The Natural History of Growth Failure: Importance of Intrauterine and Postnatal Periods

Marie T. Ruel

Food Consumption and Nutrition Division, International Food Policy Research Institute, Washington, DC, USA

The natural history of stunting was described by Waterlow (1) at a previous Nestlé workshop on linear growth retardation in developing countries, held in 1986 in Thailand. The main focus was on describing when stunting starts in populations affected and how long it takes for children to become significantly stunted. Waterlow also addressed the issue of international growth reference standards and elaborated on the potential for catchup growth. His findings were that linear growth retardation starts in the second 3 months of life and that average growth increments remain below the reference standards until at least the end of the second year. Waterlow also hypothesized that growth retardation might even start earlier than the first 3 months of life, but the data he used to examine this question showed no evidence that this was the case. Thus, he concluded that growth rates were comparable with the reference standards during the early postnatal period, but that they started to fall off within a few months and returned to normal after 3 years of age (1).

Data availability was relatively limited at the time, however, especially data from nationally representative samples from developing countries. In addition, many of the reports describing growth patterns of children from developing countries were based on weight-for-age data as opposed to the more accurate indicators of height for age to describe linear growth retardation and weight for height to describe wasting. Few datasets had measurements at monthly intervals, especially in the early postnatal months, and cross-sectional datasets were often not available on sufficiently large samples to allow the analysis to be stratified by age groups. Great progress has been achieved since the late 1980s in correcting these problems, especially with the availability of the Demographic Health Surveys (DHSs) that have now been carried out in a large number of developing countries. These data, although cross-sectional, are particularly useful because they provide information on children’s weight, height, and age and they allow stratified analyses by age group. One of the aims of this chapter is to update previous findings on patterns of growth faltering in children from developing countries using newly available DHS data.
from various countries in three main regions of the world (Latin America, Africa, and Asia).

The chapter is divided into two main sections. The first reviews differences in the geographical distribution and age patterns of wasting and stunting in children 0–36 months of age in developing countries. The emphasis is on describing patterns of growth failure: the timing of growth retardation, the age differentials in the prevalence of wasting and stunting, and the timing and magnitude of maximum growth deficits. The analysis also describes the main features of urban/rural differentials in growth patterns. The second section uses a case study from Guatemala to explore the contribution of intrauterine and postnatal growth retardation to overall malnutrition and the potential reversibility of intrauterine growth retardation (IUGR). The data used are from the Institute of Nutrition of Central America and Panama (INCAP) longitudinal trial carried out between 1969 and 1977 (2) and the follow-up study of 1987–88 (3). First, the importance of stunting at birth in a country with severe problems of childhood stunting is described. Then, stunting at birth is compared with other indicators of IUGR to test their ability to predict the risk of future stunting and the possibility of catchup growth from a nutritional intervention. These issues are particularly relevant for resource allocation for the effective screening and targeting of nutritional interventions.

PATTERNS OF GROWTH FALTERING IN DEVELOPING COUNTRIES

DHS datasets from 28 countries from three regions of the world (18 from Africa, 7 from Latin America, and 3 from South Asia) were used to examine geographical and age differences in growth patterns. The main criteria for selecting the countries were the following: (a) The dataset was released during the 1990s, (b) data on child anthropometry were available, and (c) there was a sample size of at least 500 children in both the urban and the rural sample to allow stratified analysis of anthropometry by age group. For countries that had data available for more than one period during the 1990s, the latest DHS dataset available on the Macro International website (www.macroint.com/dhs) at the time of analysis (1999–2000) was selected. There was a relatively good balance of datasets available from countries in Latin America and Africa, but unfortunately only three datasets from Asia had childhood anthropometric data. This is especially unfortunate because this is the region with the most severe problems of childhood malnutrition in the world globally and a region where both severe wasting and stunting occur simultaneously.

