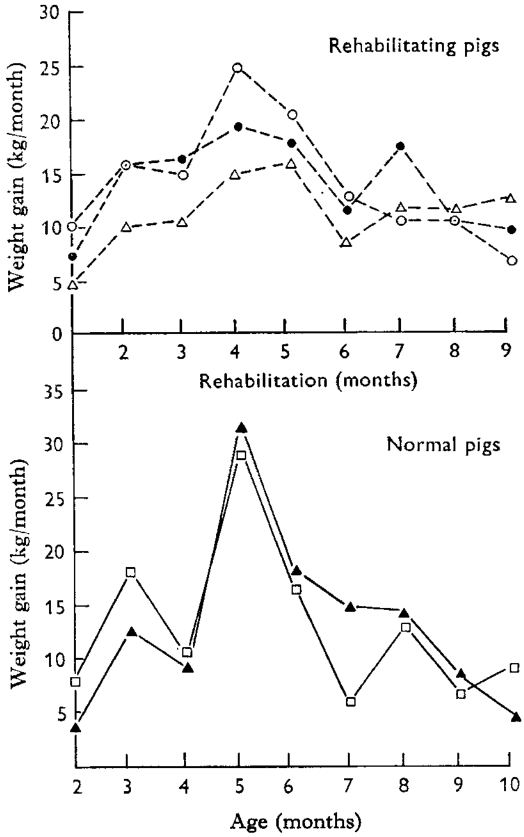


Fig. 2. The mean rates of gain of normal and rehabilitating pigs from approximately the same starting weights. □, nine normal females; ▲, five normal castrates; ○, three rehabilitating females; ●, three rehabilitating males; △, four rehabilitating castrates.

Growth of rehabilitated and control pigs to their mature size

Fig. 2 shows the rates of gain in weight (in kg/month calculated as the gain between successive months) of fourteen control and ten rehabilitating experimental pigs. The records begin from the time when the different groups of animals were of similar body-weight and this explains why the curves for the rehabilitated animals cover the period from the 1st to the 9th month of rehabilitation (13–21 months of age) and the curves for the normal pigs cover the period from 2 to 10 months of age. The maximum rate of gain was attained by the normal pigs at about 5 months of age and was greater for castrated males than for gilts. The gains for the rehabilitated animals resembled those for their controls, but their maximum rates were not so great, and the gilts put on weight more rapidly than the males and castrates. In most of the animals the fastest rates of gain were registered a similar length of time after the body-weights had been the same. The peak for the rehabilitated castrates was, however, only 16 kg/month and came slightly later. At the end of 9 months of rehabilitation or 10 months of age (normal animals) the rates of gain were very much the same for all the animals. The low rate of gain shown by the control animals at 4 months cannot be explained in full but may have been associated with a temporary increase in the incidence of intestinal worms in these animals.

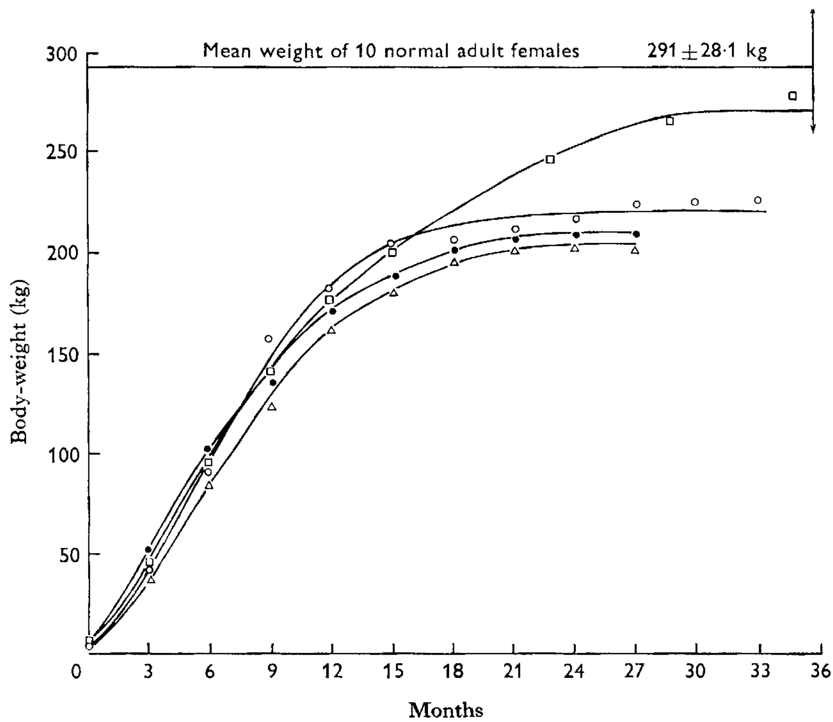


Fig. 3. Increase in weight of pigs rehabilitated after 1 year of undernutrition and of normal pigs being reared from the same initial weight. □, nine normal females; ○, three rehabilitating females; ●, three rehabilitating males; △, four rehabilitating castrates.

