The Importance of Genetic Influences on Growth in Early Childhood with Particular Reference to Children of Asiatic Origin

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The growth of long bones and vertebrae, on which body stature largely depends, takes place through activities within the growth plate, a zone of dividing cartilage cells separating the primary from secondary ossification center that deposits substances containing mineral salts to form the bone matrix. As the rate of growth slows, the growth plate becomes narrower until eventually bone in the main shaft joins with bone in the epiphysis, eliminating the growth plate in the process. No further growth is then possible, the epiphysis being "closed." At the same time as bone growth takes place, constant remodeling and molding are also occurring in order to sustain the changes in shape demanded by the growing skeleton. For normal skeletal growth to take place, dividing cells must be provided with an optimum metabolic milieu supplying energy, amino acids, vitamins, minerals, and various endocrine secretions. Without balanced provision of these substances, growth will fail.

The growth curve of any child is the outward expression of the interaction between its genetic potential and environment. Its various stages—embryonic, fetal, neonatal adjustment, infancy, later childhood, and the adolescent climax—merge smoothly and imperceptibly with each other to provide, under normal conditions, a smooth continuum from conception to adulthood. In most instances this growth journey proceeds harmoniously, avoiding or at least mastering many potential obstacles on the way. On occasions, however, the journey is deviant, either setting off in the wrong direction (as might occur with genetic errors) or, more commonly, falling foul of environmental hazards, the most prevailing of which in the present world being a lack of food. These distortions lead to failure by the child to achieve its genetically endowed physical growth potential, becoming stunted. Faltering of linear growth rate is one of the most sensitive of all measures of socioeconomic adversity within any community.

The aim of this chapter is to provide some insight into how genetic makeup influences the linear growth program in both individuals and populations so as to see in perspective the problems of growth stunting, especially its epidemiology.
ONE OF MAN'S MOST HERITABLE Traits

In 1889, Francis Galton in his treatise *Natural Inheritance* (1) recognized the beautiful regularity in the stature of a population that shows "small dependence on differences of bringing up." Together with recognition of correlation of height with midparental size, he was among the first to document stature as one of man's most heritable traits and in so doing set the scene for the beginnings of a scientific appraisal of genetic influences in the control of growth. The importance of genetic influences, dictated by a plethora of genes of small effect residing on many chromosomes, is evident in many ways: variations of size between individuals within the same ethnic group; much higher coefficient for height between monozygotic than dizygotic twins; the close relationship between the mean height of parents and mature stature of their offspring; and differences between the growth curves of boys and girls with the earlier puberty growth spurt in girls and in boys a higher peak growth velocity, to give but a few examples (2).

Where there is abnormal genetic makeup, stature is often affected. Some single-gene anomalies, as occur for example in achondroplasia, cause marked stunting and disproportionate growth. Girls with Turner syndrome (45,XO) grow at a slightly slower than average rate throughout childhood, fail to experience a puberty growth spurt, and end up as very short adults (3). Children with Down's syndrome (trisomy 21) grow slowly from fetal life through the early years after birth, resulting again in shorter than average adult stature (4). These examples, in both normal and abnormal children, leave no doubt as to the importance of genetic factors in controlling and directing the growth program, though how in individual children this genetic control, coded in the DNA template, is transcribed into actual growth processes is still incompletely understood. What we do know is that for the growth genotype to be allowed its optimum expression, growing cells must be provided with an adequate milieu supplying energy, amino acids, fatty acids, vitamins, minerals, and endocrine substances. (See R. D. G. Milner, *this volume*, and R. Rappaport, *this volume*, for a detailed consideration of the physiological determinants of growth.)

TIMING OF PHYSIOLOGICAL GENETIC INFLUENCES

In all living organisms there is an intrinsic "genetic momentum" that results in a minimal growth performance even under the most hostile environmental conditions.