OVERALL PATTERNS OF STUNTING AND WASTING

Stunting

Figures 1 presents overall patterns of stunting, by urban and rural areas of selected developing countries and by geographic location. Stunting is highly prevalent in all three regions, but more so in Asia and Africa. Prevalences below 20% are generally considered low, 20–29% moderate, 30–39% high, and ≥40% very high (3). In Latin
America, most urban areas have stunting prevalences in the low range (<20%), except for Guatemala, which ranks high. Rural areas, on the other hand, have stunting prevalences ranging from low (for three countries) and moderate (two countries) to high and very high (Peru and Guatemala, respectively). In Asia, both urban and rural, the prevalences of stunting rank mainly in the high and very high categories (except for urban Bangladesh, which ranks in the low prevalence category). Africa also shows prevalences of stunting in the low and moderate categories for urban areas and in the moderate, high, and very high categories for rural areas. Madagascar, for example, ranks very high for both urban and rural areas, whereas Malawi and Tanzania show high prevalences of stunting in urban areas and very high prevalences in rural
areas. Rural areas of West Africa rank in the moderate and high stunting categories (except for Niger, which ranks very high), rural areas of Central and South Africa rank among the high stunting countries, and all but two countries of East Africa (Kenya and Uganda) rank in the very high stunting category. Worldwide, the highest prevalences of stunting are found in rural areas of (in descending order) Madagascar (48.4%), Guatemala (46.7%), Nepal (46.1%), and Malawi (42.6%).

Urban/rural differences are generally larger in Latin America than in Africa or Asia. For five of the seven Latin American countries studied, the prevalence of stunting in the urban areas was approximately half the prevalence found in the rural areas. Among the African countries, the magnitude of differences is generally lower than 40%, although the differences are close to one-half in some countries of West Africa (Burkina Faso, Cote d’Ivoire, Ghana, and Senegal). In Asia, urban/rural differences in the prevalence of stunting are all lower than 40%. Although the prevalence of stunting is consistently lower in urban than in rural areas in all countries studied, Fig. 1 shows that considerable stunting is also found in urban areas of the developing world. An important aspect to keep in mind is the fact that socioeconomic differentials in poverty and malnutrition are markedly larger in urban than in rural areas (4). This raises concern about the use of overall city averages to characterize poverty or malnutrition in urban areas, because these statistics hide the large within-urban differentials that exist between socioeconomic groups. Clearly, socioeconomic differentials also exist in rural areas, but they are of smaller magnitude and thus do not affect global estimates so much. We have shown previously that although overall malnutrition rates are lower in urban areas, the prevalence of malnutrition among the most disadvantaged urban children often rivals the levels found among the rural poor (4).

Wasting

The cutoff points used to classify countries relative to the prevalence of wasting are as follows: <4% for low prevalence, 4–7% for high, and ≥8 for very high prevalence of wasting (3). Our analysis of the DHS data confirms previous findings that Latin America has little or no wasting and that Asia has very high prevalences of wasting (3,5–8) (Fig. 2A). Also, as documented previously, Africa, on average, does not have such high levels of wasting as Asia, although some West African countries (Benin, Niger, and Mali in particular) as well as Chad from middle Africa show extremely high levels of wasting, even higher than those found in the Asian countries studied here (Fig. 2B). The prevalence of wasting in these countries is >10% in both urban and rural areas, and the prevalence in Mali is even >20%. Note that the African countries with the highest levels of wasting are not the ones that showed the highest levels of stunting. For instance, Mali and Chad, which have wasting prevalences around 20%, rank in the moderate to high range for stunting. On the other hand, countries of eastern and southern Africa like Madagascar, Zambia, and Malawi, which show the highest prevalences of stunting, have generally low levels of wasting. Niger is one exception, with both very high stunting (in rural areas) and very high wasting in both
urban and rural areas. Worldwide, Mali, Chad, Niger, and Bangladesh (in descending order) have the highest overall levels of wasting.

At the country level, differences in wasting between urban and rural areas are much smaller than the differences in stunting between the two areas, and this is true for all three geographical regions.

**Summary of Global Patterns of Wasting and Stunting and Comments on Their Etiology**

Our findings are consistent with previous descriptions of the worldwide magnitude and geographical distribution of wasting and stunting (3,5–8), although we used
more recent nationally representative datasets than most previous reports. Our analysis supports the general view that moderate levels of stunting and no wasting are found in Latin America, that Asia has the highest levels of both stunting and wasting, and that Africa has a mix of high stunting/moderate wasting, particularly in eastern and southern Africa, and moderate stunting/high wasting in western African countries.

