

Growth and health

Isabella Leitch

A general introduction to a discussion on growth and health might take either of two main forms. It might review what we know about the material requirements for growth: the total energy needs, the grams of protein required to give this or that amount of soft tissue growth, the grams of calcium and phosphorus required for growth of the skeleton; it might discuss the functional importance of substances that make little material contribution to growth, and finally, sum up what we know about the inter-relation of diet and resistance to infection. These are the subjects with the details of which most of the observational and experimental work in nutrition is, at the moment, concerned. It would take days to sum up the information; and, when that had been done, I doubt whether we should be able to do more than reaffirm general principles. These general principles are already well known. The concept of a balanced diet has become part of our 'common knowledge'.

Perhaps partly because I shirked that major task, and partly because I think it is good, now and then, to lay aside the microscope and look at things with the naked eye, I have chosen the second approach. In very general terms I am going to ask what we mean by good growth and health and what the one has to do with the other.

In experimental work with animals most often weight increase is taken as if it were synonymous with growth; less often growth is defined as increase in one or more linear dimensions. Obviously increase in weight may be increase in bone or muscle or fat or merely water or some combination of any or all of these; and some linear dimensions may be just as difficult to interpret. But suppose we had some complicated technique combining weights and measures and isotope studies and specific gravity of the body, what would it tell us about growth? It would be no more than a means to a crude chemical analysis without killing the subject, and, by itself, it would be just as interesting or as sterile as any other chemical analysis.

Before we can get up any real interest in the result we must know what changes in the structure and composition of the body mean in terms of some standard of value. What is value in growth? Why do we say this animal or person is well grown and that is not? We mean a great deal more, I think, than merely that the one is bigger or heavier than the other. The term well grown and the attitude of mind behind it imply some target of performance which we consider to have been approximated or missed.

Nature and nurture

In an attempt to clarify that idea, I have built up an argument in picture form. In the first place we must go back to the basis of growth, which depends on nature and nurture, but we should not require either complex genetical concepts or the refinements of chemistry to make the argument plain.

The fundamentals of genetics, or all of them that need concern us here, have been known for thousands of years; they are part of everyday language, everywhere. 'Like begets like'; 'like father, like son'; 'what is bred in the bone ...'; 'you cannot make a silk purse out of a sow's ear': these and similar clichés exist in all languages, sometimes in quaint forms, but with the same meaning. Even primitive man must have recognized the sometimes astonishing resemblance of child to parent, and even so-called primitive races of mankind have long practised systematic selection and breeding of their domestic stock. It is equally true that there has long been an awareness of merit in human reproduction. The ancient Greeks, and even earlier civilizations, had clear views on eugenics; and the same awareness, the same urge to 'good breeding' certainly persists in full force, and sometimes in strange forms, at the present day. I do not know whether, or to what extent, Plato's plans for human stud farms materialized, but modern and better informed people accept similar ideas.

We have no galaxy of clichés about nurture, which is not difficult to understand. It is still impossible to disentangle the effects of different components of the environment, especially in human populations where they can seldom be varied or controlled independently, and where cause and effect are so liable to be confused. For instance, tailors were, at one time, perhaps still are, accepted as of poor physique. But that is not because tailoring interferes with growth that is, in any case, already past, but because the relatively unfit gravitate to occupations that require relatively little physical strength. But better and worse diets were certainly distinguished and are, to some extent, embodied in racial beliefs of one kind and another. Perhaps the attribution of the peculiar virtues of the English to the eating of roast beef may enjoy a scientific rebirth with the ascent of the 'animal protein factor' to its present prominence in experimental work.

The effect of nurture on the concrete and visible expression of nature has, in the past, received too little attention at the hands of geneticists and plant and animal breeders. Let us review briefly what nurture can do to inhibit the expression of nature and what the significance of that inhibition may be.

In plants

Baur (1911) has described two dandelion plants, one grown high in the Alps on poor soil and in the cold, the other in the warm and fertile plain. If the dandelion were an important food plant, the verdict would be that the big, succulent plant is the better. But consider a different plant. Exactly the same occurs if edelweiss is transported to a warm and fertile garden. It loses its grey colour and white hairs; it stretches out and becomes green and leafy, the flower looks different; in fact an uninteresting plant. The Alpine is the plant that is prized. This little story has two morals. The first is that if one has seen a plant, or animal, only in one environment, all one knows about its genetic

constitution is what it can do in that environment and not what it may do or not do in any other. The second is that the terms good and bad, better and worse, as descriptions of plants or animals, are not absolutes, but are valid only in relation to environment, and that they imply some criterion of value which may be utilitarian or aesthetic or both. This is now, of course, well recognized in relation to crop production. For instance, a very large chapter in the history of Canadian agriculture is the story of the search for varieties of wheat that would survive the dry, cold climate of the prairies, that would grow and ripen in the short growing season, and that would resist indigenous diseases.

In animals

This is not yet so well realized in regard to animals, so let us look at what the wrong nurture can do to animal nature.

McMeekan's (1940*a-c*, 1941) papers on the growth of pigs show (Pl.1, fig.1) how a well-bred or 'improved' pig grows. One can see the change in shape as the animal grows from birth to the approved 200 lb bacon pig. In shape, the original wild pig at the fully grown stage is much more like the improved pig at birth than at maturity. The important thing is that the characteristics that are valued by the pig farmer, who is going to sell pork or bacon, are those which develop last. The most prized parts of the pig are the loin and hind-quarters. And the quality of the joints depends on the distribution of lean and fat. The same is, of course, true of beef and mutton.

A series of most important experiments, started in Dr Hammond's laboratory in Cambridge and continued in New Zealand, was made to test the effect of feeding on the development of the carcass of pigs and lambs. They showed in the most striking fashion that poor feeding not only stunts, but also delays indefinitely and, if continued long enough, permanently prevents, the full development of the later-developing and best parts of the carcass. If a bacon pig is underfed for half its short life and then given a full ration, this 'low-high' pig will certainly put on weight. But the important point is that skeleton and muscle will not grow as they would have done if they had had the opportunity at the right time, and the extra food will be used mainly to lay on fat (McMeekan, 1940*c*, Pl.27).

