Developing World Perspective: 
The Importance of Growth for Short-Term Health

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**Abstract**

Recently, concern has been raised about the potential adverse long-term consequences of rapid child growth. Rapid early childhood weight gain is associated with increased likelihood of being overweight or obese later in childhood and of having risk factors for the development of chronic disease such as insulin resistance and elevated blood pressure. This has led to concerns about the wisdom of promoting catch-up growth in infants born small for gestational age or in children with poor growth after birth. In considering the costs and benefits of promoting catch-up growth, we must not lose sight of the immediate health threats to children in resource-poor environments in developing countries where child morbidity and mortality remain high. The literature on short-term consequences of growth is limited by its focus on attained size as an indicator of prior nutritional status, but generally shows that children with evidence of poor prior growth are at greater risk of morbidity and mortality from common infectious diseases, including lower respiratory infections and diarrhea. In these settings, failure to promote compensatory growth may have devastating short-term consequences.

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**Introduction**

An extensive literature documents the relation of poor child nutritional status, indexed by small body size, to increased risk of morbidity and mortality and poor developmental outcomes. Nutritional rehabilitation of severely malnourished children, feeding programs, and family- or community-based interventions for moderately malnourished children aim to reduce those risks. To return a previously malnourished child to a healthy growth trajectory, short-
term compensatory growth is needed. Improved growth rates, and larger attained body size are frequently used metrics for evaluating the success of treatment.

Recently, concern has been raised about the potential adverse long-term consequences of rapid growth. Rapid early childhood weight gain (typically defined as crossing of major centiles on commonly used growth charts, or a change in weight-for-age z score of >0.67 units), is associated with increased likelihood of being overweight or obese later in childhood [1] and of having risk factors for the development of chronic disease such as insulin resistance and elevated blood pressure [2]. This has led to concerns about the wisdom of promoting rapid growth in infants born small for gestational age (SGA) or in children who experienced poor growth after birth [3]. In considering potential long-term detrimental effects of promoting rapid weight gain in previously undernourished children, we must not lose sight of the immediate health threats to those children in resource-poor environments in developing countries where child morbidity and mortality remain high. In these settings, failure to promote compensatory growth may have devastating short-term consequences.

The so-called ‘catch-up dilemma’ is reminiscent of the small-but-healthy debate in the 1980s. Economist David Seckler argued that short stature reflecting mild to moderate malnutrition was a healthy state that could be considered adaptive on a population level, since smaller people require fewer resources [4]. In contrast, a series of critiques published in Human Organization in 1989 highlighted the costs of mild to moderate malnutrition in terms of functional impairment and increased morbidity and mortality risks [as an example, see Martorell 5]. Chapters on morbidity and mortality risks related to child stunting were featured in a 1986 Nestle workshop on linear growth retardation in less developed countries [6]. Thus, in the words of George Beaton: ‘The story is not new; perhaps it does require periodic retelling to differing audiences’ [7].

The meaning of growth and the extent to which small body size represents an adaptation to enhance survival or a pathological response to constrained resources is at the root of the small but healthy debate and the ‘catch-up dilemma’. The role of adaptation was also the focus of a recent dialogue among human biologists on how to interpret deviations from normal growth trajectories [8]. Growth is a nonspecific indicator of overall health, influenced strongly by nutritional adequacy, infections, and many other environmental factors as well as genetic and epigenetic factors. Accordingly, growth measures are often used to reflect child health at the population level, as for example, in UNICEF’s annual State of the World’s Children reports. Growth measures, interpreted primarily as indicators of child nutritional status, are also used to predict subsequent health and developmental outcomes. This latter use of growth as an indicator of nutritional status is the focus of this paper on the short-term implications of poor growth for infectious disease incidence,
The Importance of Growth for Short-Term Health

severity or duration, and mortality in developing countries. Other papers in this volume address developmental outcomes associated with growth.

While we may be interested in the consequences of growth, the literature mostly provides information about size. This is because growth is most often represented by attained size, typically expressed in relation to a growth reference based on healthy children. Attained size at a given age is assumed to represent prior growth, with ‘normal’ size reflecting adequate prior nutrition and absence of disease. Length or height deficits and stunting are interpreted as measures of chronic undernutrition resulting from the cumulative adverse exposures over a relatively long period of time, while deficits in relative weight (wasting) are thought to reflect more recent and/or acute insults [9].

Why Is Poor Child Growth Related to Increased Morbidity and Mortality?