Although we did not specifically look at the issue of the association between wasting and stunting at country or regional level, our data seem to support previous findings from Victoria (5) showing a lack of correlation between wasting and stunting prevalences in Latin America and Africa but evidence that such a correlation exists in Asia. Data have continued to accumulate to support these findings since they were first described, but, to our knowledge, no apparent progress has been made in understanding the differential etiology of wasting and stunting and why they are present in the same populations and individuals in some regions of the world but not in others.

Two main schools of thought have emerged during the 1980s on this issue. The first argues that wasting and stunting are two manifestations of the same phenomenon (9) and that they share a common etiology—that is, deficient nutrient intakes and high rates of infectious diseases. According to this theory, children faced with chronic but moderate nutritional deficiencies first experience a reduction of their rates of linear growth, then stop growing in height if the insults persist, and eventually become wasted as the severity and the duration of the insults increase. This implies that stunting and wasting are a continuum in the manifestation of a common problem, the main difference being its intensity.

The second school of thought contends that, based on biological, epidemiological, and statistical grounds, wasting and stunting clearly represent different processes of malnutrition (10,11). The argument against a common etiology is based on various findings: (a) the lack of correlation between wasting and stunting at the population level, (b) the lack of consistent response (i.e., reductions in wasting or stunting) to changes in specific environmental factors as a result of either secular trends or specific interventions, and (c) differences in age patterns of wasting and stunting. The defenders of this theory agree that wasting and stunting do share the common causes of insufficient energy intakes and infections, but they believe that at least some different causes are involved. Their hypothesis is that overall energy deficits lead to both wasting and stunting but that increasing energy intakes and reducing infectious diseases would lead to reductions in wasting, without necessarily reducing stunting. This is thought to reflect the fact that stunting may be related to other limiting nutrients such as zinc, vitamin A, calcium, or folate. The high levels of stunting without wasting in Latin America and in some countries of East Africa would support this hypothesis.

To our knowledge, no progress has been made since the late 1980s in testing any of these theories at the global level. The excellent review of the phenomenon referred to as the “Asian enigma” (12) provides a fascinating description of the series of cumulative insults that may explain the excessive levels of malnutrition found in South Asia from preconception through to the end of the preschool years. The
article, however, does not differentiate between wasting and stunting and does not address the issue of why some children become stunted, others become wasted, and yet others become both wasted and stunted.

Although there is a clear consensus that both stunting and wasting are associated with poverty, inadequate food intake, and high rates of infections, there is still no explanation for the large variability observed in wasting and stunting prevalences between countries and regions of the developing world. A World Health Organization (WHO) review in 1986 concluded that more research was needed on the natural history of the processes that lead to wasting and stunting and on the effectiveness of interventions to prevent and treat these nutritional deficiencies (10). Fourteen years later, these exact same questions remain unanswered.

AGE TRENDS IN GROWTH FALTERING

This section examines similarities and differences in age patterns of growth among preschool children between regions and countries and between urban and rural areas within countries. The main focus is on describing the age patterns of growth faltering, starting from where children are at birth or soon after compared with the growth standards, how they grow in the first few months of life, the timing and magnitude of peak growth faltering (when it starts, how severe it gets, and at what age), and when and where it stabilizes thereafter. Patterns of both stunting and wasting are described.

Age Trends in Linear Growth Retardation and Stunting

With respect to linear growth patterns, the countries of Latin America included in our analysis can be divided in two groups. The first group, which includes Brazil, Colombia, and the Dominican Republic, shows less severe overall levels of linear growth retardation and also shows patterns of growth in urban areas that are relatively close to the reference standards (mean z scores remain between 0 and −0.5 throughout the first 3 years of life). Figure 3A illustrates the age patterns of mean height-for-age z (HAZ) scores using Brazil as an example of a country from that group. The second group, which is composed of Bolivia, Guatemala, Nicaragua, and Peru (see Fig. 3B and C), shows much more severe growth faltering, generally starting soon after birth (in the second or third month) and deteriorating progressively up to 18 months of age. The lowest mean HAZ score (nadir) for all seven countries of Latin America is at 18 months of age, and this is true for both urban and rural areas. In rural areas, mean HAZ scores at 18 months range from −1.16 for the Dominican Republic to −2.72 for Guatemala and in urban areas from −0.61 to −1.72 for the same two countries (see Table 1). These low levels of HAZ scores translate into high prevalences of stunting at 18 months, with prevalences as high as 74% in rural Guatemala, 56% in rural Peru, and around 45% in rural Bolivia and Nicaragua (Table 1). Even in urban areas, prevalences of stunting at 18 months of age reach 46% in Guatemala and 28% in Bolivia. After 18 months of age, both the mean HAZ scores and the prevalences of stunting tend to stabilize. The HAZ scores stabilize at low levels, especially for
countries in the second group (around $-2$ for rural areas and $-1.5$ for urban areas). For Brazil, Colombia, and the Dominican Republic, the mean HAZ score after 18 months stabilizes at around $-1$ for rural and $-0.5$ for urban areas.