Fig. 3 shows the averaged growth curves of nine normal sows and the mean weight at maturity of ten others. The animals used for the mean weight at maturity were either the mothers or grandmothers of the undernourished pigs which were to be rehabilitated. Fig. 3 also shows the averaged growth curves of four rehabilitated castrates, three sows and three boars. None of the rehabilitated animals attained the weight or stature of the normal ones, and this confirms the findings of Widdowson & McCance (1960) on rats, Lister & McCance (1965) on guinea-pigs and Lister *et al.* (1966) on poultry in showing that undernutrition at the appropriate age and for a sufficiently long time may prevent such animals from attaining their genetically ordained stature and weight, however well they may be rehabilitated. The curves also show that when the pigs were rehabilitated they did not grow for the same length of time as the normal ones. Growth in fact ceased after about 21–24 months, so that the pigs that had been undernourished ceased to grow at about the same, or a slightly younger, chronological age than the normal pigs—whatever the stature they had attained at that time. This is a result similar to that obtained by Widdowson & McCance (1963) on rats and by Lister & McCance (1965) on guinea-pigs. The pigs, moreover, stopped growing before their epiphyses had been joined to the shafts as judged by dissection and X-ray appearance after death (Pl. 2*a*). Pl. 3 shows the appearance of a fully rehabilitated animal and of one conventionally reared.

Table 1. *Mean body-weight (kg) and organ weights (g) of rehabilitated and control pigs*

(Figures in parentheses refer to dissected animals and express

$$\frac{\text{the weight of the organ}}{\text{the fat-free body weight}} \times 100)$$

	Rehabilitated						Control sows	
	Sows		Boars		Castrates			
No. of animals	3	(2)	3	(3)	4	(2)	3	(3)
Body-weight	220		216		213		270	
Weight of: Lungs and trachea	1673	(1·28)	1257	(0·90)	1614	(1·38)	2732	(1·53)
Heart	589	(0·43)	620	(0·45)	576	(0·50)	897	(0·50)
Liver	2783	(2·26)	2576	(1·85)	1958	(1·67)	3381	(1·89)
Spleen	227	(0·17)	281	(0·20)	249	(0·21)	480	(0·27)
Kidneys	541	(0·40)	594	(0·43)	315	(0·27)	664	(0·37)
Adrenals	—		10·9	(0·01)	9·2	(0·01)	17·8*	(0·01)
Stomach	1215	(0·73)	1433	(1·03)	1244	(1·06)	1596	(0·90)
Small intestine	1607	(1·32)	1773	(1·28)	2238	(1·91)	2155	(1·20)
Large intestine	5398	(5·50)	2470	(1·78)	4831	(4·12)	6173	(3·45)
Brain	113·5	(0·07)	136·0	(0·10)	120·7	(0·10)	143·6	(0·08)
Spinal cord	64·4	(0·05)	64·0	(0·05)	57·8	(0·05)	80·0	(0·04)

* Only one pair weighed.

The weights of the internal organs and the body-weights of rehabilitated female, male and castrate pigs, and those of female control animals are given in Table 1. The absolute weights of the organs of the rehabilitated animals, except for the weight of the small intestines of the castrates, were smaller than those of the control animals, but when the organ weights were expressed per unit of fat-free body-weight, there were few differences. There was possibly some evidence that the relatively small spleens, which were so typical a feature of the undernourished pigs (McCance, 1960), remained small after rehabilitation. The kidneys of the castrates were also small—a finding recorded in pigs by McMeekan (1941) although he made no comment on it. This result has been observed previously in other species (Korenchevsky & Dennison, 1934; Kochakian, 1944; Dorfman & Shipley, 1956) and has been attributed to the fall in androgen production following castration (Leathem, 1964).

The weights of the brains and spinal cords tended to be appropriate for the body size in the rehabilitated and control animals. The chemical composition of these organs has been investigated (Dickerson, Dobbing & McCance, 1967).

Tables 2–4 give detailed information about the carcass dimensions of the rehabilitated and control pigs. The information in Tables 2 and 3 shows that the skeletons of the rehabilitated animals were always smaller than those of the controls (compare also

Table 2. *Carcass measurements (mm) of rehabilitated and control pigs*

(Mean values with standard deviations)

	Rehabilitated			Control	
	Sows	Boars	Castrates	Sows	Boars
No. of animals	3	3	4	3	2 (range)
Length of side	989 ± 28·7	1014 ± 34·3	970 ± 21·6	1159 ± 23·6	1105 (1028–1182)
Length of hind-leg	660 ± 37·0	677 ± 41·0	714 ± 16·5	752 ± 19·8	853 (832–874)
Length of loin	469 ± 6·5	454 ± 9·3	453 ± 4·1	537 ± 7·4	646 (609–683)
Depth of side	487 ± 3·9	487 ± 8·7	513 ± 5·8	564 ± 8·2	568 (547–589)
Back fat: shoulder	63 ± 10·4	57 ± 8·4	75 ± 11·1	71 ± 3·1	45 (41–49)
mid back	35 ± 4·6	36 ± 3·9	49 ± 5·2	40 ± 2·5	25 (22–28)
loin	36 ± 5·3	45 ± 4·8	49 ± 5·2	48 ± 2·5	23 (22–24)