First, infections and indicators of poor growth such as stunting and wasting share common underlying causes. Poverty, low maternal education, poor sanitary conditions, crowding, inappropriate child feeding practices and poor health care relate to increased exposure to infectious disease pathogens and at the same time underlie inadequate dietary intakes of essential macro- and micronutrients. Moreover, since prior morbidity affects growth, small size may serve as a proxy for prior morbidity. Thus, when an association of stunting or wasting with increased morbidity is observed in cross-sectional studies, a causal association cannot be inferred.

Second, poor growth and infectious disease morbidity are reciprocally related in a synergistic manner: infections increase nutrient needs, depress appetite and accelerate nutrient losses, and poor nutritional status compromises immune function and increases susceptibility to disease [10]. Chandra [11] identified malnutrition as the most common cause of immune deficiency worldwide. Immune function may be influenced by specific micronutrients as well as overall protein-energy malnutrition. In resource-poor settings, multiple nutrients may be lacking, making it difficult to isolate the specific causes of compromised immunity in epidemiologic studies. However, it is well known that prenatal nutritional insufficiency, manifested as low birthweight or SGA and postnatal protein-energy malnutrition manifested as wasting are associated with thymic atrophy, decreased T-lymphocytes, and impaired cytokine responses to infection [for a review, see Cunningham-Rundles et al. 12]. This can result in an increased risk of opportunistic infections, and a reduced response to vaccines. Similarly, zinc deficiency impairs cell-mediated immunity [13] and linear growth [14]. Nutritional rehabilitation can reverse the effects of malnutrition on the immune system, with improvements that parallel or lag behind those in growth [15]. The infectious diseases most affected by malnutrition are those that are most prevalent and contribute to high young
child mortality rates in developing countries, namely, pneumonia, diarrhea, measles, and tuberculosis [11].

**Extent of Malnutrition, Morbidity and Mortality in Developing Countries**

Underweight, wasting, and stunting remain as significant child health problems in many parts of the world. Extensive information on the prevalence and health consequences of child underweight, stunting and wasting was recently summarized based on data from 139 countries in 2005, using definitions based on the WHO child growth standards [16]. 20% of all children in low and middle income countries had a low weight-for-age z score (WAZ<-2), 32% were stunted (height-for-age, HAZ, <-2), and 10% were wasted (weight-for-length or height, WLZ, <-2). Low weight-for-age was most prevalent in south-central Asia (33%) and east Africa (28%). Stunting prevalence exceeded 40% in 40 countries, with most of these being in Africa and Asia. Wasting prevalence is highest, and affects the largest total number of children in Asia: 16% of infants in low and middle income countries, and 27% of South Asian babies were born weighing less than 2,500 g [16].

High under-five mortality rates accompany high rates of undernutrition in these regions. In 2006, 4.8 million children in Sub-Saharan Africa, and 3.1 million in Southeast Asia died before reaching their 5th birthday. In West and Central Africa, the under-five mortality rate was 186 per 1,000 live births. Despite some progress, improvements were insufficient to represent adequate progress toward meeting MDG for the Middle East and North Africa, South Asia and sub-Saharan Africa [17].

**Epidemiologic Evidence Relating Stunting and Wasting to Subsequent Morbidity and Mortality**

*Mortality*

Undernutrition is implicated in more than 50% of all deaths, and of those, mild-to-moderate as opposed to severe malnutrition was the underlying cause [18]. The risk of death from common childhood infectious diseases relates to the degree of malnutrition. Using data on overall and cause-specific mortality from eight low income countries, Black et al. [16] found that the odds of mortality increased with degree of wasting and low weight-for-age, and to a slightly lesser degree with stunting. For example, compared to children with a WLZ >–1, those with WLZ <-3 were 8.7 times more likely to die from pneumonia. Even with mild underweight (WAZ between –2 and –1), odds of death from diarrhea were more than doubled compared to children with WAZ >–1. Though there is some evidence that, paradoxically, malaria incidence
is reduced in wasted children, authors of comprehensive reviews concluded that improved nutritional status lessens the severity of malaria episodes and results in fewer deaths [16, 19].

**Morbidity**

Epidemiologic studies relating underweight, stunting and wasting to subsequent incidence or severity of infectious disease face many methodological difficulties, most of which center on how to deal with reciprocal causality and shared underlying causes of poor nutrition and infection. In addition, hospital-based studies have been criticized for being carried out on highly selected populations. The best evidence comes from community or population-based longitudinal studies which measure growth status prior to the onset of disease, and adequately adjust for confounding variables. Most studies relate child size at beginning of an interval to morbidity in the subsequent interval. In very few studies, autoregressive terms were added to account for prior history of illness.