Growth potential

The moral of this is that growth potential is not a thing that one can speed up and slow down, as one can accelerate or retard a chemical process, and get the same result in the end. If one does not use the full potential all the way along one does not get full development. One may get the same ultimate weight but one will get a carcass that is different in shape and in composition. Now we have an idea of what is implied by a well-grown pig. If it is also fat, that should be 'finish' added when the correct framework of bone and muscle has grown to the desired size.

It may seem that it is a long step from bacon pigs and prime beef or mutton to man. Not so long a step perhaps.

An old diagram, drawn by Stratz and widely reproduced, e.g. by Brody (1945), of the change in shape that occurs in man during growth shows that, in essentials, the change is the same as in the pig. Development is from the cephalo-thoracic region forwards and backwards and the hind-quarters mature last. If the rate of growth is sufficiently slowed the adult is not only small but underdeveloped, with normal or nearly normal size head, moderately retarded trunk and relatively short legs.

Let me make quite clear at this stage that this does not necessarily mean that all small adults are underdeveloped. There are still all too many whose small total height is attributable to rickets in infancy. Often they are people with a high growth potential whose parents were quite simply ignorant of any special need for material for calcification of bone. But, apart from these, and possibly differences of build due to variations in endocrine balance, it would be expected on general principles that children continuously underfed would grow into underdeveloped adults. If this is so, a difference in body proportions should be demonstrable in association with differences in height wherever persistent differences in nurture are known to exist. And that, I submit, can even now be confirmed by observation in any city in this country, if one looks for it.

Proportionate growth of children

The graph depicting social gradient in height of schoolchildren between 1927 and 1935 which is to be found in *Food, Health and Income* (Orr, 1937) does not represent the extreme difference at that time. Selection of data for poor urban council schools gave a curve that was still lower. I tried to find recorded data to test my hypothesis about difference in shape, but unfortunately leg length is a measure not often recorded. At that time I found only one set of data (Hansen, 1932) from which similar conclusions had been drawn and which quoted, from an earlier observer, that 'Full typical development in man implies, relatively to body length, short torso, long arms and long legs'. It is a set of measurements of Copenhagen school children, which included not leg length but sitting height, and deduced that taller children had relatively longer legs and were relatively heavier. (The difference would probably have been more obvious if leg length had been measured.) Anyway, when the Carnegie U.K. Dietary and Clinical Survey was planned at the Rowett Research Institute in 1937, cristal height (height from the floor to the highest point of the iliac crest), as a measure of leg length, was included in the measurements to be made. Statistical analysis of the data, divided into groups according to expenditure on food, has only recently been completed and the gist of the results is as follows. I am indebted to Mr Quenouille of the Statistical Department, University of Aberdeen, for the information.

The three measurements, height, cristal height and weight were analysed to find out which predicted most reliably the expenditure group to which a child belonged. It was found that cristal height was consistently better than total height for indicating expenditure group and, for age groups under 12, height was better than weight. In other words, the difference in leg length was relatively greater than that in either total height or weight.

That is to say, this idea was statistically confirmed as far as social gradient is concerned; and confirmed at a time when that gradient was already rapidly diminishing. But I shall not transgress into Dr Weir's field and talk of secular changes in growth at this stage. Instead, I am going to suggest that the consciousness of this difference is universal and so fundamental that it colours both our literature and our art. In romantic literature, the hero and heroine are always long limbed. If the heroine is small, and of course some readers like her that way, it is always expressly stated that she is 'perfectly proportioned'. Conversely, if the villain has to be large, he is also coarse,

brutish or gorilla-like. The same is true in art. One will admit I suppose that advertisements, because they aim to use the most popular imagery, might be a good mirror of popular taste. High-class fashion journals depict women with an extreme length of limb, and decorative art does the same for both men and women. One may think some of the drawings absurdly elongated. Yet, compared with photographs of ballet dancers and mannequins there is, in fact, no great difference in proportions. A very small increase in the ratio of leg length to total height has a surprising effect on appearance. Conversely, a proposition may be proved also by the Euclidean device of *reductio ad absurdum*. When the artist wishes to depict the lower orders, as such, or the comic, he draws people with exaggeratedly short legs and makes them fat, with results which suggest the 'low-high' pig.

Growth target

All this, to my mind, implies that there is a general awareness of a growth target which is physiologically sound because it means full development. And, quite unconsciously no doubt, because the later-developing parts, the legs, suffer most in underdevelopment, their elongation and perfection of shape become the symbol of perfect growth.

The next question is whether this symbolism is purely aesthetic (with a possible tinge of snobbishness) or whether it has some other connotation. And that brings us to health.

What is meant by fitness

It would of course be absurd to suggest that all tall people are necessarily healthy or that all small people are sickly. It might be, and indeed it is, argued that there is no tougher adult than the undersized survivor from the slums of some of our cities. But that is an argument that cuts both ways. If they have survived the tempest of the slums because they are specially tough, then it seems a pity that so many of the breed die young and that they are not beautiful as well as tough when they do grow up. If I argue on these lines I am given one of two answers. The first is that, of course, they are the product of 'natural selection' and therefore obviously superior, the doctrine of the 'survival of the fittest'. The greatest disservice, perhaps, that scientists ever did for mankind was to produce this association of ideas between natural selection and improvement. The entirely false analogy it suggests with such metaphors as winnowing chaff from grain; the altogether false idea that 'the survival of the fittest' implies some absolute virtue and not merely fitness to survive in a given environment, whatever it may be, have done much to hamper and nothing to promote either agriculture or human progress. Progress in any branch of science means increasing control over nature; this is true everywhere from the tilling of the soil to the splitting of the atom: to be content to abandon a large section of the population to the mercy of a man-created, evil environment is so unscientific as, fortunately, to have something quite unreal about it.

The second answer I get is that size has no virtue in itself and that what we want is 'strength'. It is difficult to pin this school of thought down to an exact definition of strength. If they mean 'brute' strength in the general sense of ability to toss cabers and lift heavy weights, or similar muscular performances, that, I should think, is of minor, and rapidly diminishing, social significance, and, in any case strength in that sense is so closely dependent on training that it is hardly worth arguing about.