Table 3. *Dimensions (cm) of the long bones of rehabilitated and control pigs*

(Mean values with standard deviations)

	Rehabilitated			Control	
	Sows	Boars	Castrates	Sows	Boars*
No. of animals	3	3	4	3	2
Femur: length	24·0 ± 0·44	25·8 ± 1·27	25·8 ± 0·57	28·1 ± 0·29	30·7 (30·0–31·4)
width	3·3 ± 0·30	2·9 ± 0·52	3·1 ± 0·14	3·5 ± 0·10	4·3 (4·2–4·4)
Tibia fibula: length	21·5 ± 0·85	23·7 ± 1·43	23·0 ± 0·75	25·7 ± 0·33	28·7 (28·1–29·3)
width	3·0 ± 0·10	3·0 ± 0·21	2·8 ± 0·10	3·3 ± 0·10	3·7 (3·5–3·9)
Humerus: length	21·9 ± 1·05	23·7 ± 0·93	23·1 ± 0·92	24·7 ± 0·49	27·3 (26·7–27·9)
width	3·9 ± 0·20	3·8 ± 0·12	3·6 ± 0·11	4·7 ± 0·13	4·7 (4·6–4·8)
Radius length	16·4 ± 1·70	17·3 ± 1·28	16·5 ± 0·97	19·3 ± 0·62	21·6 (21·2–22·0)
Ulna length	22·1 ± 2·20	23·9 ± 1·97	23·0 ± 1·43	25·2 ± 0·85	27·2 (26·6–27·8)
Radius + ulna thickness	4·5 ± 0·52	4·7 ± 0·44	4·6 ± 0·38	5·4 ± 0·23	6·5 (6·5–6·5)

* Mean values and ranges.

Table 4. *Measurements of the longissimus dorsi of rehabilitated and control pigs made according to the National Pig Progeny Testing Board* (1959)

(Mean values and standard deviations)

	Rehabilitated			Control
	Sows	Boars	Castrates	sows
No. of animals	3	3	4	3
Measurement:				
A (mm)	99·3 ± 6·43	109·0 ± 9·63	91·9 ± 8·43	116·7 ± 5·05
B (mm)	50·6 ± 7·29	55·0 ± 6·85	47·5 ± 9·00	54·1 ± 6·44
C (mm)	45·6 ± 5·02	45·0 ± 4·97	57·2 ± 5·74	36·7 ± 5·87
J (mm)	15·0 ± 1·91	8·2 ± 1·43	15·0 ± 2·17	10·5 ± 6·56
K (mm)	50·0 ± 1·78	49·3 ± 2·09	64·0 ± 2·59	49·7 ± 1·58
Area (cm^2)	37·4 ± 4·87	38·1 ± 4·34	28·75 ± 5·78	42·7 ± 4·05

Pl. 2*b* with 2*c*). They also show that the amount of recovery varied from limb to limb and with the sex of the animals; the females always showed a better recovery than either the males or castrates which, on the whole, were very similar. Table 4 indicates the dimensions of the longissimus dorsi muscles. The rehabilitated animals had similar thicknesses of subcutaneous fat which were not very different from those found in the controls, and this seems to indicate that the smaller, rehabilitated, pigs contained proportionally more fat than the controls. A general appraisal of the carcasses of the rehabilitated pigs (with the possible exception of the boars) therefore suggests that they were relatively fatter, had smaller skeletons and were less muscular than the normally reared stock; Table 5 shows that this was confirmed by the dissections.

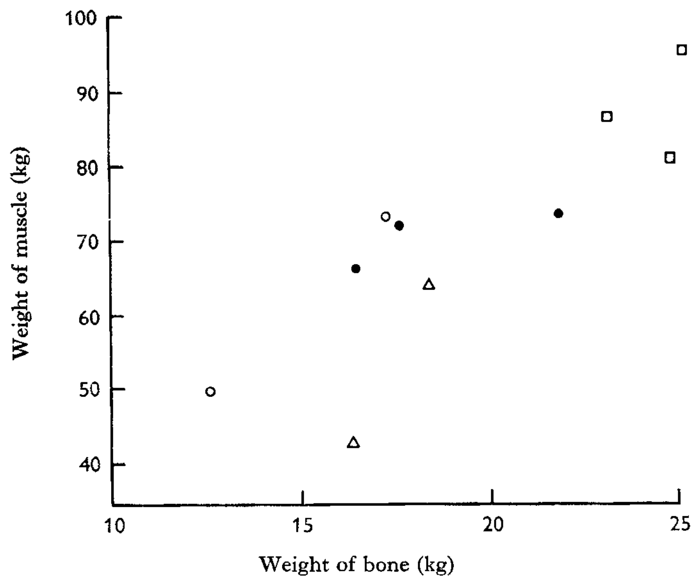


Fig. 4. Relationship of muscle to bone in (*a*) three normal sows, (*b*) seven rehabilitated animals. Symbols as in Figs 2 and 3.

