Growth of School-Age Children

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Nutrition researchers have paid less attention to the periods of growth in midchildhood and adolescence than to early childhood. The reasons we have focused on growth in early childhood are compelling. The magnitude of growth faltering, the absolute rates of undernutrition, and the strong association between growth faltering and morbidity and mortality in young children have drawn our collective intellect, our research energies, and our programmatic efforts to the period of early childhood. So, you might consider the subtext of this chapter: Childhood growth in the schoolage years—can we continue to assume it is good enough?

The 1995 World Health Organization (WHO) monograph Physical Status: The Use and Interpretation of Anthropometry(1) devotes a chapter to adolescence, although the period just before adolescence, from 5 to 10 years of age, receives almost no attention. In the introduction to the chapter on adolescence, the committee explains: “Historically, the rapid changes in somatic growth in adolescence, the problems of dealing with variation in maturation, and the difficulties involved in separating normal variations from those associated with health risks have all discouraged workers from developing a body of knowledge about adolescent anthropometry that would link it directly with health determinants and outcomes” (p. 264).

They are right: I have certainly been perplexed on many occasions as I have attempted to write something new and useful about this remarkable period of human development. It is difficult to draw strong conclusions from the evidence at hand. But I hope to make the case in this chapter that the issues are fascinating and important and deserve more of our collective intellect and energies in the future. I hope to highlight some useful future directions.

To focus my efforts, I have used the simple framework shown in Fig. 1. The lifespan is divided into three stages: preschool, school-age, and adult. In each of the stages, we are interested in growth itself (the boxes), the extent to which environment—including nutrition—can influence growth (the ovals), and the functional consequences of variations in growth (the circles). For simplicity, I have represented all of preschool growth as one stage; one could further separate that period into prenatal and postnatal growth. For the moment, let us leave the boundary between
preschool and school-age growth vague. School-age growth, according to my
assignment, includes prepubertal, pubertal, and postpubertal components.

We also expect that, however we define these stages, growth in a given period in-
fluences growth in the subsequent period; hence the horizontal arrows. We also know
that preschool growth influences some school-age consequences, for example, cog-
nition and intellectual development. Fetal or early childhood growth might also af-
flect aspects of reproduction in adolescence and adulthood; thus, Gambians born in
seasons of deprivation (with lower birthweights) have different postpubertal mortal-
ity rates than do Gambians born in seasons of plenty (2,3). We cannot be sure how
much this effect is mediated through fetal growth per se, but it is at least a strong cor-
relate; hence the diagonal arrow.

In this chapter, I will focus on four questions, indicated by the numbers on Fig. 1.
First, I will describe school-age growth itself. What is the normal pattern of growth,
and how much does it vary within and between populations? To answer this question
will consume most of the chapter. Second, does this variation in school-age growth
affect the final attained size of adults? Third, what evidence do we have that the vari-
ation in growth in this broad period is attributable to concurrent environment as op-
posed to earlier “imprinting” or genetics? Fourth and finally, is variation in growth
in midchildhood and adolescence linked to functional outcomes that would motivate
public health actions?

Throughout the chapter, I will give most attention to linear growth, although I will
also make some observations on ponderal growth. It is simplest to display only one
gender on each graph, and I will display data describing boys only, whenever possi-
ble. In choosing a gender to focus on, it seemed appropriate to choose boys because
there is evidence suggesting that growth of boys is more vulnerable to environmen-
tal influences than growth of girls.
QUESTION 1: WHAT IS THE NORMAL PATTERN OF GROWTH, AND HOW MUCH DOES IT VARY?

I will look at this question first within healthy populations. Karlberg, on the basis of mathematical modeling of longitudinal growth, divided human growth into three phases: infancy, childhood, and puberty (4–6). This infancy–childhood–puberty (ICP) model fits the data well and is relatively simple. Conceptually, it is appealing because the three stages correspond to different hormonal control mechanisms. For the period of fetal life and early infancy, these hormonal controls are unknown (but not dominated by growth hormone), for the period of childhood, the control is mainly growth hormone, and the period of puberty is controlled by sex hormones. School-age growth includes childhood and puberty growth in this model.

From the third year of life until puberty, linear growth velocity is well described by a decelerating line. The pubertal growth spurt is superimposed on the line of childhood growth. The timing of the pubertal growth spurt varies considerably, both between and within populations—one of the challenges alluded to by the recent WHO Expert Group quoted above. Not only does the pubertal growth spurt vary in timing, it also varies in shape. Generally, children who enter puberty at an early age have a shorter, more dramatic spurt compared with children who enter puberty at a later age (7).

Because of the variability in timing and shape of the pubertal growth spurt, the shape of the average linear growth of a group of children is quite different from the linear growth curve of any individual child (7). The average pubertal growth spurt in a group of children is longer and flatter than any individual child’s growth spurt.

A related result of combining adolescent growth data from a group of children is that the variability around the average value is very much inflated throughout the period of pubertal growth. This increased variability begins at the age that the early maturers take off (around 11.5 years for boys in Western reference samples) and does not end until the age that the late maturers complete their spurt (around 18 years for boys in Western reference samples). For example, in the cohort of Swedish boys that Karlberg modeled, the standard deviation of height velocity was 0.7–0.9 cm/year in boys from 3 to 11 years of age but increased to around 1.2 in 11 year olds and ranged from 1.5 to 2.6 cm/year in boys from 12 to 17 years of age (4). Note that whereas pubertal growth is associated with large standard deviations (as is infancy growth), variation in childhood growth is relatively small and remarkably constant from age 3 to 11 years.

When one plots attained height (rather than height velocity), the variability in growth appears to accumulate throughout childhood. In midchildhood (age 3–11 years), this accumulation of variation is gradual and constant. In adolescence, the greater variability of growth is added to accumulated variation of growth in early and midchildhood, and thus the standard deviations become markedly bigger. This is shown in Fig. 2, in which I have plotted the absolute magnitude of 2 SD from the National Center for Health Statistics (NCHS) reference median value for height for age for boys.