I am reminded of an early attempt to assess physical fitness by measuring vital capacity. The list of persons examined, in descending order of merit, began with boy scouts and ended with beggars and gentlemen.

The idea in the minds of some, at least, of these objectors appears to be that the tall child is something of a 'hot-house plant', a false and dangerous analogy. Obviously if one turns hot-house plants straight out into a cold and unsheltered garden, they will suffer more than plants reared in the open garden, if they can be so reared. But that does not mean that they are inferior or less desirable plants. It simply means that the hot-house is a more desirable environment, for a particular purpose, than the garden. We come back, full circle, to the dandelion and the edelweiss. It all depends on whether we prefer them well grown or stunted and picturesque, if they are picturesque when stunted.

Development in relation to health

Before we leave this question of 'strength', it is perhaps worth while to ask whether height, *per se*, is of any disadvantage in muscular work. An athletic friend of mine thinks it is in football and another suggests that a high proportion of well-known light-weight boxers have come from Glasgow. It would be of great interest to have statistics of the heights and weights of distinguished athletes. So far, I have been able to find only one study, a very recent one (Tappen, 1950) of the world's champion weight-lifters and their records. There are three lifts: the two-arm military press, the two-arm snatch, and the two-arm clean and jerk. The order of weight lifted in the first two is on the average about one and a half times body-weight, and in the third about five times body-weight. In all, the performance is closely correlated with body-weight: coefficients 0.85, 0.82 and 0.80. Since weight and height are themselves correlated, efficiency increases also with height. When weight is held constant, the press lift is hampered by height, slightly but significantly; in the snatch, height has a slight but not significant favourable effect on performance. Hence, as far as this goes, height is a slight handicap where the stance is rigid, and the performance relatively slow, but where speed and agility are required in addition to muscle strength, then height is positively correlated with lift.

So much for feats of muscular strength. Where 'strength' involves also endurance there is indirect evidence that height, weight and performance are correlated. For instance, the lower limit for admission of regular recruits to the navy has been consistently higher than that for the army; and that for the air force (flying personnel) was, at least to begin with, still higher. That meant, of course, that these services were recruited from progressively higher strata of society. Further, within these services, rates of sickness and invaliding decreased as height and weight rose. Put the other way round, rejects, including those who passed the height test and were rejected on medical examination, were, on the average, smaller than those accepted; and, in times of depression, when the supply of labour exceeds the demand, the same is true.

Development in relation to resistance to infection

We have then, so far, no evidence against the view that better development implies greater physical fitness. What other criteria, in the present state of knowledge, can be applied? Morbidity

data are few for the general population and it is difficult to disentangle the causes of sickness. Since all the social circumstances, housing, sanitation, spacing of population and hence exposure to infection, as well as education and, on the whole, facilities for prompt medical attention, improve with, and at about the same rate as, growth, it is difficult to judge whether inhibition of growth, itself has any effect on morbidity. Examples could, I think be cited from animal experiments but I prefer to draw such deductions as may be from human populations. Evidence is afforded by the wartime history of tuberculosis. In both world wars, where diet deteriorated to the extent of inhibiting the growth of children, the incidence of tuberculosis rose in proportion. Such an increase might be attributed to simultaneous deterioration in housing, hygiene, isolation and hospital treatment, such as did occur. If we take the two wars separately the deterioration of the environment by destruction of houses, overcrowding, blackout, and failure of isolation were incomparably greater in the second than in the first, but the effect on tuberculosis, especially in Germany, was much less. Not only was the actual rise less, but the transition from the benign and chronic to the virulent military disease (which destroyed the immunological theory of racial immunity), did not occur in the general population, but was seen only among starving refugees and displaced persons. It looks as if underfeeding, which produces underdeveloped people, also interferes with the processes which determine immunity or susceptibility to tuberculosis.

The study of this subject is greatly hampered by the impossibility of recording accurately both attack rate and mortality rate; and even, when we have both, we so often do not have age incidence, and that may have a decisive effect on mortality. For instance, infective hepatitis, during the last war, occurred five times as often in British as in Indian troops, but case mortality in Indians was five times that in British troops. Mortality rates were therefore similar (Witts, 1947*a,b*) and, if only mortality rate and not also case mortality were known, it might be concluded that there was no difference between the two populations at risk. But the attack rate was probably governed by previous exposure and acquired immunity; the case mortality by the health, probably the nutritional state, of the men. It appears likely that similar sequences of events account for much excess mortality in poor children early exposed to acute infection, and apparent toughness of the survivors in later life.

Let us look at another indication, namely morbidity rate for bronchitis, concerning the same children in the Carnegie U.K. Dietary Survey on whom the physical measurements were made (Table 1). We find the longer-legged children suffered less bronchitis than the short at all ages. Since there is neither complicating immunity mechanism nor specific cure for bronchitis, we might argue that the constitution built up when the complete harmonious pattern of growth is unfolded is, in some way, superior to that associated with inhibition of growth, however slight.

The trend of evidence then is that the better-fed and therefore better-developed children and adults are 'fitter', measuring fitness by muscular strength, and 'healthier', measuring health by absence of morbidity where we can get a picture in which the complications can be at least partly resolved.

We cannot go much further in this analysis at present. It would be of the greatest interest to be able to trace accurately

Table 1 Percentage incidence of bronchitis (Unpublished data of the Carnegie U.K. Clinical and Dietary Survey, 1937–1939)

Weekly food expenditure per head of family				
Age (years)	Up to 5s.	5s. to 7s.	7s. to 9s.	9s. or over
Boys				
0–4	20.5	15.4	10.3	4.3
5–9	10.6	11.4	15.0	6.4
10 and over	5.0	4.0	3.2	2.1
Girls				
0–4	17.1	11.1	7.1	–
5–9	8.4	6.7	3.4	4.3
10 and over	4.1	2.8	1.4	1.5

Table 2 Mean age at death of males who have completed 16 years of life (Computed from mortality rates, Registrar-General, 1938)

Social class	...	1	2	3	4	5	All
Mean age at death (years)	...	65.3	65.6	60.4	60.9	60.3	61.4

the further history of well- and ill-grown people in terms of living and dying and causes of death. I can sum up the general picture (Table 2), in terms of social class, which connotes a general difference in standard of perfected growth.