Another important feature of the growth curves observed in Latin America is the fact that in many countries, including some of the better-off countries, low mean HAZ values at 0–1 or 0–2 months of age are observed, suggesting that children are born stunted as a result of intrauterine linear growth retardation. (Although the DHS
<table>
<thead>
<tr>
<th>Countries by region (yr)</th>
<th>Stunting prevalence (%)</th>
<th>Wasting prevalence (%)</th>
<th>HAZ at 0–1 mo</th>
<th>WHZ at 0–1 mo</th>
<th>HAZ at 18 mo</th>
<th>Stunting prevalence at 18 mo (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>U</td>
<td>R</td>
<td>U</td>
<td>R</td>
<td>U</td>
<td>R</td>
</tr>
<tr>
<td><strong>Latin America</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bolivia (1998)</td>
<td>16.0</td>
<td>29.9</td>
<td>1.4</td>
<td>2.7</td>
<td>-0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.19&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Brazil (1996)</td>
<td>7.8</td>
<td>19.0</td>
<td>2.3</td>
<td>2.5</td>
<td>0.04</td>
<td>-0.43&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Colombia (1995)</td>
<td>9.6</td>
<td>14.4</td>
<td>0.9</td>
<td>1.8</td>
<td>-0.22</td>
<td>-0.45&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Dominican Rep. (1996)</td>
<td>7.6</td>
<td>15.7</td>
<td>1.8</td>
<td>1.5</td>
<td>-0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.09&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Guatemala (1995)</td>
<td>31.6</td>
<td>46.7</td>
<td>3.5</td>
<td>4.3</td>
<td>-0.30&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.37&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Nicaragua (1998)</td>
<td>14.5</td>
<td>25.4</td>
<td>2.0</td>
<td>3.7</td>
<td>0.05</td>
<td>-0.19</td>
</tr>
<tr>
<td>Peru (1996)</td>
<td>13.1</td>
<td>33.7</td>
<td>1.0</td>
<td>2.0</td>
<td>-0.17</td>
<td>-0.17</td>
</tr>
<tr>
<td><strong>Africa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>West Africa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benin (1996)</td>
<td>20.0</td>
<td>24.1</td>
<td>13.0</td>
<td>14.0</td>
<td>-0.06&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.01</td>
</tr>
<tr>
<td>Burkina Faso (1992)</td>
<td>19.8</td>
<td>35.8</td>
<td>9.8</td>
<td>13.8</td>
<td>0.31&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.48</td>
</tr>
<tr>
<td>Côte d'Ivoire (1994)</td>
<td>15.3</td>
<td>29.0</td>
<td>7.9</td>
<td>8.0</td>
<td>-0.01</td>
<td>-0.12</td>
</tr>
<tr>
<td>Ghana (1993)</td>
<td>16.1</td>
<td>28.5</td>
<td>8.6</td>
<td>11.6</td>
<td>-0.26&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.15&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Mali (1995)</td>
<td>21.0</td>
<td>31.6</td>
<td>22.1</td>
<td>22.2</td>
<td>0.24</td>
<td>0.02</td>
</tr>
<tr>
<td>Niger (1998)</td>
<td>29.0</td>
<td>40.4</td>
<td>13.2</td>
<td>18.6</td>
<td>0.14&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.10</td>
</tr>
<tr>
<td>Senegal (1992)</td>
<td>12.4</td>
<td>24.8</td>
<td>7.6</td>
<td>10.1</td>
<td>-0.09</td>
<td>-0.12</td>
</tr>
<tr>
<td>Togo (1998)</td>
<td>14.3</td>
<td>24.1</td>
<td>9.0</td>
<td>13.0</td>
<td>-0.15&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.15&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Middle Africa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cameroon (1997)</td>
<td>21.7</td>
<td>31.0</td>
<td>4.4</td>
<td>6.2</td>
<td>0.28&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.09&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Central African Rep. (1994)</td>
<td>26.7</td>
<td>36.0</td>
<td>5.9</td>
<td>8.8</td>
<td>-0.03</td>
<td>-0.09</td>
</tr>
<tr>
<td>Chad (1997)</td>
<td>25.7</td>
<td>35.3</td>
<td>17.0</td>
<td>18.5</td>
<td>0.48</td>
<td>0.62</td>
</tr>
</tbody>
</table>