In prenatal life, growth is heavily influenced by the mother through the intrauterine environment she provides for the fetus. At this focal point converge her own genetic makeup, nutrient intake, health (past and present), and lifestyle (in particular, cigarette consumption). So dominant are these intrauterine environmental influences that the fetal genome has been estimated to account for only about 20% of the variance in birth size (5). This powerful maternal control on fetal growth (totally dominating at this stage any paternal influences), recognized first
by Walton and Hammond in their studies of Shetland pony/shire crosses in 1938 (6), has probably evolved in order to allow the safest possible conditions for partu-
rition. For this to operate effectively, the fetal genotype must be subservient to local maternal environmental factors, especially those with the capacity to con-
strain growth. Thus, correlation between length at birth and midparental height is very small, only about 0.2 (2). This changes rapidly over the subsequent 18 months or so, when it reaches its adult value of 0.5. Correlation of length at birth with adult height is also small (about 0.3), but by the age of 3 it has risen to 0.8 (2). These improvements in height correlations with time are outward manifesta-
tions of genetic influences on growth rate, which somehow are brought into play once the child is freed at birth from prenatal maternal influences.

When considered against a background of conventional clinical centile stan-
dards, these genetically controlled "target-seeking and self-stabilizing" effects (7) are often seen as growth shifts with trajectories moving in either upward ("catch-
up") or downward ("lag-down") directions. One study (8) from the late Professor David Smith and his colleagues in Seattle of well-nourished, healthy American children has shown that against a seven-point centile grid (97, 90, 75, 50, 25, 10, and 3), about two-thirds of children crossed more than one centile line over the first year. Those of extreme size at birth showed the greatest shift. Babies relatively long at birth (>90th centile) with a genetic makeup (judged by midparental stature) for smaller size tended to shift downwards after cruising for several months in the higher centile channels at their intrauterine growth rate. Where pa-
rental size was for greater stature, they tended to continue more in the centile channel in which they found themselves at birth. In most of the children, a "steady state" was achieved by about 18 months. In contrast, children small at birth (height <10th centile) born to tall parents showed "catch-up" growth that began almost immediately after birth, most accomplishing their growth shift by about 6 months. Small babies of smaller parents tended, on the other hand, to re-
main in the lower channels in which they were born. Possibly some of the most vivid of all examples illustrating the driving force of the offspring’s genetic makeup in channeling early growth can be found where the prenatal environment has exercised a pathological influence on growth. The baby malnourished prena-
tally from maternal hypertensive disease will show very marked early "catch-up" growth. The baby grossly overgrown at birth as a result of poorly controlled ma-
ternal diabetes provides a mirror image of growth deceleration.

The first couple of years after birth must therefore be viewed as a time when the expression of genes controlling the growth program emerge out of the shadow of maternal intrauterine influences in order to direct the individual baby’s growth trajectory into its genetically predetermined channel. It is this that underlies the remarkable heterogeneity in growth profiles in individual babies over this time. Thus, whenever growth shift occurs, it is worthwhile bearing in mind that genetic influences should be considered as a possible cause as well as those of environ-
mental origin. Failure to do so may sometimes, in individual cases, give rise to mistaken diagnoses of growth failure or, even on occasions, of obesity. This state-
ment will obviously apply less to children in poor countries, where environmental effects on growth will overshadow the more subtle genetic influences, than to children brought up in more privileged environments, but it is nonetheless an important principle to be aware of.

Why genes controlling the switch to "lag-down" should operate later than those influencing "catch-up" is not known. (Might it be that continued smallness is to the detriment of the small baby, whereas the big baby is not in so much haste to find its genetically determined channel?) What is important in day-to-day practice, however, is to recognize that "lag-down" or, as I consider it, "physiological growth failure" does occur and that its occurrence in an individual child should not automatically be considered stunting.

ARE THERE GENETIC ORIGINS TO DIFFERENCES THAT EXIST IN THE STATURE OF DIFFERENT RACIAL GROUPS?