However, when the amounts of muscle and bone in the carcasses were compared, they were found to be appropriate to each other when the weight of fat was excluded. This is illustrated in Fig. 4 where the weight of muscle in the rehabilitated and the control pigs has been plotted against the weight of bone in the corresponding carcass. The carcasses of the castrates may have contained slightly less muscle even than those of the males and females of the same weight (see also Table 4).

Table 6 shows the mean percentage of water, fat and nitrogen in four of the muscles of the normal and rehabilitated pigs. Just as there was relatively more subcutaneous and intermuscular fat (recorded together as total fat in the tables) in the rehabilitated carcasses, so too was there more intramuscular fat and the amounts varied from muscle to muscle. There was little or no difference in the amounts of water in the muscles of the two groups, but the concentration of nitrogen in the muscles of the rehabilitated animals tended to be lower than in those of the control group. In view of the small

Table 5. *Composition of the carcasses of rehabilitated and control pigs*

(Figures in parentheses express $\frac{\text{weight of the tissue}}{\text{total carcass weight}} \times 100$)

Component	Rehabilitated: Sows		Castrates		Boars			Control sows		
Muscle (kg)	72·6 (35·5)	49·7 (38·6)	43·1 (25·8)	63·8 (31·8)	71·8 (40·0)	73·1 (43·6)	66·4 (44·3)	87·0 (42·0)	80·1 (36·6)	95·0 (39·5)
Bone (kg)	17·4 (8·5)	12·7 (8·4)	16·4 (9·8)	18·4 (9·2)	17·6 (9·8)	21·9 (13·0)	16·4 (10·9)	23·1 (11·1)	24·9 (11·4)	25·1 (10·4)
Fat (kg)	98·5 (48·1)	57·8 (45·0)	91·9 (55·0)	101·1 (50·4)	77·4 (43·0)	61·6 (36·8)	57·6 (38·4)	84·3 (40·5)	89·6 (40·9)	97·8 (40·5)
Remainder (kg)	16·0 (7·8)	8·8 (6·8)	15·8 (9·4)	17·6 (8·8)	12·9 (7·2)	11·4 (6·8)	9·6 (6·4)	14·4 (6·9)	24·8 (11·3)	22·5 (9·3)
Total carcass weight (kg)	204·5	129·0	167·2	200·9	179·7	168·0	15·0	208·8	219·4	240·4

Table 6. *Composition of muscles from five rehabilitated (two males, two females and one castrate) and three control pigs*

(Figures expressed as g/100 g fat-free tissue; range of values in parentheses)

Muscle	Rehabilitated: Water	Fat	Nitrogen	Control: Water	Fat	Nitrogen
Longissimus dorsi	75·6 (75·0–76·0)	13·37 (5·60–28·86)	3·63 (3·39–3·84)	74·9 (74·6–75·2)	3·77 (3·08–4·61)	3·88 (3·85–3·91)
Longissimus arricus	75·3 (73·9–76·0)	6·83 (3·20–14·41)	3·64 (3·52–3·74)	75·3 (75·0–76·6)	2·44 (1·93–2·92)	3·81 (3·75–3·87)
Psoas major	76·7 (76·0–77·8)	4·68 (2·04–6·72)	3·37 (3·21–3·49)	76·9 (76·6–77·0)	2·79 (2·08–3·14)	3·49 (3·44–3·54)
Biceps femoris	76·6 (76·1–77·1)	6·44 (4·32–9·46)	3·41 (3·16–3·65)	76·6 (76·3–76·8)	3·82 (3·25–4·43)	3·58 (3·53–3·62)

number of both sexes of animals examined, it was not possible to evaluate this statistically. This unexpected finding (Dickerson & McCance, 1964) is being further investigated.

Other observations

The rehabilitated boars were highly potent and the rehabilitated females which were allowed to mate produced satisfactory litters which grew as well as those born to conventionally reared stock (Lister, 1965). This finding supports and extends those of Asdell & Crowell (1935) and Ball, Barnes & Visscher (1947) that after delayed puberty reproductive capacity was not impaired.

Post-mortem examinations of the rehabilitated pigs showed that they had no pathogenic disease and were in good health at the time of slaughter. The failure of one female to breed was explained by an imperforate cervix.

DISCUSSION

There is no doubt that, if animals are subjected to a period of undernutrition severe enough and early enough in their development, they do not attain the full size ordained for them by nature—even if they are given unlimited amounts of good food thereafter. This failure to make good is not necessarily due to closure of the epiphyses, and Hrůza & Fábry (1957) have shown that rats can recover and regain their full possible size if they are given extraneous growth hormone. Similar experiments have not yet been carried out on other animals, but it is reasonable to suggest that growth ceases in all at an age when the bones for some reason cease to respond to the amount of growth hormone in the circulation.