To summarize, I draw three major conclusions from these graphs describing healthy growth. (a) The late preschool and school-age years encompass two distinct phases of growth. Karlberg (4) has called these two phases childhood, characterized
by relatively slow and slightly decelerating growth with constant variability with age, and puberty, characterized by fast growth with varying variability with age. (b) The variability in normal linear growth accumulates over time in a population of children. The standard deviations for attained height become gradually wider throughout childhood and markedly wider as the population moves through puberty. Therefore, the value for $-2$ $z$ scores of height for age becomes progressively further from the typical value. When we examine $z$ scores of populations using an external reference, a population that is tracking along a negative $z$ score is becoming progressively shorter than the reference population in terms of the mean or median height. This is true during both childhood and puberty but becomes more pronounced during puberty. (c) We can expect that any age-based cross-sectional cutoff that we use in this period to separate normal children from abnormal children will become increasingly inaccurate in adolescence. This will certainly be true of the use of $-2$ $z$ scores of height for age to define a stunted child.

QUESTION 1, PART 2: HOW MUCH DOES CHILDHOOD AND PUBERTY GROWTH VARY BETWEEN POPULATIONS, AND IS THE BIOLOGY ELASTIC?

I will show four different patterns of linear growth in populations of children who experience different adverse circumstances. I have chosen these not because they are necessarily typical of developing countries in the year 2000, but because they illustrate the magnitude of variation in growth during childhood and adolescence.
Figure 2 shows the standard way in which I will display growth patterns. To avoid the use of z scores, which obscures the changing magnitude in variability in growth during this period, I have plotted the absolute difference between the median (or mean) height at a given age of the study population and the median height of the NCHS reference data at the same age [after the example of Martorell et al. (9)]. Positive values on these graphs mean that the study population is taller than the NCHS median, and negative values mean the study population is shorter than the NCHS median at the plotted age. I have also displayed the values for −2 and +2 SD from the NCHS median. Figure 2 illustrates how a healthy individual [de Montbeillard’s son, around 250 years ago, plotted from Tanner (7)] and a healthy population [Swedish boys in the last half century (4)] appear when plotted in this way. It is also apparent that healthy individuals (like de Montbeillard’s son) depart from the reference much more markedly than healthy population medians (e.g., the Swedish sample).

**Pattern 1: Prepubertal Catchup Growth**

Prepubertal catchup growth occurs in stunted children who are adopted into better environments. Proos et al. (10–12) have described the growth of 107 Indian girls adopted into Swedish families. The group of girls as a whole was significantly stunted when adopted but showed a remarkable capacity to recover height deficits during the childhood phase of growth (Fig. 3; note that this graph is unique in this chapter because it displays data from girls.) When adopted, their mean age was 3.7

![Graph](image)

**FIG. 3.** Pattern 1: Prepubertal catchup growth. Height deficits of a cohort of Indian girls adopted in Sweden relative to the National Center for Health Statistics (NCHS) median heights of girls. Also plotted are data from an individual girl from the same sample who illustrates rapid prepubertal catchup growth followed by early puberty. [Swedish—Indian data from Proos et al. (10–12); NCHS data from WHO (8)].
years, and their mean height-for-age z score was around −2.3. However, during their first 2 years in Sweden, their mean height-for-age z score rose to −0.8. This means they gained around 5 cm more than the NCHS reference children in a 2-year period. The mean final height was 154 cm compared with the NCHS median of 163.7 cm at 18 years (a deficit of 9.7 cm for the Indian–Swedish girls).

Some but not all of the girls also experienced early puberty, and they attained their adult heights at unusually early ages in adolescence. One such girl is plotted along with the cohort means in Fig. 3. She grew much more rapidly than the NCHS median during two periods of her childhood. In her first 2 years after adoption, she gained over 20 cm relative to the reference. Her growth then slowed but took off again in her early pubertal spurt. As she was an early maturer, she became relatively tall for her age, temporarily exceeding the reference median. But after puberty, her growth ended early in adolescence, leaving her a moderately short woman of 156 cm.

These data are not unique. Marked recovery from stunting in the childhood phase has been observed in adopted Korean children, malnourished Peruvian children taken out of their deprived home environments, South African children successfully treated for kwashiorkor, and English children moved from abusive homes into foster care, reviewed by Golden (13).

**Pattern 2: Late Catchup Growth**

There are several reports of late catchup growth. It is difficult to say whether this growth should be considered pubertal (using the term as defined in the ICP model); it might instead be a prolongation of childhood growth after puberty. With use of the NCHS reference data to define it, the catchup happens in the middle to late teenage years. None of the datasets that I found to be showing this pattern were without some cause for concern over data quality. The largest dataset illustrating this pattern is from US slave data in the early 1900s (14), and this is what I have plotted in Fig. 4. The concern with this dataset is the quality of the measurements, for both age data and height data. However, the author rules out several sources of systematic bias, and I expect that random errors will not have a great influence on the mean values by age, given the sample size of at least 200 children in each 1-year age group. Thus, I do not think that this pattern is spurious. Other studies showing this pattern are the rural Kenyan schoolchildren studied by Kulin et al. (15) and the Turkana children studied by Little et al. (16). However, both of those studies included very small numbers of children, less than 20 in each gender-specific 1-year age group.

The male slaves were extremely stunted by 4–5 years of age—more stunted than modern-day malnourished populations in Guatemala or rural Nepal (see below). In the childhood phase of growth, they appear to grow parallel to the NCHS median, meaning that they would be gaining rather rapidly in z scores. This is somewhat remarkable in itself. By early adolescence, they are around the NCHS 3rd centile, like many developing country populations today. However, the most striking feature of this population is that growth continues well beyond 18 years of age, and by adulthood the male slaves had an average height deficit of only 6 cm compared
with the NCHS median. Steckel (14) attributes the improvement in height from the preschool period to adulthood to the better nutrition that was provided to slaves in midchildhood, around the age of 8, at which age they became economically valuable workers.