**Respiratory Infections**

Pneumonia is the leading cause of morbidity and mortality in developing countries. In their review of pneumonia and acute lower respiratory infections, Victora et al. [20] found that most studies focused on WAZ, with a preponderance of studies showing a dose-dependent increase in morbidity risk as WAZ declined. A recent study in Kenya, based on 4 years of surveillance data, found that moderate to severe malnutrition was associated with increased risk of lower respiratory tract infections caused either by respiratory syncytial virus or other pathogens, with stunting being a more important risk factor than wasting [21].

**Diarrheal Diseases**

Long ago, Waterlow [22] noted that the peak prevalence of diarrhea follows the onset of growth faltering, striking in particular, during the period immediately following weaning. He cautioned, however, that while there is a higher prevalence of diarrhea in children who are more malnourished, this association tells us nothing about cause and effect. Extensive work, much of it by Robert Black and his colleagues, has explored the interrelationships of nutritional status and diarrheal disease, finding that diarrhea affects subsequent growth, and that poor child nutritional status increases diarrheal morbidity. In one study, a dose response was found for WAZ, HAZ, and WLZ at the beginning of a 60-day interval with duration, but not incidence of *Escherichia coli*-related diarrheal disease in that interval. The strongest association was with the degree of wasting [23]. Two studies that adjusted for prior diarrhea found increased risk of incident diarrhea associated with low WLZ (Bangladesh [24]) or low WAZ (Egypt [25]), but others found that prior nutrition affected diarrhea only in children with no prior history of diarrhea [26]. Yoon et al. [27]
studied both diarrheal and acute lower respiratory morbidity and mortality in nearly 10,000 Filipino children and found that low weight-for-age was the strongest predictor, with peak effects between 6 and 11 months for diarrhea, and from 12–23 months for respiratory infections.

One study modeled size and growth separately in a birth cohort of >3,000 Filipino infants followed for 2 years. Less diarrhea was recorded in children with faster weight gain, and this effect was amplified in children who were small. [17] Also notable was that this analysis used methods to account for the joint underlying causes of poor weight gain and morbidity, the sequence of events using lagged variables, and the within-child correlation of repeated measures of diarrhea.

The epidemiologic literature provides substantial evidence that poor child growth, indexed by short stature, low weight for age or low relative weight, relates to increased risk of morbidity and mortality associated with diarrhea and respiratory infections in developing countries. While this evidence is interpreted as causal, few studies used methods that strongly support causal inference. Evidence from interventions to improve growth is sparse. Scrimshaw [28] summarized evidence from the INCAP studies showing a reduced number of illnesses per child in response to a supplementary feeding program and from a study in Mexico that also showed a decrease in infectious disease morbidity following supplementation with a daily snack. The lack of studies on how complementary foods may affect acute lower respiratory infections is also noted in a recent review by Roth et al. [29].

**Benefits of Catch-Up Growth**

Aside from the literature on very preterm infants, surprisingly few studies address the health benefits of catch-up growth in children. In their study of Brazilian infants, Victora et al. [30] found that SGA infants with rapid weight gain had 65% fewer hospital admissions, and lower mortality rates than other SGA infants. They note that their paper is the first report on this topic and, unfortunately, subsequent studies have not added to this sparse literature. Some papers have, however, looked at body composition. In general, catch-up growth is characterized by a disproportionately higher rate of fat relative to lean tissue gain. While potentially detrimental in the long run, increased energy stores may be important as buffers against excess weight loss during periods of diarrhea [31]. In contrast, evidence from a large New Delhi birth cohort showed that gain in body mass index during infancy and early childhood correlated more strongly with adult lean mass than with adiposity or central adiposity [32]. Moreover, without early compensatory weight gain, SGA infants have later deficits in height. Both of these observations represent potential beneficial effects of early weight gain [3].
Conclusion: Future Research Needs for Evaluating the Tradeoffs

The literature on short-term consequences of growth is limited by its focus on attained size as an indicator of prior nutritional status, but generally shows that children with evidence of poor prior growth are at greater risk of morbidity and mortality from common infectious diseases. Few studies have directly addressed the effects of prior growth or evaluated potential benefits of improvements in growth status. Thus, insufficient knowledge has been accumulated to adequately judge the possible tradeoffs between short-term health and survival and long-term disease risk in developing countries. In light of continuing high prevalence of child undernutrition, morbidity from diseases such as pneumonia and diarrhea, and increased mortality from these diseases attributable to malnutrition, the tradeoffs for children in resource-poor settings may be quite different from those of more affluent children. Victora et al. [30, 33, 34] have been an especially strong voice, reminding us of the public health importance of understanding these tradeoffs, especially in these settings and in identifying needs for future research. These are noted and expanded below.