This shows that, for those who have avoided death in childhood, there is a difference of 5 years in mean age at death between the highest and lowest social classes. An analysis of the reasons, in terms of certified causes of death, is not strictly relevant to this discussion, and I will conclude the argument with one further point. The 'low-high' pig, first stunted in growth and, when it is then well fed, becoming obese rather than 'finished' will be remembered. This, I think, has its parallel among human populations, when privation increases with size of family and some degree of comfort is attained only after growth has ceased. It is, of course, most obvious where adult occupation is not strenuous. That is one form of obesity. The other is that of the well-fed, well-grown person who is vigorous and athletic in youth and then sits back into the physical inactivity of an office and the comfort of a motor car, but continues to eat about as much as he did when young and active. Table 3 (Keys, 1949) shows the cost of obesity in terms of the weighting of life insurance premiums.

The moral is not quite the same for both groups. The obesity of the well-grown can no doubt be debited to gluttony and sloth, but I doubt whether that of the ill-grown can be prevented except by the continuation of a spartan regime throughout life, which seems a bit hard. The real answer is good feeding in youth, disciplined eating and habits in later life.

Summary

To sum up briefly: we have seen that there is a physiological basis for preferring tall and long-legged people because, in general, that type represents completion of growth and appears to connote a certain superiority of constitution. I have suggested that there is an awareness of merit in this type which is reflected in literature and art. It is reflected also in the utilitarian sphere

Table 3 The cost of obesity (America) (Life insurance premiums weighting)

Height (ins)	Premium				
	100 Weight (lb)	115 Weight (lb)	129 Weight (lb)	141 Weight (lb)	154 Weight (lb)
60	90–169	170–180	181–195	196–209	210–220
64	98–180	181–194	195–206	207–223	224–236
68	110–198	199–214	215–227	228–245	246–259
72	126–219	220–236	237–252	253–268	269–284
76	142–246	247–263	264–283	284–298	299–314

of selection for employment. This is rational up to a point, but even where robustness and muscular strength are of little or no immediate importance, there is still a tendency to discriminate against the short, and still more against the generally small person unless he is exceptionally gifted. If one is heavy, one may get by on the basis of being a 'solid' man, but that may have its penalties too.

This discrimination may obviously be of considerable social importance and it would be interesting to have an analysis of mental ability, or of socially valuable performance, in relation to attained physical growth.

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Commentary: Isabella Leitch (1890–1980)— a personal memoir

John Pemberton

Isabella Leitch was a remarkable woman. I came to realise this soon after I started work at the Rowett Research Institute in September 1937 where I had been appointed to carry out the clinical examinations of the children in the Boyd Orr survey. My colleague, the late Angus Thomson, and I frequently sought her advice, which was often blunt but always helpful.

Isabella Leitch had spent the 1914–18 war carrying out research in genetics and plant physiology at Copenhagen University with Prof. A Krogh, a Nobel prize winner in medicine, and she had obtained her DSc at Aberdeen University in 1919 on the basis of published and unpublished papers.

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In spite of the fact that she was, by then, an experienced research worker, she was unable to find a research post in Aberdeen. At length, in 1923, she found a job as temporary librarian at the Rowett Research Institute.¹

She soon found herself involved in various research programmes, first in animal nutrition and later, when the Institute became involved in human nutrition, in that area too. Her encyclopaedic knowledge and sharp critical mind made her a key figure and she became adviser and personal assistant to the director, John Boyd Orr.

In the 1930s, the signs of specific nutritional diseases such as scurvy, rickets and pellagra, were well known, but there was little agreement on the early symptoms and signs of



malnutrition. Certain changes in the skin, anaemia, defective night vision, increased frequency of infections and slower rates

of growth were suspected to be signs of malnutrition and we looked for these.

Angus Thomson and I often consulted Dr Leitch (as we always called her), and she was able to give us valuable advice on what was worth looking for and measuring in the children we were going to examine. One such piece of advice she gave us, which has turned out to be very significant, was to measure leg length as well as total height.

The Boyd Orr survey was completed in July 1939 but, because of the outbreak of war in September and the dispersal of most of the research workers, the full results were not published until 1955.²

A few papers on specific aspects of the survey were published before this. One of these was Isabella Leitch's paper on growth.³ In this she drew attention, among other things, to a finding from the survey that the 'longer legged children suffered less bronchitis than the shorter legged at all ages'.

Although I occasionally experienced her severe criticism, as when she returned the draft of an article I was proposing to send to *The Lancet* blue pencilled all over, I remember her with respect and affection. She was one of those scientists whose honesty, knowledge, high standards and willingness to help the less experienced workers, enhance the reputation of an entire research institution.

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Commentary: The catch-up dilemma— relevance of Leitch's 'low-high' pig to child growth in developing countries

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Isabella Leitch's paper¹ is exciting reading in many ways. Our main difficulty in writing this commentary was choosing a single topic from her several original ideas that have major

relevance to the nutritional problems of developing countries today. We opted to discuss the possible hazards of catch-up growth.

Leitch's analogy of the growth of pigs and humans is thought-provoking. Based on research on pigs who were starved early in life and then fed appropriately, she observed that 'skeleton and muscle will not grow as they would have done if they had had the opportunity at the right time, and the extra food will be used mainly to lay on fat'. She then uses this analogy of the 'low-high' pig to propose that humans who suffered malnutrition in early life would be better off by remaining thin than

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by putting on weight: 'growth potential is not something that one can speed up and slow down ... and get the same result in the end'. She concludes by arguing that this condition 'can probably be prevented only by the continuation of a spartan regime throughout life, which seems a bit hard'.

Leitch's 'low-high human' has short legs, since leg length is more heavily compromised by malnutrition than trunk (or crown-rump) length. Her discussion of the aesthetic appeal of long legs and of its associated health benefits is both amusing and scientifically challenging.