**Pattern 3: Prepubertal Stunting Combined with Late Catchup Growth**

The cross-sectional pattern of growth shown by the Bundi people of Papua New Guinea differs greatly from the NCHS reference population at every stage of childhood [from Malcolm as described in Martorell et al. (9); Fig. 5]. The Bundi consume a diet based on sweet potatoes, which is low in energy, fat, and protein. Bundi children are extremely stunted by the age of 5 years—somewhat more stunted than the US slaves at a similar age. Unlike the slaves, however, the Bundi become progressively shorter relative to the NCHS median through the entire childhood phase of growth. Menarche is substantially delayed, and during adolescence, they recover a significant proportion of their height deficit. As adults, the Bundi are very short, but not as short as you might predict based on their prepubertal heights.

A less extreme example with a similar pattern overall is the sample of rural Hyderabad boys measured longitudinally by Satyanarayana et al. (17) from 3 to 18 years of age (Fig. 5). In that publication, Satyanarayana et al. divided the sample into three groups based on their degree of stunting at age 3. I have plotted the mean values for the sample as a whole to represent the population of rural Hyderabad. These Indian boys were very stunted by age 3 but not as extremely stunted as the Bundi boys. In the childhood phase, they continued to fall away from the NCHS
median, but not very steeply. By 18 years, the boys had recovered about the same amount of height that they had lost from age of 3–11.

The Indian sample is important to this discussion because the pubertal parameters of the Preece–Baines model were calculated. The age at takeoff for the Hyderabad sample was 10.7 years, identical to that observed in the British boys (17). However, the age at takeoff was more variable in the Hyderabad sample than in the British sample, and it is clear from the subgroup analysis of Satyanarayana et al. that puberty was delayed in the boys who were most stunted. It is noteworthy, however, that there was some degree of adolescent catchup in the sample as a whole, in spite of the fact that the mean pubertal parameters resembled those of the British sample. Childhood stunting and catchup growth, at least mild catchup growth, can occur in populations in which average pubertal parameters are similar to those in Western groups.

Pattern 4: Prepubertal Stunting with No Late Catchup Growth

The Guatemalan children involved in the Institute of Nutrition of Central America and Panama supplementation study provide a rich longitudinal dataset (18). These children experienced rapid stunting in early childhood that was reduced by intervention with supplemental dietary energy. By 18 months of age, the mean height-for-age z score of boys is below −2 (Fig. 6). From 18 months onward, the median height of boys tracks just below the −2 z-score value for the NCHS data. This means that the Guatemalan boys are accumulating a height deficit relative to the NCHS boys. The downward slope of the Guatemalans in late preschool is less dramatic than for the
FIG. 6. Pattern 4: prepubertal stunting, with no recuperation during adolescence. Height deficits of rural Guatemalan boys relative to the National Center for Health Statistics (NCHS) median heights of boys. [Guatemalan data from Martorell et al. (18); NCHS data from WHO (8)].

Bundi, but it is significant nonetheless. From 2 to 7 years, these boys “lose” 4.6 cm relative to the reference boys, one-third of their final adult deficit.

I am confident that this pattern occurs in contemporary developing country populations because of the quality of these data and because it is also observed in at least two other very different environments. In Fig. 7, I have added to the Guatemalan plot two additional large data samples from Cebu, Philippines (19), and from Sarlahi, Nepal (R. Ram and K. P. West, unpublished data). The Cebu and Sarlahi cohorts are both community-based representative samples. The Cebu cohort is truly longitudinal, whereas Sarlahi is a mixed longitudinal cohort. The similarity in the growth patterns of these three groups of children is remarkable, given their origins in three ethnically and geographically distinct parts of the world. In fact, the Sarlahi sample on this graph lies directly on top of the Guatemala points. The Cebu data differ in design because the children were measured at only three exact ages, plotted as stars. The observations are very similar to those in Guatemala and Nepal. At the moment, we do not have maturational or adult data from Cebu or Sarlahi, but it will be interesting to see if the adolescent patterns in these populations are as similar as their childhood patterns.

Although these children are growing shorter compared with the NCHS median, they are growing parallel to the –2 z-score line, and therefore the prevalence of stunting is constant. For example, in the Sarlahi cohort, the prevalence of stunting by 1-year age group of boys ranges between 50% and 64% from 6 to 13 years old—the high values of 64% being observed in 8 year olds, and the low value of 50% being observed in 12 year olds (R. Ram and K. P. West, unpublished data). These two presentations of the same data—height deficits versus prevalence of stunting—suggest
two very different inferences about these populations. Based on height deficits, one concludes that the Sarlahi children are experiencing progressive stunting. But the prevalence of stunting, defined as less than $-2$ $z$ scores, is constant.

The explanation is that the variance around the NCHS median increases with age, so children at $-2$ SD are progressively further from the median with age. Is this normal growth? No, not if the median height velocity for each age is considered to be normal for a population of children. A child who at age 2 years has a $z$ score of $-2$ and who grows at median height velocity for age [taken from Fels Longitudinal Study data (20)] until the age of 10 years, will attain a height-for-age $z$ score above $-1$ by the age of 10. So, the Sarlahi children are growing more slowly as a group than the NCHS median (and the Fels Longitudinal Study longitudinal height velocities), but they are maintaining, as a group, a constant $z$ score. The same is true for the Guatemalan children and the Cebu children.

It is noteworthy that there are no large longitudinal datasets describing growth of African children. A few datasets have been published, but they are all small (when divided into 1-year increments) and cross-sectional. I hope that our ongoing work in Zanzibar will eventually start to fill this gap. Our data thus far do not provide the longitudinal growth picture one would like to have, but they provide some points for comparison. In Fig. 8, I have plotted on one graph the 1-year longitudinal height increments from two different samples of boys in Pemba Island. The preschool data come from a representative sample of 538 children aged 6 months to 5 years in one large rural village. The school-age data come from a representative sample of schoolchildren in 10 schools on Pemba Island. The older sample does not represent villages;
GROWTH OF SCHOOL-AGE CHILDREN

![Graph showing growth of children in Pemba, Zanzibar, compared to the National Center for Health Statistics (NCHS) median heights of boys. Lines represent 1-year growth increments. Data in 0- to 5-year age range from a community-based sample from one village; data in the 8- to 15-year age range from a school-based sample of children in the first 3 primary grades from 12 schools. (From R. J. Stoltzfus, unpublished data.)](image)

**FIG. 8.** Longitudinal growth of Pemba, Zanzibar, boys, expressed as height deficits relative to the National Center for Health Statistics (NCHS) median heights of boys. Lines represent 1-year growth increments. Data in 0- to 5-year age range from a community-based sample from one village; data in the 8- to 15-year age range from a school-based sample of children in the first 3 primary grades from 12 schools. (From R. J. Stoltzfus, unpublished data.)