*continues*
<table>
<thead>
<tr>
<th>Countries by region (yr)</th>
<th>Stunting prevalence (%)</th>
<th>Wasting prevalence (%)</th>
<th>HAZ at 0-1 mo</th>
<th>WHZ at 0-1 mo</th>
<th>HAZ at 18 mo</th>
<th>Stunting prevalence at 18 mo (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>U  R</td>
<td>U  R</td>
<td>U  R</td>
<td>U  R</td>
<td>U  R</td>
<td>U  R</td>
</tr>
<tr>
<td><strong>Eastern and Southern</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Africa</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kenya (1998)</td>
<td>25.1 31.2</td>
<td>5.0 7.4</td>
<td>-0.24&lt;sup&gt;a&lt;/sup&gt; -0.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.67&lt;sup&gt;a&lt;/sup&gt; 0.52&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-1.95 -2.02</td>
<td>50.9 50.6</td>
</tr>
<tr>
<td>Madagascar (1997)</td>
<td>43.1 48.4</td>
<td>5.3 7.4</td>
<td>0.07&lt;sup&gt;a&lt;/sup&gt; -0.55&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.57&lt;sup&gt;a&lt;/sup&gt; 0.77&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-2.60 -2.56</td>
<td>54.4 73.7</td>
</tr>
<tr>
<td>Malawi (1992)</td>
<td>30.4 42.6</td>
<td>2.7 7.0</td>
<td>0.05&lt;sup&gt;a&lt;/sup&gt; -0.59&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.61&lt;sup&gt;a&lt;/sup&gt; 0.68&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-1.89 -2.48</td>
<td>56.3 62.7</td>
</tr>
<tr>
<td>Namibia (1992)</td>
<td>21.5 32.1</td>
<td>7.8 9.3</td>
<td>-0.05&lt;sup&gt;a&lt;/sup&gt; -0.45&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.07&lt;sup&gt;a&lt;/sup&gt; 0.54</td>
<td>-1.37 -1.86</td>
<td>39.5 43.0</td>
</tr>
<tr>
<td>Tanzania (1996)</td>
<td>29.9 40.8</td>
<td>9.8 8.5</td>
<td>0.24 -0.05</td>
<td>-0.81&lt;sup&gt;b&lt;/sup&gt; -0.42&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-2.11 -2.37</td>
<td>57.1 59.9</td>
</tr>
<tr>
<td>Uganda (1995)</td>
<td>21.4 35.6</td>
<td>5.7 5.9</td>
<td>-0.03&lt;sup&gt;a&lt;/sup&gt; -0.35&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.16&lt;sup&gt;a&lt;/sup&gt; 0.39&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-1.53 -2.12</td>
<td>37.7 54.4</td>
</tr>
<tr>
<td>Zambia (1996)</td>
<td>28.1 43.6</td>
<td>4.8 6.6</td>
<td>0.03&lt;sup&gt;a&lt;/sup&gt; -0.28&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>0.46&lt;sup&gt;a&lt;/sup&gt; 0.45&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-1.87 -2.55</td>
<td>48.3 67.3</td>
</tr>
<tr>
<td><strong>Asia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bangladesh (1997)</td>
<td>18.6 29.7</td>
<td>12.8 17.5</td>
<td>-0.69&lt;sup&gt;a,b&lt;/sup&gt; -0.42&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>-0.71&lt;sup&gt;a,b&lt;/sup&gt; -0.70&lt;sup&gt;a,b&lt;/sup&gt;</td>
<td>-1.95 -2.62</td>
<td>51.4 71.0</td>
</tr>
<tr>
<td>Nepal (1996)</td>
<td>33.3 46.1</td>
<td>5.6 11.9</td>
<td>-0.51&lt;sup&gt;a,b&lt;/sup&gt; -0.55&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.08&lt;sup&gt;a&lt;/sup&gt; -0.27&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-2.09 -2.45</td>
<td>54.5 68.8</td>
</tr>
<tr>
<td>Pakistan (1990)</td>
<td>27.6 37.7</td>
<td>8.4 13.0</td>
<td>0.16 -0.39&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.19 -0.46&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-2.1 -2.25</td>
<td>48.2 62.5</td>
</tr>
</tbody>
</table>