Under the experimental conditions employed, Widdowson & McCance (1963) found that rats stopped growing at a fixed chronological age whatever size they had reached at that time and whether they had had a period of undernutrition or not. Lister & McCance (1965) found that guinea-pigs did the same whether they were perfectly nourished throughout their lives, poorly nourished throughout or poorly nourished till weaning and then rehabilitated. The present experiments indicate that pigs behave in the same way, but cockerels do not (Lister *et al.* 1966). The latter grow well at an age when their brood mates have already reached their full genetic stature and ceased to grow, and yet, in spite of this, they do not achieve the same stature. McCay, Crowell & Maynard (1935) found that some increase in weight was still possible in rats after a normal life span of severe undernutrition. If this was true growth in bone size there may not be such a clear line of distinction between rats, guinea-pigs and pigs on the one hand and poultry on the other as the recent experiments suggest; this can only be settled by further work, and the results are not likely to alter the main facts recently brought to light.

The growth of man is slow compared with that of all the usual experimental animals, but it is governed just as surely by the plane of nutrition. The evidence that a continuously low plane of nutrition reduces the rate of growth is convincing and requires no discussion, and there is now good evidence that it leads to smaller adults (Coon, 1939; Leitch & Boyne, 1960; Oppers, 1963), but man's growth is not so strictly limited chronologically as that of rats, guinea-pigs and pigs. He can attain the full stature of which he is genetically capable by 18 years or earlier if his plane of nutrition has been continuously high, but if he has not done so he can go on growing for a number of years and possibly attain it by the time he is 23–25 (Oppers, 1963). The evidence from rural communities which are not well fed confirms this (King, Foucauld, Fougere & Severinghaus, 1963; Bäckström-Järvinen, 1964) and so do the records of army recruits (Morant, 1950). Man's ability to go on growing after the age at which it is possible for him to attain his full stature explains why the increase in man's final stature over the last few decades (Boyne & Leitch, 1954) is not nearly so great as is the increase in height at 18 years. In his ability to grow after the age at which he can attain his full genetic stature if well fed, man has a growth pattern which resembles that of poultry to some extent, but he is probably not peculiar among the mammals in this, for cattle (Crichton, Aitken & Boyne, 1960) and sheep (Allden, 1961) have been reported to behave in the same way.

An animal whose growth has been delayed by undernutrition may respond to a period of adequate nutrition by growing at a rate in excess of that to be expected in animals of a similar age, so-called compensatory growth (Bohman, 1955). In this study, the rates of growth of the pigs on rehabilitation were in excess of those to be expected in pigs 1 year old, but they were less than the growth rates of normal animals of the same size. This result is somewhat similar to that of Widdowson & Kennedy (1962) who found that rats were unable to reach normal adult size after they had been undernourished for the 3 weeks from birth until weaning, and they explained this in terms of the specific growth rate (Medawar, 1945). After weaning, when all the rats were allowed access to unlimited food, the animals which had been undernourished from birth multiplied their weight rather more quickly than the rats which had grown optimally from birth, but in spite of this, they were unable to reduce their deficit in absolute weight, because their rate of growth declined with time. Furthermore, after about 3 months of rehabilitation, their curves of incremental growth were indistinguishable from those of normal animals of the same age. As with the rats, the rehabilitated pigs did not reach the normal adult size because their rate of growth was insufficient to compensate for the shorter time during which growth could take place.

The metabolic allocation of the food consumed between growth and the deposition of fat unquestionably varies with age and this may well explain why the rehabilitated animals tended to be fat, for they were 1 year older than the normal animals when they were growing over the same weight range. The permanent stunting which McMeekan & Hammond (1940) predicted after undernutrition in pigs has been demonstrated, but, apart from abnormalities of the teeth (McCance, Ford & Brown, 1961; Owens, Tonge & McCance, 1965) and possibly of the skeletal muscles, the form and composition of the bodies were appropriate for their size. The latter might not have been expected in view of the findings of McMeekan (1940*a*–*c*) and Pálsson & Verges (1952) on the order of development of various bodily tissues. It is known, however, that under particularly severe food restriction, the proportions of muscle and bone may be altered (Dickerson & McCance, 1964). If differences persist even when allowances are made for the varying amounts of fat in the bodies (Wilson, 1954*a*, *b*;

Luitingh, 1962; Butterfield, 1963), it seems unlikely that the order of development of the various parts and tissues is responsible (Elsley *et al.* 1964).

There are therefore several questions to be answered before one can assess accurately the future of an undernourished animal: (1) Does it belong to a species which can grow only till it is a certain age and which will not attain its full size and stature unless it has already done so by that time? (2) Does it belong to a species in which considerable growth is possible after the age at which growth normally ceases? If so, for how long is such growth possible and why does it stop? (3) Would the administration of growth hormone facilitate the attainment of the animal's full genetic capabilities?

We are grateful to Drs M. Ingram (the Director) and R. W. Pomeroy for providing generous dissection facilities at the ARC Meat Research Institute's laboratories. We should also like to thank Dr D. A. T. Southgate for checking the chemical analyses reported in Table 6 and Dr E. M. Widdowson for helpful discussion at all stages of the work. The X-ray work was carried out in the Department of Anatomy, University of Cambridge, by Mr J. A. F. Fozzard, whom we also thank. The animals were fed, reared and cared for from birth by Terry Cowen.