It represents schools. It is almost certainly a biased sample, if our purpose is to make inferences about all Pemban children. The exact nature and magnitude of that bias are impossible to determine without a community-based comparison.

Like every population that I have plotted thus far, Zanzibari children experience significant stunting in early childhood. The magnitude of stunting is very similar to that of Guatemalan and Cebu children. The rate of height loss relative to the reference slows dramatically beyond age 3, to an average rate of 0.3 cm/year. From these limited data, it appears that Zanzibari children do better in the late preschool years than the Guatemalan children. However, in later childhood, the Zanzibari children have distinctly slower height velocities than expected, based on the NCHS reference. At these ages, the rate of height loss in the Zanzibaris resembles that of the Bundis. As these are longitudinal height increments of schoolchildren, if better-off children were more likely to be in school, the bias induced by a school sample would tend to create the opposite pattern—that is, a school-based sample growing better than the village-based sample. But instead we see school-enrolled children growing more slowly than the NCHS reference population and more slowly than their preschool counterparts.

I offer two possible explanations for this surprising pattern. The first is that the data are biased. Possible sources of bias are secular trends in food availability from 1994 to 1997, making the recently studied and recently born preschool children not comparable with the schoolchildren, or making the 1-year growth increments in one or both studies nontypical. Another possible source of bias is that we have combined a
preschool sample from one village with a schooler sample taken from all districts on the island. Perhaps Kengeja Village, where we conducted the preschool study, is a relatively well-off village. And finally, we are extrapolating limited preschool data across a gap in our data from 5.5 to 8 years.

Alternatively, the data might be true. I think this is the most reasonable judgment, especially for the data on the schoolchildren, which come from a representative island-wide sample of nearly 3,000 schoolchildren measured at three longitudinal time points. There are also other contemporary data that corroborate that rural East African children have slow height velocities at this age. Figure 9 displays 6-month height increments for 8-year-old boys in several populations compared with the Fels Longitudinal Study median height increment at the same age. The three studies of East African boys yield very similar estimates.

The Zanzibar preschool and school-age data raise the question of whether school may be a nutritionally adverse environment. It seems unusual to ask this question, because we usually think of school as a privileged environment in contexts like rural Africa. But Zanzibari schoolchildren, like many rural schoolchildren in developing countries, typically walk to school after very little breakfast and do not have access to a meal until they return home in the afternoon. During the long school morning, children snack on a piece of fruit, at best. Is it possible that preschool children at home are consuming more food than young schoolchildren? There is support for this hypothesis from Papua New Guinea, where it was reported that children in the village grew better than school-enrolled children of similar age and that provision of milk to schoolchildren improved their growth (21).

![6-month height gain (cm)](image)

![6-month weight gain (kg)](image)

**FIG. 9.** Longitudinal 6-month height and weight increments of 8-year-old boys measured in contemporary studies, compared to Fels Longitudinal Study reference data from US 8-year-old boys (20). [Zanzibar data from R. J. Stoltzfus (unpublished); Kenya coast data from Lawless et al. (26); Kenya, Mexico, and Egypt Collaborative Research Support Program (CRSP) data from L. A. Allen et al. (unpublished)].
Finally, I want to summarize the patterns we have observed across these various population samples. Figure 10 shows the period height deficits for boys in each of the populations displayed thus far. I have divided childhood into three periods that fit the available data but that also correspond, where possible, to the ICP model of Karlberg (4). Growth before age 2.5 years is dominated by the infancy curve in that model, whereas growth from 2.5 to 11 years represents the childhood phase. From age 11 years to adulthood, growth is the sum of the continued childhood component with puberty superimposed. Even in a Western population, the period from 2.5 to 11 years in boys contains almost no puberty component.

The amounts of growth occurring in these periods—expressed here as height deficits relative to a single external reference—vary substantially between populations. The most uniform aspect of this figure is that each of these populations experiences significant stunting in early childhood. The magnitude varies, but in each case, a height deficit of at least 7 cm has occurred by age 2.5 years. In contrast, the periods of childhood and adolescence show much greater variability between populations and are sometimes negative, sometimes close to zero, and sometimes positive.

Thus, regarding the second part of Question 1 (How much does school-age growth vary between populations?), the answer is that it varies a great deal. It varies at least as much as early childhood growth. An interesting conclusion from Fig. 10 is that linear growth retardation—defined as the accumulation of height deficits—occurs before puberty. Although there is variation in pubertal growth between and within populations, populations of children in deprived environments tend to grow as much as the NCHS reference population (e.g., Guatemala) or more than the NCHS reference population during adolescence (e.g., slaves, Bundi, Hyderabad).
QUESTION 2: DOES VARIATION IN CHILDHOOD AND PUBERTAL GROWTH AFFECT ADULT HEIGHT?

I think the answer to this question is yes. There is not a uniform pattern of growth deficits in childhood (the middle bars in Fig. 10) being mirrored by catchup growth in adolescence. I point this out because the hypothesis occurred to me earlier that childhood stunting, when present, would be compensated for by catchup growth in adolescence. However, that hypothesis is not supported by these data. Although childhood growth deficits are compensated by adolescent growth in the Hyderabad sample, no other sample shows this pattern (Fig. 10). In the US slaves, stunting stops after age 4 years (perhaps earlier, but this is where the data begin), and significant catchup growth occurs in adolescence. In Bundi, the childhood component of the height deficit is nearly as large as the infancy component, and the catchup growth experienced in adolescence compensates for only about half of the childhood deficit. The Guatemalan boys accumulate significant height deficits in both infancy and childhood, with no catchup growth in adolescence. I did not include the Indian–Swedish adopted girls, but those data further support the conclusion that rates of childhood and pubertal growth (beyond the period of infancy in the ICP model) do affect the final attained height. And despite the occurrence of early puberty in some of those girls, the adult height is consistently better than predicted by the height of the girls at adoption. Catchup growth in childhood resulted in net gains in adult height in the Indian–Swedish girls.