It is against this background of genetic variation within individuals that I now turn to what seems a more contentious issue, namely, the extent to which genetic factors account for the diversity in physical size that exists between different races and ethnic groups.

Examining the physical attributes of the many groups of mankind can not fail to remind one of dramatic genetic influences. Color of skin, nature and distribution of body hair, body type, physical proportions, body composition, and rate of maturation are but a few examples of the ways in which various human subgroups have, over the eons of time, evolved and preserved through natural selection very varied physical characteristics to help accommodate and allow satisfactory adaptation to very diverse habitats. It therefore comes as some surprise to me to discover that where stature of young children is the physical characteristic under debate, this is considered by many authorities to be little affected by ethnic differences. The following statement from a paper by Habicht and his colleagues from Guatemala and published in the Lancet of 1974 introduces such an attitude: "From data collected among privileged groups of children in developing countries, we have concluded that child growth is mainly influenced by social economic status and not by race or ethnicity" (9). Yet no one can doubt the genetic origins of very obvious differences that exist between the adult heights of well-nourished and healthy different racial groups brought up in privileged environments that have allowed genetic potential achieve its full, uninhibited potential (10). Are protagonists of the equal or similar growth potential of various racial groups in early childhood implying that these differences emerge later in childhood?

Whether or not genetic factors are responsible for different patterns of growth between races is of interest not only in its own biological right. It is central to the continuing controversy of the appropriateness of reference growth standards to judge the adequacy of growth of individuals and populations and therefore ultimately to the definition of growth insufficiency (11). Those adhering to the view
of equal growth potential during early childhood for children of all races inevitably favor the notion that growth standards developed from European populations in industrialized countries are appropriate for all child populations and can therefore be used as a tool to survey epidemiologically the problem of stunting.

SOME QUERIES

While admitting to the obvious, that appalling stunting of growth is caused by malnutrition and other environmental scourges (see elsewhere in this volume), can we put gene pools of different racial groups out into the cold in their differential influences on early growth? I believe not.

To argue my case I would first like to trace some of the recent history of this contentious issue. One of the key studies to which we are referred is that of Habicht and his colleagues in 1974 (9). They compared growth in height and weight over the first 5 years in samples of children (presumably) well nourished from developed countries (United States, Britain, Australia) with children in developing countries, urban and rural, assumed to be considerably undernourished (India, Colombia, Guatemala, Thailand). The overall differences between children’s heights from developed countries were small, around 3%. In contrast, differences between these children and those living in poor urban and rural regions were much higher, approaching 12% and as high as 30% for weight. Differences related to race were therefore small compared with environmental effects. “We see no way to demonstrate with this kind of data that the small differences in growth found between different ethnic groups are due to genetic factors.” The authors concluded that growth standards derived from well-to-do children in developed countries should serve as an optimum standard of reference for all children.

In 1981, Graitcer and Gentry analyzed heights and weights of 2,366 children aged 6 months to 5 years from privileged areas of Haiti, Egypt, and Togo (private day care centers, pediatricians offices, and families of government and military officials), populations representing principally peoples of African and Indo-Mediterranean origin (12). These were compared with the reference standards from the National Center for Health Statistics (NCHS) (healthy, noninstitutionalized American children) (13), the growth standard now widely used and recommended as a reference curve to judge the adequacy of growth in childhood (11). The authors found that the fifth, 50th, and 95th centiles were very close to those of the reference population and concluded, as had Habicht (9), that “when adverse environmental influences such as increased exposure to infection and decreased food intake are optimized, ethnic and geographical differences have little if any influence on growth.” They likewise recommended the use of one reference for all countries to evaluate the impact of hostile environmental factors on the growth of preschool children. Professor John Waterlow, in a letter to the Lancet in 1980, wrote: “I am inclined to think that growth potential in the early years of life is not much affected by ethnic differences. If that is so, it seems justifiable to apply North American or European standards internationally” (14). A leading article in
the *Lancet* in 1984 authoritatively seemed to put the lid on the argument with the statement that "the controversy of whether or not growth standards for children developed in Europe and North America are universally applicable appears now to be settled in favor of those who maintain that they are" (11). It reinforced the notion that the "growth of privileged groups of children in developing countries does not differ importantly from these standards." But this brings us to the kernel of the problem. What is important? How great must differences between ethnic groups be before they can be viewed as important? My contention is that any difference is important, however small, if it has an effect on professional opinion to influence and change dietary practices and other aspects of health care.