EXPLANATION OF PLATES

PLATE 1

An undernourished pig 1 year old (left) and a normal animal of the same weight (right).

PLATE 2

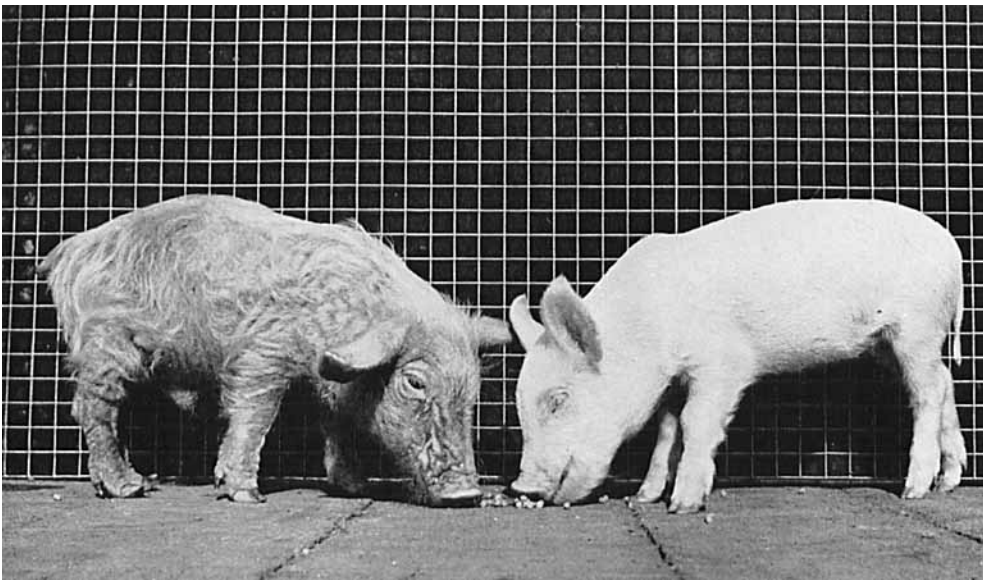
X-ray photographs to the same scale of (*a*) distorted femur of fully rehabilitated sow, (*b*) femur of fully rehabilitated sow, and (*c*) femur of mature normal sow.

PLATE 3

A fully grown normal sow (above) and rehabilitated sow (below).