QUESTION 3: WHAT EVIDENCE DO WE HAVE THAT THE VARIATION IN GROWTH IN THIS BROAD PERIOD IS ATTRIBUTABLE TO CONCURRENT ENVIRONMENT AS OPPOSED TO EARLIER “IMPRINTING” OR GENETICS?

The data from the Indian–Swedish adopted girls show, without a doubt, that variation in growth in childhood and puberty is amenable to intervention. In that sample of girls, the rate of childhood growth following adoption and the magnitude of adolescent growth were both profoundly influenced by an environmental change that included improved nutrition. It is noteworthy that the degree of catchup growth in the adopted Indian girls was greater in children who were more stunted at the time of adoption (11).

The evidence from the US slaves leads to the same conclusion. In the case of the slaves, the evidence may be weaker in terms of data quality, but it is more compelling in terms of feasibility of the changes involved. Steckel (14) argues that the recovery from stunting shown by the slaves is attributable to having more food available around the age of 8 years, but certainly the social and living conditions of slaves continued to be very adverse throughout childhood and adolescence—the point being that a stunted child might be able to resume normal growth without being adopted into an affluent European home.

The important public health question is this: Can childhood growth be influenced by feasible health and nutrition interventions? For several reasons, I would like to
focus this part of the discussion on childhood (i.e., prepubertal) growth rather than puberty and adolescent growth. The data we have reviewed suggest that children in developing countries are growing adequately in adolescence. If they continue to accumulate height deficits beyond the age of 3 years, they do it before puberty. Second, this age interval coincides with a period of time when increasing numbers of children are enrolled in some educational program. Educational settings, either preschool or in schools, offer an infrastructure that might be used to deliver interventions. Third, it makes sense to improve nutrition in settings where education is happening. Although children are constant learners inside and outside of formal educational settings, formal education profoundly shapes children’s future experiences. There is accumulating evidence that better-nourished children learn more in educational settings than poorly nourished children.

Several investigators have evaluated school-based antihelminthic treatment or micronutrient supplementation. Some of these results are summarized in Table 1. Stephenson’s group (22,23), in two trials in coastal Kenya, found that weight gains of primary schoolchildren were improved following antihelminthic treatment. Coastal East Africa is a region where geoehelminth infections are highly prevalent and nearly universal in children of this age. The series of trials done by Stephenson et al. suggests that helminth infections suppress children’s appetites and that antihelminthic treatment results in weight gains. Improved height gains have not consistently occurred in antihelminthic trials, however. In Stephenson’s own work, where weight gains have been found in three randomized trials, height gains were significant in only one study. In Zanzibar, regular antihelminthic treatment resulted in statistically significant but small improvements in linear and ponderal growth.

The results from trials of micronutrient supplementation have also been mixed. In coastal Kenya, Stephenson’s group has reported improved appetite and weight and height gains with daily iron supplementation. In a Tanzanian study, reported only in abstract form (28), the odds of stunting in primary schoolchildren were significantly

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<th>Ref.</th>
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<td>Odds ratio for stunting: 0.52</td>
</tr>
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</table>

aGrowth increment in treated group minus increment in placebo group.
reduced by provision of a micronutrient-fortified beverage. In Zimbabwean schoolchildren, daily zinc supplementation caused significantly greater weight gains in the first 3 months of supplementation, but after 12 months, no differences between treatment groups remained.

Antihelminthic treatment and micronutrient supplementation have been tried in randomized trials because they are relatively inexpensive and feasible to implement in school settings. However, the most compelling nutritional deficit during childhood may be energy. In populations that accumulate height deficits during childhood, the children are quite thin. In Fig. 11, I have plotted body mass index (BMI) by age for several of the populations I have discussed herein. Schoolchildren in Guatemala, Nepal, and Zanzibar are much thinner than children in the first National Health and Nutrition Examination Survey (NHANES I). It can be argued that US children in NHANES I are an inappropriate reference because US populations tend toward obesity. Indeed, it is possible that the BMIs of Guatemalan children should be considered healthy. But I doubt that the BMIs of Sarlahi and Zanzibar children represent optimal health. Unfortunately, we are uncertain of the desirable range of BMI in this age range for the various populations in the world. BMI data from around the world provide valuable comparisons, but we do not know whether this variation in BMI represents a variety of healthy patterns or varying degrees of malnutrition.

A second way to look at energy sufficiency is weight increments. Returning to Fig. 9, we can see the weight increments that correspond to the height increments I discussed earlier. In general, low weight increments and low height increments occur

FIG. 11. Body mass index (kg/m²) by age for boys from various populations relative to 5th and 50th centiles of US boys in the first National Health and Nutrition Examination Survey (NHANES I). [NHANES data from Must et al. (29,30); Kenya, Egypt, and Mexico Collaborative Research Support Program (CRSP) data from L. A. Allen et al. (unpublished); Guatemala data from Martorell et al. (18); Zanzibar 1994 data from 1,860 boys in primary grades 1–3 in 12 schools from R. J. Stoltzfus (unpublished); Zanzibar 1998 data from 647 boys in primary grade 1 in 10 schools from R. J. Stoltzfus (unpublished)].
simultaneously. This is consistent with the hypothesis that energy is limiting growth in these children.

A third way to look at energy sufficiency is weight-for-height z scores. In my experience, our traditional definition of wasting is highly insensitive at detecting thinness in children. In Zanzibar, the prevalence of wasting in children reaches a high value of 11% prevalence in 1 year olds and then is low after the second year of life. In schoolchildren, the consistently low prevalence of wasting by age suggests that these children are of normal weight for height. However, based on their BMI, you can see that Zanzibari children are very different from the NHANES I children. I am not certain whether the difference between these pictures derives from fundamental differences in weight for height and BMI or from differences in the reference populations. It suggests that we have some fundamental measurement issues to resolve in this age group.