Research needs include:

1. A de-emphasis on attained size, and a renewed focus on growth. We need a much more thorough understanding of age- and cause-specific effects of factors that relate to growth restriction and accelerated growth. In particular, information is needed about body composition, the immune system, and regulatory and metabolic systems.

2. Following from (1), we need studies that distinguish the effects of rapid postnatal weight gain in children with intrauterine growth restriction, those with growth deficits developed in the postnatal period, and those without prior deficits [30, 33, 34].

3. A comparison of the long-term effects of rapid linear growth vs. rapid weight gain along with studies on how rapid linear and ponderal growth are related.

4. A determination of whether ‘healthy’ catch-up is possible, and if so, how to achieve it through specific dietary manipulations.

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Adair

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The Importance of Growth for Short-Term Health


Discussion

Dr. Lucas: When we were putting together the cardiovascular bit of the symposium, we were extremely worried about exactly the issue that you expressed, that whilst the data on programming are very exciting, they have to be tempered with exactly the things that you have been talking about, and I hope that that message will become very clear. What I want to do is to perhaps sort of clarify some of the confusion we have had over the management of SGA babies, propose something to you for your view on this as someone who is able to look at both sides of this question. What I would propose is that in the phase of malnutrition or in a high-risk population we ignore programming and we focus on short-term growth. In preterm infants, we ignore cardiovascular programming, focus on brain programming and promote short-term growth. In severely growth-retarded babies in the West, I mean babies that weigh 1.5 or 1.2 kg at term, we regard those as malnourished and actually deal with the nutrition of those babies rather than focus on programming; then we are left with well moderately growth-retarded infants in low-risk environments where they are on a low centile but that doesn't seem to be any particular problem, and then we can perhaps afford the luxury of not actively growth promoting those infants but feeding them on standard diets like breastfeeding and standard formulas. Would you think that that would be a balanced message for the symposium?

Dr. Adair: Yes, I believe that’s a balanced message, but it still leaves us with questions of practicality related to where you draw the line for decision rules and your goals. As we have heard, a number of the clinicians in the audience would like to have better guidelines regarding how much growth is the right amount of growth for a small for gestational age baby. I don’t think that we have heard a clear answer to that, nor are we sure that there is a clear answer to that. I think the answer may depend on whether or not we can manipulate feeding practices to promote healthy growth. Perhaps the composition of what children are fed can be altered to affect fat gain compared to growth in lean body mass, which is important because taller people suffer less from cardiovascular disease, and people with more skeletal muscle tend to be more insulin sensitive. The problem is that promoting very rapid growth results in excess deposition of body fat, which is undesirable, but where we draw the line is really challenging.

Dr. Cooke: I would agree with that, this is an area wherein the science of nutrition has not been applied in a very balanced fashion. Studies are needed which not only evaluate growth and composition but also intermediary metabolism, in terms of insulin resistance, etc. Additional considerations are the more immediate effects of nutrition on immune, pulmonary and, perhaps, gastrointestinal function. So I agree and think we need a much more balanced perspective.
**Dr. Martorell:** I was thinking about an apparent contradiction in some of the presentations. We heard earlier about research using animal models that show that post-natal growth restriction promotes longer life, for example, in rats. Then, we just heard from Dr. Adair that malnutrition and growth failure are powerful predictors of mortality. There is therefore a contradiction that we need to reconcile. Dr. Adair has told us why malnutrition and growth failure kill children. Children in poverty, first of all, live in settings of poor environmental sanitation and suffer from high rates of infection. Second, poorly nourished children have compromised immune systems and have higher case fatality rates compared to children who are well. This brings me to ask about the immune system of the animals made to fail in growth. Did they in fact have a depressed immune system? My guess is that they did but that they lived longer because they lived in the laboratory, free of infections. Had they lived in the real world, with significant exposure to infections, many would have died as a result of their depressed immune systems just like we observe in malnourished children. The research about growth failure and longevity in animal models has been conducted in a very artificial setting, and we have to be careful to recognize this and not to extrapolate these findings to humans.