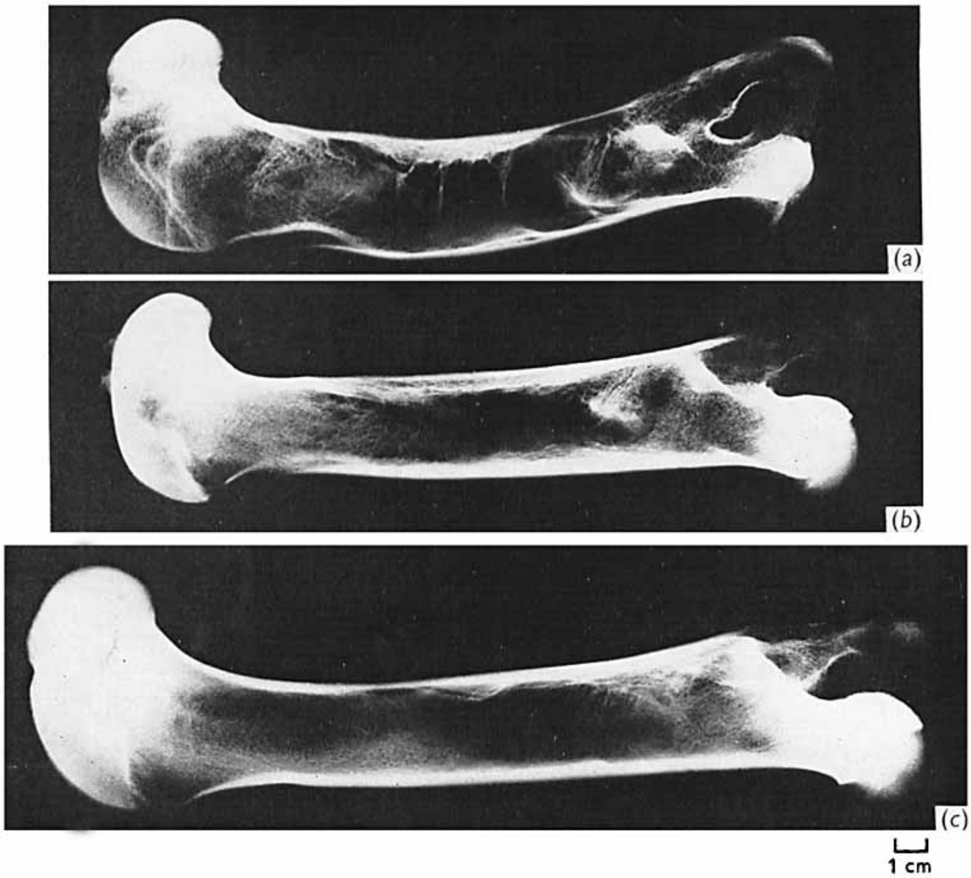
REFERENCES

Allden, W. G. (1961). *Wool Technol. Sheep Breed.* **8**, no. 2, p. 81.
Asdell, S. A. & Crowell, M. F. (1935). *J. Nutr.* **10**, 13.
Bäckström-Järvinen, L. (1964). *Annls Paediat. Fenn.* **10**, Suppl. 23.
Ball, Z. B., Barnes, R. H. & Visscher, M. B. (1947). *Am. J. Physiol.* **150**, 511.
Bohman, V. R. (1955). *J. Anim. Sci.* **14**, 249.
Boyne, A. W. & Leitch, I. (1954). *Nutr. Abstr. Rev.* **24**, 255.
Butterfield, R. M. (1963). In *Symposium: Carcass Composition and Appraisal of Meat Animals*, paper 4–1. [D. E. Tribe, editor.] Australia: CSIRO.
Čabak, V., Gresham, G. A. & McCance, R. A. (1962). *Br. J. Nutr.* **16**, 635.
Coon, C. S. (1939). *The Races of Europe*, pp. 10, 323. New York: McMillan.
Crichton, J. A., Aitken, J. N. & Boyne, A. W. (1960). *Anim. Prod.* **2**, 45.
Cuthbertson, A. & Pomeroy, R. W. (1962). *J. agric. Sci., Camb.* **59**, 207.
Dickerson, J. W. T., Dobbing, J. & McCance, R. A. (1967). *Proc. R. Soc.* B **166**, 396.
Dickerson, J. W. T. & McCance, R. A. (1964). *Clin. Sci.* **27**, 123.
Dickerson, J. W. T. & Widdowson, E. M. (1960). *Biochem. J.* **74**, 247.
Dorfman, R. I. & Shipley, R. A. (1956). *Androgens*. New York: John Wiley and Sons Inc.
Elsley, F. W. H., McDonald, I. & Fowler, V. R. (1964). *Anim. Prod.* **6**, 141.
Hrůza, Z. & Fábry, P. (1957). *Gerontologia* **1**, 279.
King, K. W., Foucald, J., Fougere, W. & Severinghaus, E. L. (1963). *Am. J. clin. Nutr.* **13**, 106.

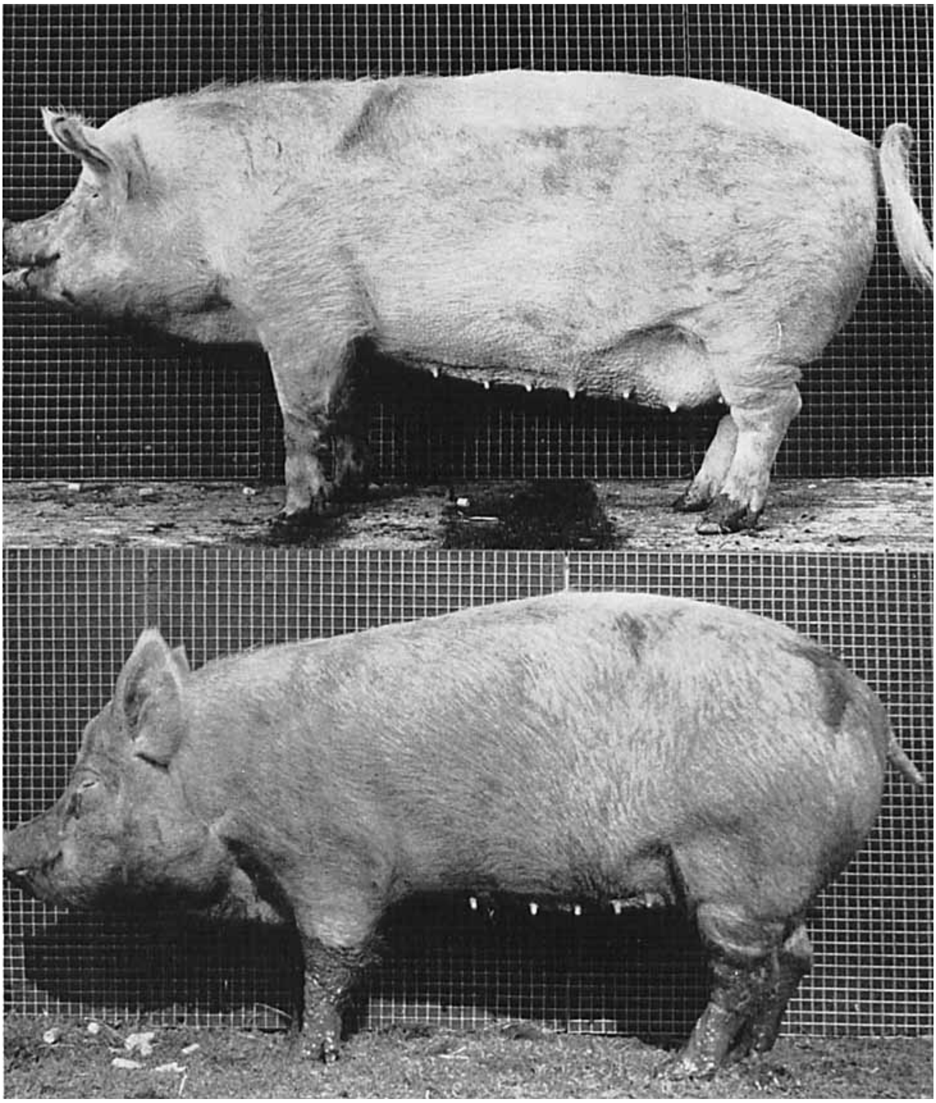


D. LISTER AND R. A. McCANCE

(Facing p. 798)