What is noteworthy, however, is that in the studies referred to, which have had considerable influence on recent thinking concerning genetic influences on early growth, little reference has been made to children whose origins are in the Far East—Chinese, Japanese, and Indo-Malays—who make up more than a quarter of mankind. These ethnic groups are currently classified as Asiatic (10), and it is this term that is used in this chapter. As adults brought up in fortunate socioeconomic circumstances, they are smaller than Europeans and Africans (10). But to what extent is this the consequence of variations in growth after birth during early childhood rather than later on? Before the case for a similar early genetic growth potential can be accepted for all races, with the crucial implications of such a statement for the definition of stunting, the early growth of these children must be analyzed in further detail and compared with that of other populations.

To this end I have analyzed eight sets of growth data published over the past 20 years from Japan (15,16), Hong Kong (17,18), Singapore (19), Korea (20), Thailand (P. Khanjanasthiti et al., *unpublished data*), and Taiwan (21). Children making up these studies were brought up in what were generally considered by the various authors to be favorable socioeconomic conditions. A growth curve has been constructed from mean heights at various ages over the first 5 years (1, 3, 6, 9, and 12 months, and 2, 3, 4, and 5 years) and compared with mean curves of linear growth from three sources chosen to provide a spectrum of growth profiles from other ethnic groups growing up in socioeconomically adequate environments: (a) NCHS curves (representing essentially a European pattern) (13); (b) Nigerian children from Ibadan (22); and (c) Northern Indian children from Delhi (representative of Indo-Mediterranean peoples) (23). The major source of comparison is with the NCHS data in view of the ubiquitous adoption of these curves as a reference (see elsewhere in this volume). I realize that there are several sources for error and inconsistencies in my approach to ethnic comparisons—differences in sizes of populations studied, different methods of measurement, diversities in environmental conditions, and variable time of assessment, to give but a few. Admitting to these, I do believe that the resulting mean growth curve is near to the truth for these Asiatic children.

Over the first year the average percentage differences in height measured at five different points (1, 3, 6, 9, and 12 months) between the biggest and smallest Asiatic populations is about 3%; from 1 to 5 years, about 4%. These differences are
of similar magnitude to the differences between Habicht's privileged groups in European children of European origin (9). Figures 1 and 2 show the mean growth curves against a background of the range of heights of the Asiatic populations studied. Over the first few months or so, growth in length follows a pattern very similar in all study groups. Thereafter, there begins a very definite period of growth deceleration reflected as a downward shift, which continues for a few years. Figure 3 makes a more simplified comparison of the mean Asiatic curve with the NCHS data, showing a marked downward shift over the first few years after birth. Figure 4 gives a more detailed comparison of the Asiatic growth curve as a percentage of the NCHS median. The downward shift reaches to about 95%.

A more quantifiable and universally better applied measure of this downward shift can be obtained by analyzing changes in the standard deviation score (z score) of the NCHS reference (Fig. 5). Within the various populations studied, there was considerable variability in growth pattern, but all followed a similar downward trend. In both boys and girls there is, over the first 3 years, a shift of approximately 2 SD. Over the next 2 years, there is some upward shift, but not much.