**Dr. Wainaina:** In my country, we don't restrict the feeding in children under 6 months of age. We let them feed as much as they want because we know that when the mother goes back to work the breast milk will be less available, the food might be poor, and there might be infections. If the baby is not able to feed, we ask the mother to express milk and give it in a cup or a spoon so that the child grows faster. In the national hospital where I work, we deal a lot with children from the poor background, but none of the children is obese. The main problem we have in our country now is that when these children become adults, and that's where the thrifty theory comes in, they have better access to food and this can put them at risk of developing obesity. How would you deal with this problem?

**Dr. Adair:** I think this is where Dr. Lucas' point earlier is so vital because we need to have different messages in different settings and clearly we are not going to restrict feeding of severely undernourished children because this might increase their susceptibility to infection and other adverse consequences. When we get to the child with a z score of –1, the balance becomes a little different. As we are seeing in many low and middle income countries, child obesity is increasing. If we look in many places which previously had high rates of malnutrition, child obesity is now increasing at a faster rate than adult obesity [1], so obviously we need to keep that in mind as well; it's a very difficult balance to think about. Furthermore, if there is a susceptibility issue, so that the poor child who was previously malnourished is more likely to gain weight later when exposed to better conditions, it becomes even more of a challenge for policy makers to think about what kinds of environmental changes should be made to prevent obesity in older children and adults relative to what is still needed to prevent undernutrition in young children, so we are walking a tight rope in many of these settings.

**Dr. Islam:** It is a fact that in developing countries like Bangladesh obesity is increasing among children from more affluent areas. Today, about 5–10% of these children are obese. I would like to ask how do you relate this to the fast food syndrome? Obesity was not an issue in Bangladesh until the arrival of fast food shops. Today, the city of Dhaka is full of them, and people are crazy about the food they sell. If we could deal with this syndrome, perhaps we could fight obesity better.

**Dr. Adair:** Some of the places where we are seeing fairly dramatic increases in older child and adult obesity are places where there are remaining problems with undernutrition. If we look at some of the slums in parts of India, rates of obesity and diabetes in adults are going up [2], while at the same time we see undernourished children, so they may in fact be more susceptible to some of those modernizing influences that you have mentioned. The focus has to be on prevention.
The Importance of Growth for Short-Term Health

*Dr. Islam:* Is obesity a problem among people living in the slums?

*Dr. Adair:* It is becoming a problem according to some of the survey statistics; obesity is growing in low as well as high income populations [3, 4].

*Dr. Gillman:* I would like to address Dr. Lucas about the preterm. I think a lot of discussion surrounds the more severe preterms and a majority of preterms are actually late preterms. At some point, the preterm becomes a full-term baby, and there is merely no difference between a 36.9 and a 37.1 weeker, and so to say categorically that we should feed up the preterms and not the terms, one of the questions is whether we should think a lot more subtly about the crossover between what is term and preterm and whether the sort of long-term deleterious physiology is apparent in the late preterms. We need to balance that more than we do in the more severe preterms.

*Dr. Lucas:* When I made my rather crude classification of how I might deal with different groups, Dr. Adair made absolutely the right remark which is yes that’s all very well, but what we need is better definitions of the cutoffs between those groups, and you have just given a good example where one does need better definition of the cutoff. I don’t think that any of us would disagree that a 28-week gestation malnourished premature baby needs to have his short-term nutrition focused on and be fed well, and that it’s going to improve long-term brain outcome. Concerning an SGA well baby in the West that is not at risk of malnutrition, all that has been suggested there is not an active intervention but a passive one, that is we don’t actively drive this baby up the centile chart because there is some evidence that that might not be a good idea. The cutoff point between those two is a matter of judgment at the moment simply because the data doesn’t deal well with the transitionaries. That is probably the key area for research, to try to dissect these different groups well enough so that we can decide what a low-risk baby is that we are not going to intervene and what is somewhat high risk baby is that we should.

*Dr. Adair:* Exactly, but I still worry a bit about the message because while you say you don’t want to actively promote more rapid growth, what do you do with the hungry baby? Do you not feed him or are you advocating just a lower nutrient density feed to satisfy that child?