Recent data suggest that low-high humans are indeed becoming increasingly prevalent, as obesity rapidly escalates in developing countries^{2,3} affecting individuals who also faced childhood malnutrition. Although small size at birth is not usually associated with greater adiposity in adults,⁴ children who are stunted in early childhood may have a greater risk of later obesity in countries undergoing the nutrition transition. Studies from Brazil, China, Russia and South Africa suggest that stunted children have a 2–8 times greater risk of becoming overweight.⁵ In Guatemala, stunted children are particularly prone to central fatness as adults.⁶ Leitch's low-high human might well be referred to as the 'stunted-obese' human.

At this stage, it is useful to distinguish two types of catch-up growth. Early catch-up refers to children who are born small but catch up in weight and height in infancy or early childhood. Late catch-up affects children who may or may not be born small, but who became stunted in infancy or childhood and catch up later in weight but not in height, becoming 'stunted-obese'.

In less developed countries, about one in every four newborns is growth-retarded.⁷ Growth monitoring programmes provide nutritional advice to mothers whose children are underweight or not gaining weight as expected.^{8,9} For children aged 6 months or older, this implies providing additional energy-dense and micronutrient-rich foods to promote catch-up growth.¹⁰ Since measuring child length in first-level health facilities is problematic, weight-for-age is used to assess risk and progress. Growth promotion programmes do not differentiate early from late catch-up and provide feeding advice to all malnourished children under 5 years. They have been strongly endorsed by the international paediatric and nutrition communities, and by agencies such as WHO and UNICEF during the last 30 years.

Leitch is concerned primarily with the long-term effects of late catch-up. Her hypothesis is consistent with the rapid increase in chronic disease that is already taking place in developing countries,^{11–13} and with the well-known detrimental effects of trunk and upper body fatness.¹⁴ However, recent literature arising from the Barker hypothesis¹⁵ suggests that early catch-up may also be detrimental. This is cause for much greater concern.

Evidence for long-term negative effects of catch-up growth

Leitch's paper was visionary by anticipating, by three to four decades, what later became known as the Barker hypothesis.¹⁵ Studies summarized below further advance this hypothesis by suggesting that catch-up growth in childhood may affect adult health. Some of this evidence is summarized below. It should be noted that some studies do not separate catch-up growth in

small newborns from above average growth in all children, nor test for interaction between birth size and later growth.

A 1995 report from England¹⁶ suggested that fast growth in the first year of life was associated with lower coronary morbidity. However, at least four other studies reported opposite trends. In a Finnish cohort, boys who were thin at birth but whose weight caught up had increased coronary heart disease mortality;¹⁷ for girls, catching up in height rather than weight was associated with higher coronary morbidity and mortality.¹⁸ Another Finnish study showed that subjects who were in a higher quartile of body mass index at age 7 than their birth-weight quartile appeared to have an increased risk (odds ratio = 2.3; 95% CI: 0.9–5.4) of metabolic syndrome—high blood pressure, dyslipidaemia and insulin resistance—in adulthood.¹⁹ In England, infants who gained more than 0.67 z-score of weight or length for age between birth and 2 years were fatter and had more central fat distribution at age 5 years.²⁰

Other studies report on growth from birth to adulthood. In Wales, the highest risks of coronary heart disease were for subjects whose birthweight was in the lowest tertile and their adult body mass index in the highest tertile.²¹ In Sweden, men who were light at birth (<3250 g) but whose adult height was above the median presented higher blood pressure.²²

Another relevant English study is the 60-year follow-up of the Boyd-Orr study, cited in Leitch's paper. Low-income British children who received food supplementation in the 1930s had a 13% (non-significant) increase in adult mortality compared to non-supplemented children. The study lacked statistical power to detect small but important differences in mortality.²³

Several other publications addressed the long-term consequences of low birthweight on chronic diseases. However, as Lucas *et al.*²⁴ pointed out, in many of these the association only become apparent when adult body size was adjusted for. This suggests that relative growth or change in size—and not small size at birth *per se*—was the factor associated with chronic disease, since:

$$\text{CHRONIC DISEASE INCIDENCE} = \text{Intercept} - \text{Slope}_1 \cdot \text{SIZE AT BIRTH} + \text{Slope}_2 \cdot \text{ADULT SIZE}$$

or

$$\text{CHRONIC DISEASE INCIDENCE} = \text{Intercept} + \text{Function}(\text{ADULT SIZE} - \text{SIZE AT BIRTH})$$

Results of these studies suggest that early catch-up in both weight and height may affect adult health. Although the evidence supporting the fetal origins hypothesis is compelling, important methodological concerns have been raised^{24,25} and additional research is needed to provide solid ground for policy making, particularly regarding the effects of catch-up.

Evidence for short-term beneficial effects of catch-up growth

We will now review some of the evidence that catch-up growth has short-term advantages. Although the literature on catch-up growth itself is limited, there is strong evidence on the association between malnutrition, morbidity and mortality.

Weight-for-age deficits markedly increase the risk of death in children under 5 years. A meta-analysis²⁶ showed that, compared

to children with weight-for-age of 80% or more of the NCHS growth reference, the relative risks of dying were equal to 11 for children below 60% of the reference, 3 for children between 60 and 69%, and 2 for those between 70 and 79%. Reviews on nutrition and morbidity also confirm that malnourished children are at a much higher risk of developing serious infections.^{27–29}

These findings have been interpreted as indicating that catch-up growth is associated with beneficial health outcomes, as it would shift children who are born small to a lower risk category. However, stronger evidence would arise by comparing children who (1) were born small and remained small, (2) were born small and caught up, (3) were born large and failed to grow, and (4) were born large and remained so. We are not aware of any studies reporting on morbidity or mortality outcomes for these four groups of children.