The data in the studies referred to are widely used in their various countries as reference growth standards. It is not recorded that the children were anything other than healthy, although it is possible that within the group as a whole, some children's growth might have faltered for pathological reasons. It is difficult, therefore, in my opinion, to avoid concluding that the early growth pattern of Asiatic

![FIG. 1. Mean curves of growth in length from birth to 12 months in boys and girls of various racial groups. 1: Asiatic (Japan (15,16), Hong Kong (17,18), Singapore (19), Korea (20), Thailand (P. Khanjanasthiti et al., unpublished data), and Taiwan (21)]. 2: Northern India (23). 3: NCHS (13). 4: Nigeria (22). The hatched area represents the range of means for Asiatic children (15–21; P. Khanjanasthiti et al., unpublished data).](image-url)
FIG. 2. Mean curves of growth in length from 1 to 5 years in boys and girls of various racial groups. Numbers 1–4, hatched area, and sources of data as in Fig. 1.

FIG. 3. Mean curve of growth in Asiatic children (heavy curve; refs. as in Fig. 1) compared with the NCHS centiles (11).
children is different from that of the NCHS reference. If the early period of growth faltering was caused by difficulties at weaning, catch-up would have been expected after this time with provision of a good, well-balanced diet. This does not take place.

A final comparison I wish to make is between the 3-monthly increments of the combined Asiatic data and those presented by Professor Waterlow (J. C. Waterlow, this volume) from several studies of poorly fed babies from developing countries (Fig. 6). There are some real similarities that might tempt one to speculate on nutritional reasons for the faltering of growth shown by the Asiatic children. I believe, however, that a sizable part of the faltering is of physiological origin. I propose that this downward shift after birth in Asiatic babies makes a sizable contribution to the eventual smaller adult size of Asiatic peoples.
These views receive some indirect support from other published studies. In 1965 Ashcroft (24) in Jamaica studied the size of 5,000 children aged 11 to 17 years from similar upper and middle socioeconomic classes attending eight fee-paying secondary schools in Kingston. The statures of African, Afro-European, and European children were similar, indicating a similar potential for growth in height. That of the Chinese children was, however, consistently smaller. At 11 years the boys were 3 cm shorter. An earlier puberty growth spurt saw this narrow to about 2 cm at 13 years. The gap then widened until at 17 years the differences between the Chinese children and those of African and European origin amounted to about 7 cm. Ashcroft was firmly of the opinion that this difference was genetic in origin. Barr and his colleagues from San Francisco in 1972 (25) also showed in 7,500 children aged 5 to 14 years that, in general, children of "yellow skin" were markedly shorter and lighter than white and black children. Tanner, in his 1976 review of population differences in body size, shape, and growth rate, also concluded that genotypic differences did exist between Asiatic peoples and those of European and African origin (26).

CONCLUSIONS AND IMPLICATIONS

I wish now to summarize briefly my thesis of the relevance of genetic factors to linear growth in early childhood.

1. Differences in genetic makeup undoubtedly account for variations in size between individuals within any ethnic group who are brought up in comparable environmental conditions. This genotype has a particularly strong effect in the first few years after birth, when the child moves away from heavy maternal influences on prenatal growth. This will often result in considerable shifts in both upward as well as downward directions, which must be always borne in
mind in evaluating the profile of early growth in an individual child. These shifts are greater in children born at the extremes of size.

2. Variations in size between different racial groups, even allowing for hostile environmental factors, also frequently have a genetic basis. Pronouncements on the uniformity of genetic growth potential in infants and young children of the major racial groups are premature and do not apply when children of Asiatic origin are compared with those of European, African, and Indo-Mediterranean origin. The early growth characteristics of Asiatic children differ in their basic profile, showing a deceleration of growth rate over the first couple of years of postnatal life. Superimposed on standard growth curves, these show a downward shift and a continuation thereafter in lower-centile channels.

3. These characteristics of the Asiatic growth curve are likely to be more of genetic than environmental origin and make a major contribution to overall smaller adult stature. These children seem not to have a genetic potential “to approach the European/American norm” (14).