*Dr. Lucas:* No, all that has been suggested there is that if you had a moderately growth-restricted well baby in a low-risk environment, you wouldn’t use any special form of nutrition, the baby would either be breastfed ad libitum, and most of those babies will catch up on breastfeeding, or you formula feed with a standard formula ad libitum. All that has been suggested there is that you don’t pile in with that special nutrient-enriched diet but which you might do under some of these other circumstances that we have been talking about.

*Dr. Adair:* And particularly if we could develop targeted nutrient composition. But what about the other end of the spectrum? What about the babies that Dr. Gillman told us about this morning that are already well nourished and growing rapidly? What do we do for them? Do we change what we feed? Obviously we want to breastfeed them, but if they are growing fast on formula what do we do? Do we restrict them?

*Dr. Batubara:* I would like to comment on Dr. Adair’s data on small for gestational age babies. If a baby catches up faster, he/she will have less lean body mass later on and that will decrease the chance of having cardiovascular disease in later life. This contradicts Dr. Lucas’ findings that a small for gestational age baby that catches up faster will be at risk of cardiovascular disease in later life.

*Dr. Adair:* I think that there is still confusion about what we are referring to when we refer to catch-up. Most of the studies on humans have focused on weight gain in part because it’s so difficult to measure length, and many epidemiologic studies do not have good length data or body composition data. If a child is growing in proportion and putting on lean body mass then the adult risks may be different than if the child were
Adair

putting on relatively more weight but not gaining in length, and I think that’s one of the things that we miss when we focus on weight gain. It’s not an issue of our lack of understanding that length is important but rather a limitation of the data in the cohort studies that have adequate information from birth all the way to adulthood. There are relatively few of those cohorts available from low and middle income countries to provide that data [5]. I am working with a group now to try to fill that gap.

Dr. Batubara: I would like to hear Dr. Lucas’ comment on that.

Dr. Lucas: I do agree that all we can act on at the present time if we are going to have any kinds of public health action based on the data, is the data that’s published and the data on long-term health outcomes that’s published, apart from the data that Dr. Adair has been talking about, which is the short-term health issue in at risk communities, is largely based on weight. There is some length data in there, and inasmuch as there is length data it does interestingly go in the same direction as the weight data which at least sort of casts a little bit of doubt on what you just said about healthy catch-up growth. I think certainly the studies that we’ve been involved in have shown that catch-up in length is actually deleterious as well as catch-up in weight, but I would be the first to accept that as yet we don’t have really detailed body composition data. In the next bunch of studies, we will be able to address that question more carefully. But I think that if we are going to respond to current data, all we can do is to do simple things in keeping with what we know. I think the only public health suggestion that has been made in relation to programming is that in low-risk moderately growth-retarded babies we don’t actively intervene to drive the baby up the centile chart given our current knowledge that that might potentially be harmful, and there is no particular evidence in a low-risk population that that is a good thing to do anymore. That’s the only piece of advice that has come out of this, we are not in a position to do anything more sophisticated than that bearing in mind that the data are not more sophisticated than that, and I think that for large tracks of the world what Dr. Adair has been talking about is much more important than what the first speakers have been talking about in relation to programming, and I hope that message is very clear now.

Dr. Adair: And the other thing, I think we need to recognize that the studies of long-term outcomes that try to parse out the relative importance of child growth in the early time period, say from birth to 2 or 3 years, versus adult obesity by and large show us that the impact of adult obesity is much larger than the effects of early growth; so if we want to really deal with the problem of chronic disease, we need to prevent obesity.

Dr. Hui Li: What is healthy catch-up growth? And what is the time window for catch-up?

Dr. Adair: I think that what is optimal growth is probably proportional growth, so we need to look at weight relative to length and at where fat is deposited in the body. We want to avoid excess deposition of abdominal fat which we know is associated with increased cardiovascular disease risk. That’s why it’s going to be so important for us to understand where and how the weight is being gained in a child with rapid weight gain. I don’t know if we can say for sure what the ideal body composition of an infant is, but we may take some hints from the growth of the exclusively breastfed infants. Your second question was the window for catch-up. Again, it depends on whether we are talking about weight gain or whether we are talking about length gain. We know that with respect to adult height, most adult height differences are determined by age 2 [6], so the first 2 years of life are really critical. A number of studies of the long-term effects of excess weight gain or high BMI show that mid-childhood is also an important period for weight gain associated with adult cardiovascular disease risk, so it really depends on the outcome that we are looking at.
The Importance of Growth for Short-Term Health

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