Eighteen years ago, we started a population-based birth cohort study in Southern Brazil,^{30,31} and Leitch's paper led us to re-analyse these data. Children born in 1982 were followed up in 1984 at an average age of 20 months, and again in 1986. Being small for gestational age (SGA) was defined as a birth-weight below the 10th centile of weight for gestational age,³² and hospital admissions during 1985 were obtained by maternal recall in 1986. Table 1 shows admission rates according to SGA status and to weight gain from birth until 1984. Hospitalizations were significantly less frequent for SGA children with above-average growth than for those with below average growth; the former even appeared to have lower rates than non-SGA children with slow growth. These preliminary results should be treated with caution, as there is a possibility of additional confounding as well as of Berkson bias, as doctors may be more likely to admit malnourished children.

Summarizing, there is strong evidence that heavier infants and children present lower morbidity and mortality in developing countries, but data on the beneficial health effects of catch-up growth are limited.

Ideally, one would like to have information on the short- and long-term consequences of catch-up growth from the same cohorts. When visiting a 25% sample of our cohort at the age of 15 years, we calculated the prevalences of overweight—above the 85th percentile of the sex and age-specific body mass index⁹—for the four groups of children in Table 1. These prevalences were 9.3% for SGA children with slow growth, 16.7% for those with fast growth, 16.0% for non-SGA children with slow growth and 27.2% for those with fast growth. This

finding highlights the fact that early catch-up may be associated with both short-term benefits and with long-term risks.

Conclusions

In 1950, Leitch raised the challenging hypothesis that individuals who failed to grow optimally in early life might be better off by remaining small than by catching up. Fifty years later, her early programming hypothesis is one of the most challenging areas of research in public health.

Few nutritionists would disagree with Leitch regarding the harmful effects of late weight catch-up in stunted children, producing 'stunted-obese' adults with central body fat distribution. But there will be less consensus on whether or not early catch-up may be detrimental. Although Leitch did not go so far as proposing that fetal undernutrition would be best followed by relatively slow growth in infancy and childhood, some recent evidence suggests that early catch-up may also result in higher adult morbidity and mortality. On the other hand, the international nutrition and public health communities strongly support catch-up growth for small newborns, and there is ample, albeit mostly indirect, evidence that it is beneficial in the short term.

Assuming that both the positive effects of catch-up on child health and its negative impact on adult health are confirmed, there are additional issues to be considered. Firstly, the trade-off between serious childhood infections and late-onset chronic diseases will depend on the epidemiological setting—in poor countries where infectious disease mortality is high, catch-up would have a positive net effect whereas in developed countries the risks of chronic diseases would probably outweigh the benefits of catch-up on child health.

Secondly, economists have brought into public health the idea of 'discounting': most individuals prefer benefits in the short rather than in the long term.³³ Therefore, a positive impact on child survival would have stronger appeal than a negative impact on adult mortality of similar magnitude. This further highlights how difficult it will be to weigh the pros and cons of catch-up growth.

Thirdly, it remains to be seen whether it will ever be possible to implement selective dietary restriction to children who are small at birth. Leitch herself acknowledged that maintenance of 'a spartan regime throughout life' would be 'a bit hard'. Surely, promoting adequate fetal growth seems far more appropriate

Table 1 Frequency of hospital admissions in 1985 according to birthweight for gestational age and to weight gain from birth (1982) to the average age of 20 months (1984)

SGA (small-for-gestational age) status and weight gain from birth to 20 months (1982–1984)	Hospital admissions in 1985 ^a	Odds ratios for admission (95% CI)		No. of children
		Crude	Adjusted ^b	
SGA, weight gain below mean ^c	15.9%	2.79 (1.78–4.36)	2.28 (1.44–3.60)	214
SGA, weight gain above mean ^c	5.6%	0.68 (0.37–1.27)	0.57 (0.31–1.07)	302
Non-SGA, weight gain below mean ^d	9.5%	1.50 (1.13–1.99)	1.35 (1.01–1.80)	1572
Non-SGA, weight gain above mean ^d	7.1%	1.00 (reference)	1.00 (reference)	1499

^a $P < 0.001$.

^b Adjusted for maternal age and schooling, and family income.

^c Mean value of the change in weight-for-age z-score for all SGA infants from birth to 20 months of age.

^d Mean value of the change in weight-for-age z-score for all non-SGA infants from birth to 20 months of age.

than recommending strict diets to children who were born small.

Given current knowledge on child health in developing countries, it seems reasonable to continue to promote growth for small infants and young children. A major priority, however, is to re-analyse existing datasets from both developed and developing countries to assess the full impact of catch-up growth on health both on the short and long term. Let us follow what may be interpreted as Leitch's plea for epidemiological research: 'Lay aside the microscope and look at things with the naked eye'.

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Commentary: Early insights into height, leg length, proportionate growth and health

David Gunnell

Lifecourse epidemiology

The last ten years have seen the focus of chronic disease epidemiology shift from an almost exclusive interest in adult risk factors to a growing appreciation of the role of exposures acting at early stages of life.^{1,2} Leitch's review of growth and health,³ based on a lecture given over half a century ago, is a timely reminder that the scientific rationale for recent interest in the early life origins of adult health is grounded in research dating back more than a hundred years.⁴ But more than this it offers elegant descriptions of confounding, gene-environment interactions, a sweeping dismissal of the eugenic movement and pointers to future avenues of research which have since led to a greater understanding of early life determinants of adult health.

One focus of Leitch's paper concerns proportionate growth in animals and humans. Using examples from research into the growth of pigs and sheep, she suggests an animal's adult body proportions may provide an indication of undernutrition during development. Poor feeding may permanently retard the development of the later developing body segments—their hindquarters, a theme she returns to in later writings.⁵ She goes on to use anecdotal observation and empirical research to extrapolate these findings to humans, stating: *'High class fashion journals depict women with an extreme length of limb, and decorative art does the same for both men and women ... When the artist wishes to depict the lower orders, as such, or the comic, he draws people with exaggeratedly short limbs and makes them fat.'* (page 146). Healthy and attractive individuals, by implication, have long legs and their health may be derived from the effects of good childhood nutrition on 'hindquarter' growth. Leitch reports some early analyses of Boyd Orr's Survey of Family Diet and Health in Pre-War Britain⁶ to support this idea. These suggest that leg length (cristal height) better predicts socioeconomic position than does overall height (page 146), furthermore, longer-legged children suffer less bronchitis (page 149) than short-legged children.