4. The magnitude of these differences of genetic origin do not under normal circumstances approach those that can be caused through malnutrition and other sources of environmental deprivation, where 10 to 20% differences from the NCHS median are being shown with shifts of up to $-3$ SD over the first few years. They should not, however, be disregarded.

5. They can, in the case of Asiatic children, be sufficient to query the validity of using the NCHS reference standard for these children to determine the prevalence of stunting. At the same time, failure to recognize a sizable physiological contribution to the period of early lag-down growth can lead to mistaken attitudes by professional people to such an extent that wrong feeding practices may be implemented in an attempt to reverse the trend. This is the case in Hong Kong, where the interpretation of faltering as being caused by nutritional deficiencies has questioned more traditional methods of feeding in early childhood.

6. If deviations from a widely accepted standard are such as to cause concern, then surely they are clinically important however small in comparison they might be. To recognize genetic differences is in no way to undermine the importance of environmental causes of growth faltering. It is to create a firmer scientific basis to allow us a more reliable epidemiologic survey of the problem of stunting. This will become increasingly important as faltering growth becomes acknowledged as an important objective measure of social and environmental disadvantage.

7. Further study is called for, including careful examination of growth patterns in Chinese and other children of Asiatic origin. Leaving aside the argument of an internationally accepted growth standard as an epidemiological tool for quantifying malnutrition in different countries, the use of such a standard in the routine day-to-day clinical use in individual children might lead to an inaccurate assessment of a clinical problem. Growth failure might be overdiagnosed, and its mirror image, obesity, underdiagnosed.
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DISCUSSION

Dr. Waterlow: I am totally convinced by your presentation, but at one point you used the words "optimal standards" for the NCHS standards. I think that is a misinterpretation. Let us call the NCHS standards a reference without questioning whether they are optimal or not. For various practical reasons, we need a reference. The question we are really asking is not related to children growing below the reference, except as a method of description, but to the differences in growth between different groups of children and the biological and practical meaning of those differences. Would you agree that we can get right away from the idea of an optimal standard?

Dr. Davies: Yes, I agree. The NCHS standard is a point of reference but not an optimal standard.

Dr. Martorell: The data suggest a lower growth potential for Asiatics. However, I was a little bit disturbed by the magnitude of the difference that your data suggest. You have data from as early as the 1960s. I think it is clear that in Japan as well as in China, there have been significant increases in stature over time. So I would be cautious in terms of the magnitude of the differences. At this stage, I would be willing to contemplate a recommendation that for Asiatic populations maybe the 25th centile ought to be the denominator rather than the 50th centile, but some of your samples showed medians closer to the fifth centile, and I think that is too low.

Dr. Davies: That was an "average" growth curve, as you say, with data collected between 1965 and 1978. The more recent data that I have from Hong Kong are based on 1978 information, which was collected in maternal and child health clinics. The overall downward shift in this sample over the first 2 years would put the median nearer to the 25th NCHS centile than the fifth centile. But it is the nature of the curve that I think is of interest, because in many ways it simulates quite closely the growth curve of children who are bigger at birth and who then drop down, as Smith et al. have shown (1).

Dr. Nabarro: Are you able to give us some rough idea of what your curves indicate in terms of velocity of linear growth? From looking at them quickly, there seems to be a consistent reduction in actual velocity compared with the expected velocity that one can derive from the NCHS figures. Is that correct?

Dr. Davies: I can't give you any absolute figures, but over the first couple of years, especially from 6 to 24 months, the velocity seems to be consistently lower, after which time there appears to be normal velocity.

Dr. Nabarro: Do you know roughly what the percentage of expected linear growth was?

Dr. Davies: No. What I have done is to compare the attained height at various ages; I haven't looked at velocities, only 3-month increments.