D. LISTER AND R. A. McCANCE



D. LISTER AND R. A. McCANCE

Kochakian, C. D. (1944). *Am. J. Physiol.* **142**, 315.
Korenchevsky, V. & Dennison, M. (1934). *J. Path. Bact.* **38**, 231.
Leathem, J. H. (1964). In *Mammalian Protein Metabolism*, p. 343. [H. N. Munro and J. B. Allison, editors.] New York and London: Academic Press Inc.
Leitch, I. & Boyne, A. W. (1960). *Nutr. Abstr. Rev.* **30**, 1173.
Lister, D. (1965). The effect of nutrition before and after birth on adult size and structure. PhD Thesis, University of Cambridge.
Lister, D., Cowen, T. & McCance, R. A. (1966). *Br. J. Nutr.* **20**, 633.
Lister, D. & McCance, R. A. (1965). *Br. J. Nutr.* **19**, 311.
Luitingh, H. C. (1962). *J. agric. Sci. Camb.* **58**, 1.
McCance, R. A. (1960). *Br. J. Nutr.* **14**, 59.
McCance, R. A. (1964). *J. Pediat.* **65**, 1008.
McCance, R. A., Ford, E. H. R. & Brown, W. A. B. (1961). *Br. J. Nutr.* **15**, 213.
McCance, R. A. & Widdowson, E. M. (1962). *Proc. R. Soc.* B **156**, 326.
McCay, C. M., Crowell, M. F. & Maynard, L. A. (1935). *J. Nutr.* **10**, 63.
McMeekan, C. P. (1940*a*). *J. agric. Sci., Camb.* **30**, 276.
McMeekan, C. P. (1940*b*). *J. agric. Sci., Camb.* **30**, 387.
McMeekan, C. P. (1940*c*). *J. agric. Sci., Camb.* **30**, 511.
McMeekan, C. P. (1941). *J. agric. Sci., Camb.* **31**, 17.
McMeekan, C. P. & Hammond, J. (1940). *Emp. J. exp. Agric.* **8**, 6.
Medawar, P. B. (1945). In *Essays on Growth and Form Presented to D'Arcy Wentworth Thomson.* [W. E. Le Gros Clark and P. B. Medawar, editors.] Oxford: Clarendon Press.
Morant, G. M. (1950). *Proc. R. Soc.* B **137**, 443.
Mount, L. E., Lister, D. & McCance, R. A. (1963). *Br. J. Nutr.* **17**, 407.
National Pig Progeny Testing Board (1959). *Rep. natn. Pig Progeny Test. Bd* **2**, 17.
Oppers, V. M. (1963). Analyse van de acceleratie van de menselijke lengtegroei door bepaling van het tijdstip van de groeifasen. Thesis, University of Amsterdam.
Owens, P. D. A., Tonge, C. H. & McCance, R. A. (1965). *J. dent. Res.* **44**, 1159.
Pálsson, H. & Verges, J. B. (1952). *J. agric. Sci., Camb.* **42**, 1.
Pratt, C. W. M. & McCance, R. A. (1964). *Br. J. Nutr.* **18**, 613.
Tulloh, N. M. (1963). In *Symposium: Carcass Composition and Appraisal of Meat Animals*, paper 5–1. [D. E. Tribe, editor.] Australia: CSIRO.
Wallace, L. R. (1948*a*). *J. agric. Sci., Camb.* **38**, 93.
Wallace, L. R. (1948*b*). *J. agric. Sci., Camb.* **38**, 243.
Wallace, L. R. (1948*c*). *J. agric. Sci., Camb.* **38**, 367.
Widdowson, E. M., Dickerson, J. W. T. & McCance, R. A. (1960). *Br. J. Nutr.* **14**, 457.
Widdowson, E. M. & Kennedy, G. C. (1962). *Proc. R. Soc.* B **156**, 96.
Widdowson, E. M. & McCance, R. A. (1960). *Proc. R. Soc.* B **152**, 188.
Widdowson, E. M. & McCance, R. A. (1963). *Proc. R. Soc.* B **158**, 329.
Wilson, P. N. (1954*a*). *J. agric. Sci., Camb.* **44**, 67.
Wilson, P. N. (1954*b*). *J. agric. Sci., Camb.* **45**, 110.
Wilson, P. N. & Osborn, D. F. (1960). *Biol. Rev.* **35**, 324.

Printed in Great Britain