A fourth way to look at energy sufficiency is dietary intake data. The Collaborative Research Support Program (CRSP) data provide a unique picture of dietary intake of 8 year olds in three settings: Egypt, Mexico, and Kenya (L. A. Allen et al., Nutrition CRSP Project final report, 1992, unpublished). If the relatively slow growth of the Kenyan children is caused by malnutrition, we should see one or more dietary differences that support this hypothesis. Table 2 shows possible nutrient intake deficits that could be the cause of growth retardation in Kenyan 8 year olds. Most notable are the low energy, fat, and animal protein intakes of the Kenyan schoolchildren compared with Egyptian and Mexican schoolchildren. Writing about results of multivariate analyses of child anthropometry, the authors state: “For Kenya, energy intake appears consistently as a highly significant determinant of child anthropometry when controlling for maternal anthropometry, sex, and SES [socioeconomic status] and sanitation. When energy is entered as the sole intake variable, it is always positive and highly significant. . . . When energy intake is entered with the other macronutrients, it generally continues to be a highly significant determinant for toddler and schooler size. . . . For Kenya (with energy in the model), animal protein is positively related to toddler length and schooler height only when SES is omitted, but disap-

<table>
<thead>
<tr>
<th>Nutrient variable</th>
<th>Egypt (n = 63)</th>
<th>Kenya (n = 38)</th>
<th>Mexico (n = 84)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy kcal/d</td>
<td>1,759 ± 346</td>
<td>1,434 ± 242</td>
<td>1,849 ± 445</td>
</tr>
<tr>
<td>kcal/kg/d</td>
<td>78 ± 20</td>
<td>70 ± 10</td>
<td>88 ± 21</td>
</tr>
<tr>
<td>Protein, g/d</td>
<td>54.3 ± 11.1</td>
<td>42.5 ± 8.3</td>
<td>53.2 ± 12.6</td>
</tr>
<tr>
<td>g/kg/d</td>
<td>2.4 ± 0.6</td>
<td>2.1 ± 0.4</td>
<td>2.5 ± 0.6</td>
</tr>
<tr>
<td>Animal protein g/d</td>
<td>17.5 ± 6.0</td>
<td>2.9 ± 2.3</td>
<td>10.1 ± 6.4</td>
</tr>
<tr>
<td>g/1,000 kcal</td>
<td>9.9 ± 2.6</td>
<td>2.0 ± 1.5</td>
<td>5.6 ± 3.6</td>
</tr>
<tr>
<td>Fat, total (g/d)</td>
<td>43.4 ± 10.1</td>
<td>17.8 ± 4.9</td>
<td>41.9 ± 12.3</td>
</tr>
<tr>
<td>Iron, (mg/d)</td>
<td>10.9 ± 2.4</td>
<td>14.5 ± 2.7</td>
<td>11.9 ± 3.2</td>
</tr>
<tr>
<td>Zinc, (mg/d)</td>
<td>8.0 ± 1.8</td>
<td>7.2 ± 1.4</td>
<td>9.4 ± 2.4</td>
</tr>
</tbody>
</table>

Values are means ±SD.
pears when SES is included in the regression." These results do not prove that dietary deficits are causing poor growth in these children. But, indeed, it would be surprising if children with the protein and energy deficits seen in the Kenyan children were able to grow at normal rates.

To the extent that linear growth retardation occurs in the childhood years, it occurs slowly. Rather large height deficits accumulate over the childhood period because the childhood phase is relatively long—about 8 years between infancy and puberty. The longest of these trials, the Zanzibar and Zimbabwe trials, have been 1 year. Stephenson's trials (22, 23) have generally been 8 months or shorter (see Table 1). If the slow growth retardation that happens in childhood is caused by marginally deficient diets or chronic infections over nearly a decade, we cannot expect to make significant differences in those growth trajectories in less than 1 year or even in 1 year. These short trials confirm that the biology is elastic. However, we need sustained interventions to yield sustained improvements in growth.

So, to answer Question 3, the evidence suggests that growth can be improved during the childhood phase, but the magnitude of growth improvements observed in studies of 3–12 months' duration is disappointing. On the other hand, no intervention has ever been sustained for a long enough period in a developing country context to find out whether the gains in growth can be sustained and become biologically significant. From a programmatic standpoint, the potential to improve growth during the childhood phase is much smaller in any year of life than it is in infancy. To make substantial improvements during the childhood period will require fundamental and long-term changes in children's environments.

**QUESTION 4: WHAT ARE THE FUNCTIONAL CONSEQUENCES OF SLOW GROWTH IN CHILDHOOD?**

This question is perhaps the most important one for public health decision making, but we have little evidence to bring to bear on it. The answer requires studies of various aspects of child development in environments of suboptimal childhood growth. In Zanzibar, we have just completed a study of cognition and learning in first graders, and we will see whether children's cognitive development is associated with smaller growth increments. At present, short adult stature with its known functional sequelae (obstetric risks and low work performance) is the most compelling reason for concern, inasmuch as childhood height deficits appear to comprise a significant proportion of adult height deficits in several of the populations I have reviewed in this chapter.

**CONCLUSIONS**

The main points from this review can be summarized as follows:

- There is substantial variation in childhood growth beyond the first 2.5 years of life that is unrelated (or not only related) to the timing of puberty. We need to come to
some consensus on how to assess this variation. Is it healthy for a population to accumulate a height deficit as long as the prevalence of stunting is constant (i.e., to track at $-2z$ scores of height for age)? Is it healthy for a population to track at the 5th centile of BMI if wasting prevalence is low?

• There is ecologic (i.e., cross-population) evidence and some randomized trial evidence that suggests that this variation is related to environmental factors, including nutrition.

• The variation in childhood growth accounts for a substantial proportion of variation in adult height.

• There is a lack of data on the functional consequences (or even correlates) of slow growth in childhood.