Dr. Nabarro: It may be necessary for us to think more about velocities. Several of us who have been working with economically disadvantaged Asian communities have observed variation in the velocity of linear growth, particularly in the first 2 years of life, dropping down sometimes to 50 to 60% of what is expected and then picking up again at the end of the second year. Dr. Davies' suggestion would imply that there is a reduction in the maximum potential for growth in some Asian populations, but at least the linear velocity remains relatively constant throughout the period. By contrast, those of us who find more variable velocities (compared with the NCHS reference) may well feel confident that we are dealing with something that is more environmental than genetic.

Dr. Martorell: That is a very good point; Japanese data, for example, show a constant distance from the NCHS 50th percentile.
Dr. Guesry: Could the slowdown of linear growth at the age of 3 to 4 years, which you observe in your cross-sectional data, not be caused by changes in the nutritional status of the population during the last 10 years? Wong Hock Boon in Singapore, for instance, mentioned that during the last 15 years there has been an increase in stature of about 5 cm in boys around 15 years of age (2).

Dr. Davies: Yes, I agree with that possibility. However, a study from Jamaica (3), made 20 years ago, looked at the heights of schoolchildren of Chinese, African, and European descent and was able to detect even then a difference of about 7 cm between Europeans on the one hand and Chinese on the other. I believe that the major differences in size between ethnic groups emerge in early childhood and not later on in life.

Dr. Aponso: Have you looked into genetic differences among races and ethnic groups not merely related to total length or height but to upper and lower segment? For example, are certain people taller or shorter because they have longer or shorter legs? Has this been looked into in different races?

Dr. Davies: All this is very well documented in Eveleth and Tanner's book (4). The smaller lower body segment of children of Asiatic origin is first observed at 18 months to 2 years; it is not evident in the first couple of years. I don't think that this differential growth of upper and lower body segments contributes to the initial decrease in linear growth.

Dr. Martorell: Professor Tanner and colleagues published a study on the secular trend in stature in Japan (5). The Japanese are now taller, and it is interesting that the increases in stature largely reflect increases in the length of the extremities, so that today the young people from Japan have body proportions similar to the British population. It has all been in leg growth.

Dr. Gopalan: In many of the Asian countries, there are such tremendous socioeconomic disparities that when we speak of populations considered to be living in specific socioeconomic conditions, it can be erroneous. The stratification in different classes—high and middle income, slum groups, and the truly affluent group—is extremely complex. In China, the differences between the North and the South are so wide that people are talking about using different standards. On the basis of certain population groups, to make a generalization about Asian children may perhaps be a bit premature. One has to see what sort of socioeconomic conditions we are talking about. Have the affluent groups really been in a position to express their full genetic potential?

Dr. Mukherjee: Growth is interrelated with social, environmental, and nutritional factors, especially in low socioeconomic groups (6). How could we differentiate the effects on growth of social and environmental factors and of genetic factors in low socioeconomic groups? How could we distinguish, among children from lower socioeconomic groups, those whose growth is poor because of social and environmental factors or because of genetic factors?

Dr. Davies: Your question is really very complex, and I am unable to answer it. What I'm trying to draw attention to in my chapter is that there is not a universal equal growth potential among all children, taking Asiatic children simply as an example.

Dr. Waterlow: In relation to Dr. Mukherjee's point, it has been suggested that more intelligent people tend to marry better. You could construct a model by which genetic factors have promoted a good environment. I don't know whether this would hold in India.

Somebody said that populations in Asiatic countries are bursting at the seams, and this I think is indeed true in such countries as Indonesia, Nepal, and India, whereas in Africa space and land seem not usually to be the limiting factors. Many African groups are no-
mads, living in the savannah. There was a question about body build: African legs are longer in adults than those of Europeans. So my question is: Would you regard the genetic smallness that you have described as a useful adaptation for survival in a highly overcrowded population with enormous pressure on food supply?