HAZ, height-for-age z score; WAZ, weight-for-age z score; WHZ, weight-for-height z score; U, urban; R, rural.

<sup>a</sup>Data are for children 0–2 mo old as opposed to 0–1 mo.

<sup>b</sup>HAZ or WHZ indicate values of less than or equal to −0.25.
datasets do not provide information on length at birth per se, for many countries, the sample sizes were large enough to stratify the analysis by monthly age intervals, so that information was available on the anthropometry of 0- to 1-month-old children. In cases where sample sizes were too small, we analyzed the data for 0- to 2-month-old children. We do not claim that this information is as accurate as having birth length and weight data, but it provides some indication of the levels of growth retardation already detectable during the very early postnatal period.) The mean HAZ scores of infants at 0–1 or 0–2 months of age, as available for the 28 countries studied, are presented in Table 1. In Latin America, infants living in rural areas of Brazil and Colombia have mean HAZ scores of −0.43 and −0.45 during their first month and first 2 months of life, respectively. All countries of the second group show some degree of stunting in early life as well, particularly Guatemala, with mean HAZ scores of −0.37 and −0.30 for rural and urban children, respectively. Guatemala is also the country with the highest overall stunting prevalence of the whole region.

The patterns of linear growth retardation in Africa are similar to those described for Latin America. Deceleration in growth velocity (compared with the reference standards) is observed as early as the first month of life in many countries and especially in rural areas of eastern and southern Africa, where more severe stunting is observed (see, e.g., Fig. 4A and B). Among West African countries (Benin, Cote d’Ivoire, Senegal, and Togo, e.g.), which have lower levels of stunting, linear growth follows the reference standard curve for up to 3 or 4 months after birth, at which age it starts to deviate gradually from the reference curve (see Fig. 4C). For all African countries, the mean HAZ score decreases gradually with age to reach a nadir at around 18 months, as in Latin America. The average HAZ scores at the lowest points are also similar to the values found in Latin America. Central and East Africa have the lowest mean HAZ score at 18 months (−2.85 for Chad and approximately −2.5 for Madagascar, Malawi, and Zambia), whereas in East Africa, the lowest mean HAZ score at 18 months is found in Niger (−2.42) followed by Mali (−2.17). Prevalences of stunting at 18 months are also very high in many countries of middle, eastern, and southern Africa, with prevalences of 60% and more in rural areas of more than half the countries (Table 1).

The differences between urban and rural areas within countries are generally smaller in Africa than Latin America, and urban areas of Africa tend to have higher levels of linear growth faltering than urban centers of Latin America. This is likely to be related to the overall lower levels of urbanization found in Africa (13).

In Africa, low HAZ scores at birth or in the first month (or 2) of life are found only in rural areas of the eastern and southern regions, where five of the seven countries studied have HAZ scores less than or equal to −0.25 (Table 1). Note that these countries are also the ones with the highest overall stunting prevalences among preschoolers in Africa and the ones with the most severe problem of stunting at 18 months of age. In West Africa, only urban areas of Ghana have a low mean HAZ score in the first month of life (−0.26). Many countries of West Africa actually have mean HAZ scores of ≥0 soon after birth. This is also true for urban areas of many countries from middle, eastern, and southern Africa. Thus, overall, intrauterine linear growth retardation may be less prevalent in Africa than in Latin America. However, as in Latin
America, the countries of eastern and southern Africa that have the most severe problems of stunting are also the ones that show evidence that the problem starts in utero, as seen by their high prevalence of low HAZ scores during the early postnatal period. In these countries, mean HAZ scores are below 0 soon after birth, and there is no evidence of any catchup during the first 3–4 months (see Fig. 4A and 4B). The growth curves show a gradual, almost linear fall in mean HAZ score until it reaches a nadir at 18 months.
Although data from only three Asian countries were available for analysis, the growth patterns shown in Fig. 5 clearly illustrate the well-documented severity of the linear growth retardation problem in Asia. Bangladesh and Nepal, in particular, have striking examples of HAZ growth curves that start around \(-0.5\) z score in the first month of life in both urban and rural areas and continue to show gradual deterioration until they reach the very low \(-2.5\) z score around the age of 18 months. Although rural children show almost a linear deterioration with age starting from