• Karlberg’s ICP model merits more widespread use. We have been so perplexed by the variation in pubertal growth that we have been discouraged from studying both childhood and pubertal growth. It appears that the purely childhood period of growth, from around the age of 3 to 11 years, is an important period in terms of the height deficits that can accumulate and is a reasonably simple phase of development for fruitful study. Karlberg’s infancy, childhood, and puberty phases of growth are more useful concepts than the arbitrary division of preschool and school-age growth, which presumes that the biology of human growth is somehow linked to Western schooling patterns.

REFERENCES

DISCUSSION

Dr. Frongillo: You have done an admirable job in pointing out that when we look at growth patterns in individual children or populations, we are inherently making a comparison with some expectation that we have about that child or about that population. You’ve pointed out that it is easy for us to get confused about our expectations, because we tend to think of a child or a population tracking at $-2$ $z$ scores as growing normally, when, in fact, what it really means is that they are losing ground progressively.

I have a proposal about the use of the term “catchup growth,” which you tried heroically to clarify. The term is a statement about a comparison between what we are observing and what we might have expected to observe, and I now think there is probably no single term that has more obscured our thinking. My proposal is that rather than attempting to save it, we all agree when we leave today never to use the term again!

Dr. Haschke: I wonder whether you could focus on a population where there is substantial so-called stunting and a low final adult height, but which lives in a very favorable environment, which is Japan. So far, we have not discussed the Japanese population. It would be interesting to know if there have been any reports about
factors causing slow growth in the Japanese. Is it only genetic, or are there environmental factors in the Japanese population that also contribute? It can’t be poverty or low socioeconomic status.

Dr. Stoltzfus: I would like Dr. Ulijaszek to comment on that. My understanding is that the Japanese have experienced an incredible secular trend in height and are not short people any more.

Dr. Ulijaszek: They have indeed shown an enormous secular trend in height, and it has not stopped yet. One factor in their former short stature might have been their consumption of phytoestrogens, which may affect growth.

Dr. Ramakrishnan: Have you seen any evaluation of school feeding programs? In India, for example, this is a politically very attractive proposition and has been done in some of the southern states for a long time.

Dr. Stoltzfus: It is a very relevant question. It begs to be done, and it would be good to include that in this chapter. One of the difficulties about school feeding programs is that it is hard to draw causal inferences. Micronutrients and anthelmintics are nice because you can randomize children to different interventions, even within the same classroom. With school feeding programs, you can’t do that, so it is more difficult to draw causal inferences based on such programs.

Dr. Walker: We’ve conducted a randomized trial of school breakfasts for 1 year, and we did find significant benefits for growth, but they were small. That was an efficacy trial. When you move to the effectiveness of school feeding programs, the benefits we may expect for growth are probably going to be quite small.

Dr. Stoltzfus: Do Jamaican children experience stunting, as I defined it, during the school-age period? It sounds to me as though they are growing parallel to the NCHS medians during the childhood phase.

Dr. Walker: As you defined it, I think their height deficit is not actually changing: they are catching up in their z scores, but their height deficit remains.

Dr. Uauy: I’d like to ask about data from schoolchildren in terms of variability. The saltatory model is now being proposed for infants. Is that also happening in school-aged children? Does the timing of the measurement become crucial in validating the variability?

Dr. Stoltzfus: I have not seen that discussed in terms of school-age children. As I understand the work on saltatory growth, the periods are so short relative to the growth increments that we measure, for example, over 6-month periods, that it does not matter a great deal. But perhaps you think differently.

Dr. Uauy: The issue gets back to velocity. From our velocity norms, if a child is found to have no growth for 1 or 2 months, the growth velocity will appear very abnormal. But if the saltatory model applies, you may go for up to 2 months without any growth and then make up with double the growth. So, for the practicing pediatrician, there may be problems when very sensitive measures such as growth velocity are used, which may show no growth for maybe a month or so and then a couple of centimeters in the following months. As a pediatrician, I know I measure my child patients very well, and it is quite common to find periods when no growth seems to have occurred over a couple of months.

Dr. Stoltzfus: The solution would seem to be to measure growth at longer intervals. At least in the USA, it is rare for pediatricians to measure children at intervals as short as 1 month.
Dr. Ulijaszek: The issue is really whether growth is stochastic or cyclic and whether cyclicity changes or whether the whole pattern is dampened during undernutrition. Then, we can begin to understand how best to look at growth velocity in relation to a functional outcome. At the moment, the most perfect tool we have is to spread the measurements reasonably far apart to minimize the effect of short-term processes.

Dr. Frongillo: There are some data indicating that growth is saltatory during adolescence, which means that children grow during a given day and then do not grow for many other days, and then have another day of growth (1). There are people who don’t think that is the right interpretation, but there seems to be broad agreement that velocity is highly variable from week to week or month to month during the adolescent period.

Dr. Uamy: You spoke about the early and late maturers. Bone age is not a really practical tool for public health purposes, so how do you think we can approach the assessment of maturation? Tanner staging may help, but do you have any practical advice on evaluating early and late maturers during various periods of growth that can be applied in the field?

Dr. Stoltzfus: That’s a very relevant question. The most useful thing seems to be height growth velocity. The variation in growth velocity is so small and constant through the childhood phase that when the pubertal growth spurt is starting, the growth velocity z score jumps. I think it would be interesting to get more growth velocity data and look at the issue of variability to see whether there are predictive factors that can be measured easily at the point of takeoff, rather than at landing.

Dr. Rivera-Donmarco: You showed that in the Guatemalan population, there are deficits in growth of about 8 cm between 0 and 2.5 years and about 6 cm between 2.5 and 11, using the NCHS standard. However, if postpubertal growth is more influenced by genetically driven factors, then perhaps if one were to use a Hispanic reference instead of the NCHS reference, I suspect that the differences between 2.5 and 11 years would be much less. Should we use the NCHS reference, or should we use a reference that is closer to the ethnic background of the population that we are evaluating?