**Dr. Davies:** I wonder if it is an adaptation to shortage of food or perhaps to living in a warm climate? If you compare the growth patterns I described with patterns of growth in the Alaskan Eskimos, who are also of Asiatic stock, then there is a difference. There is not the same faltering. The American anthropologist Coon traced the origins of Asiatic peoples to the cold climates of northeast Asia; many of the physical characteristics of the Asiatic peoples are adapted to heat conservation. If these now migrate to climates where heat conservation is a disadvantage, biological changes might evolve. I wonder whether the pattern of physical growth that I've described is more an adaptation to an environmental climate change than an adaptation to overcrowding?

**Dr. Martorell:** I have always been amazed the other way around. Why, when there is an obvious advantage to being small, in requiring less food and so on, don't we see very clear genetic differences in growth potential? It seems that the human species does not emphasize genetic mechanisms of adaptation but relies instead on phenotypic expression.

**Dr. Tomkins:** Have you any information on the way in which populations or subgroups within populations who are taller and shorter also vary over secular periods with respect to weight for height? Is there anything to back up Prof. Waterlow’s point about the pastoralists who are very tall and thin and who are exposed to quite extreme changes in the temperature of the desert areas. How has body mass index or weight for height changed over a period of time in such populations? It would be rather interesting to see that in relationship to changes in height for age.

**Dr. Davies:** I don't have any data.

**Dr. Keller:** An analogous case is the Bushmen in the Kalahari Desert. The Bushmen are very thin and certainly do not have a build that would conserve heat even through the temperature differences between night and day are extreme in the Kalahari.

**Dr. Nabarro:** Dr. Martorell has expressed surprise that there appear to be no genetic differences in the ways in which populations respond to adverse environmental circumstances. He has also suggested that there are circumstances in which it might be to people’s advantage to grow less in length or height in order that they might eat less food. Professor Davies, your results indicate that the growth of some of the children with whom you have been working deviates from the NCHS reference figures. Do you believe it possible that different groups of children, subjected to similar adverse environmental circumstances, may show different characteristics? Might children in one group respond by reducing their rate of linear growth and maintain their ratio of lean tissue mass to height, whereas children in the other group maintain the expected linear growth rate but reduce their lean tissue mass-to-height ratio?

**Dr. Davies:** I don't know how many generations would be needed before these changes take place. It is certainly likely that if you have a group of people who are biologically distinctive in their growth characteristics, they may well respond differently, but I don't know in which direction.

**Dr. Martorell:** What about the old idea that children with a faster growth potential have therefore greater needs and are thus more at risk for severe malnutrition under conditions of relative lack of food?

**Dr. Waterlow:** That is what I suggested to Prof. Davies. It might be a positive advantage when food supplies are limited. On the other hand, should we not agree that everybody
has the "right" to fulfill his genetic potential, which presumably includes growth in the same way as, under the United Nations Declaration of Human Rights, he has a "right" to health? I think there is evidence, certainly crude evidence, that for adults, it is smart to be small. It was shown in the prisoner of war camps during the last war that the small soldiers survived much better than those who were big. I've always tried to promote the slogan, "it is smart to be small," but that's for adults; you need less clothing, food, and space.

Dr. Keller: I have recently analyzed data that were collected from laborers during the second World War in Germany: body mass indices decreased progressively in parallel with the deterioration of the rationing, and this affected the tall people much more than the short people.

Dr. Davies: If the growth curve that we observe in Asiatics is truly a biological characteristic, how right are we in applying the NCHS standards? Dr. Keller, I wonder if part of the darker areas on the world map of stunting that you showed (Fig. 1 of W. Keller, this volume) can perhaps be accounted for by what I have just described?

Dr. Keller: The trouble is that what you call Asiatics make up a very small part of that, because I showed only data on that part of the world from the Philippines and Indonesia, which wouldn't really fit, and Singapore, which didn't show up because it is so small. Thus, only Thailand, Burma, and part of Nepal are left.

REFERENCES