The Boyd Orr cohort

To investigate the long-term impact of childhood nutrition on health Leitch suggests that *'It would be of greatest interest to be able to trace accurately the further history of well- and ill-grown people in terms of living and dying and causes of death.'* (page 149). Fifty

years on we now know the findings of such investigations. In particular, around 5000 of the children who took part in the study in which Leitch was intimately involved—Boyd Orr's Survey of Family Diet and Health in Pre-War Britain⁶—have been followed-up to assess whether their childhood growth patterns and body proportions predict patterns of adult health and mortality (see below).⁷

Most cohort studies that have examined height-mortality associations show that greater stature is associated with longevity. In particular prospective investigations indicate that cardiovascular mortality declines with increasing stature and this association persists after adjustment for a range of cardiovascular risk markers including birthweight.^{8–10} In contrast, tallness is associated with increased risk from a number of cancers—particularly malignancies affecting the breast, colorectum and prostate.^{11–13} In keeping with Leitch's speculations, follow-up of the Boyd Orr cohort suggests that leg length is the component of stature generating these associations. Increases in childhood leg length, but not trunk length, are associated with decreased coronary heart disease mortality and increased cancer risk.^{14,15} The detailed diet records collected in Boyd Orr's study allow a direct investigation into the role of diet in height-disease associations. Studies on the Boyd Orr cohort to date indicate that excess calorie intake may increase cancer risk whereas fruit intake may be protective.^{16,17} Few other studies have prospectively examined associations between the components of stature and adult mortality patterns. Albanes' study of adult leg length and cancer incidence in the USA, however, offers some support for the notion that leg length is the component of stature associated with increased cancer risk.¹⁸ Further research is needed to confirm these findings and replicate observations concerning leg length and cardiovascular mortality. The biological mechanisms underlying height-disease associations are the focus of current research interest.

Growth and health

What has been learnt about proportionate growth in humans in the last 50 years? Whilst a considerable amount of research interest in the early part of the twentieth century focused on health in relation to body shape and proportion,^{19,20} attention in more recent years has focused on the role of body fat and fat distribution in adult chronic disease risk.²¹ A greater understanding of linear growth in the components of stature during childhood has been derived from a number of detailed longitudinal studies. These show that whilst leg length is the component of stature responsible for the greater part of pre-pubertal height

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increases, trunk growth is greater than leg length increases in puberty.²² Thus relatively long legs in childhood may provide an indication of better pre-pubertal nutrition and together with longitudinal findings,^{14–17} this suggests that childhood exposures may influence adult disease risk. Alternatively, rather than necessarily reflecting better nutrition, relatively long legs in childhood may reflect an increased tempo of growth and early puberty. If this is the case then factors setting growth trajectories in early life, rather than the child's later environment, influence relative leg length,² suggesting that childhood circumstances are less important than prenatal and infant exposures in influencing later health. Two lines of evidence point to leg length being a measure of childhood circumstances, independent of prenatal exposures. Firstly McMeekan's animal studies described by Leitch show that poor feeding post-natally may permanently influence animals hindquarter development. Secondly, anthropometric markers of prenatal nutrition are equally strongly related to leg length and trunk length,²³ whereas if prenatal exposures set the tempo of growth in childhood it might be expected that birth measures would be more strongly associated with leg length growth.

Leitch's paper appears to have received relatively little attention when first published although some of the ideas she proposed were taken up by Dugald Baird when he gave the prestigious Cutter lecture in Harvard the following year.²⁴ Her speculations pre-date a range of recent epidemiological investigations into early influences on adult health. The suggestion that leg length is a sensitive and specific marker of childhood conditions requires fuller investigation. Leitch would, no doubt, be intrigued by recent findings in this area.

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Commentary: The contexts of the Carnegie Survey 1937–1940, and Isabella Leitch's 'Growth and health', delivered to the Nutrition Society in October 1950

David F Smith

In April 1936, at a meeting of a Sub-Committee of the Advisory Committee on Nutrition of the Minister of Health for England and Wales and Secretary of State for Scotland, there was a discussion of John Boyd Orr's recently published *Food, Health and Income*.¹ The book included a graph of height against age for different groups, showing that at age 14 some public school boys, later revealed to be from Eton College,² were, on average, over five inches taller than some council school boys. Orr, director of the Rowett Research Institute near Aberdeen, commented that this was to be expected when the diets of the two groups were compared. But the minutes of the meeting record a discussion between JN Buchan, a medical officer of health, EP Cathcart, professor of physiology at Glasgow University, HE Magee of the Ministry of Health and E Mellanby of the Medical Research Council (MRC), which show there was little consensus among the experts of the day on the desirability of height:

Buchan: Is there any greater value in height or weight? I cannot see any great advantage in it.

Cathcart: Industrially, height is a drawback.

Magee: Would you suggest that, as you know that food has to do with physique of the individual, we should feed people in accordance with the employment you think they ought to take part in?

Buchan: That was not my suggestion. My suggestion was that health was apart altogether from the question of height or weight. That was really my suggestion: if we want to feed people for good health or perfect health, the question of making them two or three inches taller does not to my mind necessarily arise.

Magee: You cannot make them two or three inches taller, you can satisfy, or you cannot satisfy, a growth impulse. That is what it amounts to.

Mellanby: You can on average. It is a good thing to be taller and stronger.

Cathcart: Industrially it is not.

Mellanby: Quite apart from that, I would much rather see a fine Colonial person walking along the streets than the average person walking along our streets.