**FIG. 5.** Age trends in mean height-for-age z (HAZ) score in Asia, by area of residence. A. Bangladesh 1996. B. Nepal 1996. C. Pakistan 1990–91. (Demographic Health Survey data).
birth, the urban children seem to grow parallel to the reference standards until about 4 months of age, at which time they start experiencing a gradual decline in HAZ scores. The levels of stunting in Asia are comparable with those of eastern and southern Africa, with prevalences at 18 months of 71% in rural areas of Bangladesh, 69% in rural Nepal, and 62% in rural Pakistan. Even in urban areas, more than half the children are stunted at 18 months of age (see Table 1).

**Age Trends in Weight-for-Height z Scores and Wasting**

Latin America, as discussed previously, has low levels of wasting overall. Figure 6 shows that this is also true when looking at patterns of change in weight-for-height z (WHZ) score over the age range between 0 and 36 months. In Latin America, children are often born with rather high WHZ scores, even in countries with high levels of linear growth retardation such as Peru and Guatemala (see Fig. 6A–C). The mean WHZ scores in most countries of Latin America in the first month of life are >0, and many are as high as 0.5. There is some slow deterioration throughout the first year, but this decline brings the WHZ score to the reference population levels (z scores around 0). Contrary to the findings for HAZ score, urban and rural areas have a similar mean WHZ scores throughout the age range.

Positive WHZ scores early in life are also found in most countries of eastern and southern Africa, which have, on average, moderate levels of wasting. In West Africa, however, countries like Benin, Burkina Faso, Mali, and Niger, in particular, have extremely high levels of wasting between 0 and 36 months of age. These countries also show significant wasting starting in the early postnatal period, as seen by their negative mean WHZ score during the first 2 months of life. Mali and Benin, for example, have a mean WHZ score of −0.6 or lower soon after birth (see Fig. 7A and 7B). Although some postnatal catchup is observed, mean WHZ scores drop steeply after the third and fourth month of life to reach levels as low as −1.5 between 9 and 18 months of age. Countries of eastern and southern Africa, on the other hand, have positive WHZ scores during the early postnatal period, but they also show a similar pattern of decline from 3 to 4 months of age until they reach their lowest point, ranging from −0.5 to −1 between 9 and 12 months of age (see Fig. 7C). The nadir for the WHZ score for all African countries is around 9–12 months of age as opposed to 18 months as seen for the HAZ score. Thus, there is evidence of severe wasting in countries of West Africa, starting soon after birth and reaching peak severity between 9 and 12 months of age.

In Asia, again, the patterns of growth faltering in weight for height are striking, especially in Bangladesh and Nepal (Fig. 8). In Bangladesh, infants are born wasted, with an average WHZ score of −0.70 in the first 2 months of life, followed by a slight catchup between 2 and 4 months of age and a consistent deterioration thereafter until a low mean WHZ of −1.4 is reached at 12 months for rural children and −1.1 at 9 months for urban children.

Owing to space limitation, information on the prevalence of diarrhea is not presented here. Our analysis of DHS data, however, shows that the prevalence of diarrhea parallels that of wasting, also peaking as early as between 9 and 18 months of age. It precedes the peak period of stunting by 6–12 months.
Summary of Findings on Age Trends in Growth Faltering and Comparison with Previous Reports

The general consensus from previous reviews of the age patterns of growth is that children in developing countries are of adequate size at birth (10,14), that they grow relatively well in the first 3 months of life, and that they start to deteriorate around their fourth month (1,11,14,15). From there on, their growth velocity is
lower than expected, and they accumulate deficit until 24–36 months old, at which age their growth tends to stabilize (1,11,14,15). Some investigators have also commented that the process of slowing in linear growth might actually start as early as the second or third month of life (1,8,15), but they had little information to support these claims.