Dr. Stoltzfus: I’m open to the opinions of others on that point. As I understood the data that were presented earlier, there is no compelling evidence that we need different genetically based growth standards up to prepuberty. However, I was very interested in Dr. Martorell’s description of the growth of Mexican–American children, who appear to grow well up until puberty and then grow more slowly during and after puberty. We need to continue to look at these issues.

Dr. Guesry: I would like to thank you for using growth velocity instead of actual size. All too often, even in this meeting of specialists, we are presented with attained growth, which is much less precise and slower to react than growth velocity. If pediatricians were to use growth velocity, they would be able to spot problems much earlier and act sooner. And if public health officials were to use growth velocity rather than attained size, they would be able to assess the effect of short-term interventions, which is very difficult with attained size.

Dr. Haschke: I fully agree that it would be very helpful to use growth velocity on a routine basis, but there are major obstacles. It is not normally feasible in pediatric practice because it requires too much accuracy, takes too much time, and you need to make measurements at exactly defined points. Most children do not come to the pediatric office at exactly defined points, so this limits the use of the velocity curves. With the new Euro-Growth curves, we have a computer program that allows one to
calculate growth velocities for weight and height, with computer simulations, when a child has been measured within the last 3 months. However, this is very new, and it has yet to be shown whether pediatricians will use the facility.

**Dr. Stoltzfus:** I strongly advocate the use of growth velocity, but I was directing my remarks more to the public health research community than to the pediatric community, and I agree there are many issues to be considered. But think back to the slide of the standard deviation of growth velocity from Karlberg’s Swedish boys: The velocity during that childhood phase is very constant and so is its variation, which suggests to me that growth velocity during that period is more robust than I expected it to be.

**Dr. Haschke:** I agree, but that is not true for early infancy.

**Dr. Guesry:** I’d like to comment on what Dr. Haschke said. First of all, accuracy is not a problem. If the measurements are inaccurate, they will also be inaccurate for attained size, and no one says we should not measure children because of problems with inaccurate measurements. As for timing, with the new program, it is now much easier to extrapolate the measurements, and so timing is much less important than it used to be. We should be encouraging people to use this modern tool. I have seen too many babies with kidney problems where the diagnosis was made late because nobody cared about growth velocity.

**Dr. Martorell:** Dr. Ulijaszek pointed out that preschool children from China are presently experiencing very rapid growth—just looking at means over time. If you see that in preschool children, do you also see it in school-aged children and in older children, or are those older ages insensitive to the rapid economic changes that are going on? If you could answer that, it might at least give you a clue about whether the response is limited to certain ages.

**Dr. Ulijaszek:** I’ve done some preliminary analyses of a transitional population in Papua New Guinea, which was a quite similar population, and it appears—at least superficially—that the effect is occurring across all ages (2). However, we need to do some proper analyses before we can feel confident about that.

**Dr. Pelletier:** It occurs to me that it would be useful to know whether there are socioeconomic differentials within a given population in the growth increments during later childhood. If there are, that suggests that the disparities in the social environment indeed have physical effects, especially on girls. That would also provide us with an answer to the question you posed: “Is growth good enough?”

**Dr. Stoltzfus:** To answer my own question, I don’t think that growth is good enough. But as a public health person, I’m awed by the work that will be required to improve growth during this period. The reason why the height deficits are so impressive is that the period is so long, not that in any year the height loss is particularly dramatic. So, a continued focus on infancy, where height losses are dramatic in a short period of time and where we can focus our efforts effectively, still appeals to me from a public health standpoint. But I don’t want us to forget these older children. We’ve neglected them because we think they are growing well, but I don’t think they are.

**Dr. Pelletier:** Did you find any studies that looked at socioeconomic differentials in the increments during this age period?

**Dr. Stoltzfus:** The study that comes to my mind is the Hyderabad one (3). The cohort was divided into three groups, and the data were published separately, but I pooled them for the purpose of this meeting. The problem with that is that the socioeconomic status divisions were based on the degree of stunting at 2.5 years. To do what you want
to do, we need to define socioeconomic status in some other way that is less strongly related to our outcome measure! I don’t know of any other data at present.

Dr. Frongillo: I would have a question for everybody. The issue of velocity references has been raised, and this has come up in the context of planning for the presentation of data from the WHO Multicenter Growth Reference Study. In currently prepared references, the contribution of measurement error—either in the construction of the reference itself or from the measurements taken on an individual child—is very small relative to the variability in growth. In other words, the signal/noise ratio is high, so measurement errors can be ignored. In a velocity determination, there are at least two measurements involved, and the signal/noise ratio is rather low: The amount of growth that is occurring relative to the amount of measurement error is actually not that high, and the latter cannot be ignored. This raises the practical question of whether we should include measurement error—and an expectation of what the measurement error is likely to be—in constructing a velocity growth reference or whether we should pretend that all the measurements are perfect and tell clinicians to temper their judgment by the fact that their measurements will have some error. This is a question that we need to answer, and I would like some suggestions.

Dr. Haschke: When we analyzed our datasets, it became very clear that in early childhood, length measurements need to be made at intervals of at least 2 months. This is the minimum, otherwise the noise (error) is so high that the use of velocity curves is very limited. If one constructs growth velocity data for intervals of 1, 2, 3, or 6 months, it is easy to see how the measurement error and therefore the variability decrease. The question is: What is the maximum acceptable variability? In my opinion, the variability in the NCHS data analyses is far too low because of the way the data were analyzed. The Euro-Growth data will have a much higher variability because they will be presented in a different way, so the technique for presenting velocity will also be different. This will also be a very interesting and important question for the upcoming WHO growth references.

Dr. Ulijaszek: With respect to measurement error, we published a paper in the British Journal of Nutrition that looks at how one can interpret measurement error (4). If you take a technical error measurement as your measurement error, that can be turned into a variance measure, which you can use as a standard deviation. This will give you confidence intervals for individual measurements so you can interpret those individual measurements. Alternatively, you can look at the proportion of variance in your population that might be attributable to measurement error. Even though you can’t strip off the measurement error, which is real, you can get some idea of the extent to which it is affecting the measurements you have, so it helps your interpretation.

REFERENCES