Cathcart: Industrially, it is not an advantage. I see recruiting people, and they have a height of 5ft 8ins and that is all they want. They are more comfortable amongst machines.³

This exchange illustrates the context in which Orr and his colleagues planned the Carnegie dietary and clinical survey that began about a year later. Controversy about the conclusions to be drawn from the data in *Food, Health and Income* was a major reason for embarking on the new project. Orr argued in *Food, Health and Income* that the diet of about 50% of the population was deficient in one or more nutrients, and he hoped this finding would usher in a new era of government intervention in the food system. He wanted a comprehensive national food policy to support agriculture and bring a good diet, and optimal growth and health, within the means of the whole population. But apart from some expansion of the existing milk-in-schools scheme, there was little action. Orr regarded the lack of agreement among the government's advisers on nutrition as a major reason for this. The large-scale Carnegie project, he hoped, would provide the data that would create consensus and oblige the government to act decisively.⁴

Much of the responsibility for designing the Carnegie survey fell to Isabella Leitch. Leitch had first been employed at the Rowett in 1923 as librarian and then as assistant to the physiology department and Orr's personal assistant. In 1929 she joined the newly establishing Imperial Bureau of Animal Nutrition which Orr directed and which was based at the Institute. The Bureau published *Nutrition Abstracts and Reviews* from October 1931, which was sponsored by the Reid Library of the Rowett Institute, the Imperial Agricultural Bureaux, and the MRC, and covered both animal and human nutrition. In this capacity she acquired an encyclopaedic knowledge of nutrition and assisted the Institute's staff with many projects, and Orr with background papers for his committee work.⁵

Between 1937–1939, when the survey was underway, and October 1950 when Leitch delivered her paper to the Scottish section of the Nutrition Society,⁶ the context of nutrition science had changed. Before the survey was completed and written up, the war had intervened. The Advisory Committee on Nutrition was allowed to lapse, but some of the Carnegie data were rapidly made available to the Ministries of Health and

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Food for use in the preparation of wartime food policies. In addition, Orr and his son-in-law, David Lubbock, who had supervised the Carnegie survey, published *Feeding the People in War-time* in April 1940, which included some of the dietary data.⁷ This publication was part of a process of lobbying by Orr and other scientists for a scientific wartime food policy. This campaign, coinciding with setbacks in the war, changes in government, and the government's need to improve public relations, led to the creation of a committee to advise the government on food policy, to which Orr and other outside experts were appointed. The Scientific Food Policy Committee was part of the cabinet's committee system, but it was soon superseded by an internal interdepartmental committee chaired by the chief medical officer of the Ministry of Health. Nevertheless, the Scientific Food Policy Committee helped to shift influence in food policy away from the Ministry of Agriculture and the agricultural lobby and towards the Ministry of Food.⁸

After difficulties early in war, British wartime food policy is generally regarded as having been a great success. But the population faced many inconveniences and there was a widespread expectation that, as soon as the war was over, rationing would be eased and the diet improved. In the event, food controls intensified. From August 1946 bread rationing was introduced for the first time, and between November 1947 and April 1948, a potato control scheme operated.⁹ In the light of the mounting controversy in the press about the effects of the food situation on health and productivity, in October 1947 the British Medical Association (BMA), decided to appoint a Nutrition Committee to prepare a report on the adequacy of wartime and post-war diet.¹⁰ Within government, senior Labour Party politician Herbert Morrison pressed upon the Prime Minister the advantages of reviving the Scientific Food Policy Committee. This, he suggested, would help the government to combat criticism with an announcement that 'scientists of unquestionable standing' were advising the government on 'how to make the best of the food situation'. However, the views of advisers who cautioned against the risk attached to involving outsiders prevailed.¹¹

Orr had now left the Rowett to become director general of the Food and Agricultural Organisation, but the BMA invited his successor at the Institute, David Cuthbertson, to join their Committee. Cuthbertson declined the invitation, although he and Leitch, who was now director of what had become the Commonwealth Bureau of Animal Nutrition, did provide some advice.¹² It had been made clear to Cuthbertson that he had been appointed director of the Rowett to work on the nutrition of animals of agricultural importance and not on human nutrition.¹³ Animal nutrition research was the original role of the Institute,¹⁴ and it was only through support such as that from the Carnegie Trust, the establishment of the Imperial Bureau, and Orr's involvement in official policy making, that he had been able to conduct human work. Cuthbertson's response to the invitation to join the BMA Committee illustrates the new atmosphere in which nutrition scientists worked in the early post-war period. There was a pressure not to stray into high-profile policy questions and politics, and, in the agricultural research institutes, to concentrate on science which would enhance agricultural productivity. The fate of the research programme of Angus Thomson at the Rowett emphasizes the situation. Before the war he had been, with John Pemberton,

one of the doctors employed for the clinical part of the Carnegie survey. After the war, he was engaged by the Institute for some research on pregnancy in the ewe, but found he was effectively forbidden to extend this into human work.¹³ By the time he spoke on 'Human fetal growth' at the same Nutrition Society conference at which Leitch appeared in 1950, he had transferred to the Department of Midwifery at Aberdeen University.¹⁵ But as Leitch was employed by the Bureau rather than the Institute, she was free to work on a wider range of topics. However, the prevailing context meant that it would not be possible to revive at the Rowett a project along the lines of the Carnegie survey, and Leitch and her collaborators relied largely on data collected by others in their later publications on growth.²

The existence of the Nutrition Society in 1950 is another contrast between the pre- and post-war context. The Society had been formed after a group of nutrition workers had been meeting informally and sending documents on wartime food policy to government departments, offending Mellanby's sense of proper procedure. In response, Orr took an initiative in 1941 leading to the establishment of the Nutrition Society. All the early meetings were on practical themes related to wartime needs but after the war some of the conferences became less obviously policy-orientated, and the Society's journal also began to publish original articles as well as conference papers.¹³

In early 1950 the BMA Nutrition Committee reported, drawing conclusions that presented few challenges to the government. The 'health of the population', they declared, 'despite the trials and tribulations of recent years, has been well maintained'.¹⁰ During the year the food situation began to ease, and public and political controversy began to abate, at least temporarily.⁹ The Nutrition Society meeting on 'Growth' therefore took place at a time when nutrition was becoming less political, there was a trend towards the disengagement of nutrition science from politics and policy, and attempts were being made to look for and emphasize longer-term scientific agendas. Leitch's paper gave her an opportunity to demonstrate her own far-reaching scientific vision and shows that her contribution to the design of the Carnegie survey had given the exercise a permanent value, well beyond Orr's commendable but more immediate objectives.

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