Our findings challenge some of these previous observations. First, it is clear that both wasting and stunting are widespread during the early postnatal period and most

probably at birth. Countries with the most severe problems of childhood stunting, for example, show evidence of low mean HAZ scores as early as during the first month of life. Similarly, in countries where wasting is most acute in the first 2 years of life, low WHZ scores are observed during the early postnatal period. Although the DHS datasets do not have anthropometric data at birth, we believe that the low mean WHZ and HAZ values found in some countries soon after birth are a clear indication of a significant IUGR problem. Thus, it appears that the excessive levels of wasting and
stunting found in some countries are probably explained, at least in part, by higher levels of IUGR.

In countries where stunting is found during the early postnatal period, there is generally no evidence of significant catchup during the first 3–4 months of life. In most countries where growth curves start below the reference standards (below 0), the curves show an almost linear decline from the first months of life until they reach their nadir at around 18 months of age for HAZ. For wasting, however, there is some catchup early on, but severe deterioration follows from approximately the fourth month of life.

Our findings show that the patterns of growth faltering in utero, as evidenced by anthropometry during the early postnatal life, follow the same regional patterns as the overall geographical distribution of wasting and stunting. Early linear growth retardation, for instance, is widespread in Latin America, in eastern and southern Africa, and in South Asia. West Africa, on the other hand, shows surprising high levels of wasting during the early postnatal period, similar to those found in South Asia.

The peak period of linear growth retardation is found systematically at 18 months in all countries from the three regions, whereas wasting peaks much earlier, between 9 and 12 months of age. Previous studies suggested that wasting was more prevalent during the second year of life and tended to peak around 24 months of age (8,11). Our findings indicate an earlier onset of wasting, with peak prevalence during the period of transition from predominant breast-feeding to the introduction of complementary foods in the child’s diet. Diarrhea also seems to peak earlier than previously thought and parallels the patterns of wasting.

Another issue that has received little attention in previous reviews concerns the urban/rural differentials in growth patterns. With regard to HAZ scores, urban and rural children generally grow in parallel, with rural children at a lower level from birth or very soon thereafter. This is particularly true in regions with high levels of stunting such as eastern and southern Africa, Asia, and some countries of Latin America. Wasting, on the other hand, does not show such marked differences between urban and rural areas, and in many countries, the two WHZ curves run very close to each other. This is also true for overall differences in wasting between urban and rural areas, which are generally of small magnitude, as well as for differences in diarrhea prevalence (13). In fact, some countries even show higher diarrheal prevalence rates in urban compared with rural areas (13).

It is generally believed that one of the most important nutritional differences between urban and rural areas is household food availability and dietary diversity (16–18). A preliminary analysis of complementary feeding practices in five countries of Latin America (using DHS datasets) shows a clear pattern of more extensive use of animal products (dairy, eggs, meat, fish, and poultry) in urban than rural areas (19). Differences of two- to threefold are found in the percentages of mothers who fed their children animal products during the previous 24 hours in some countries, with larger differences among the younger age groups (6- to 12-month-old children). These results suggest that one of the potential explanations for the widening of the gap in HAZ scores between urban and rural children after 6 months of age may be related
to differences in feeding practices and particularly in the use of animal products. This argument would also support the “limiting nutrient” hypothesis to explain differences in stunting (as described previously) as opposed to differences in energy intake or infectious diseases.

In summary, this review highlights two main points that should be taken into consideration when examining growth patterns. First, it is crucial to have as much information as possible on the status of children at birth and to have monthly data in early postnatal life to understand the nature of growth faltering during this crucial period of accelerated growth. Second, data should always be stratified by urban and rural areas. Pooling together data from children living in such distinct environments tends to veil crucial information about the specific characteristics of their growth patterns. This in turn might lead to the selection of inappropriate program and policy responses.

STUNTING AT BIRTH: A CASE STUDY FROM GUATEMALA

This section explores the intriguing questions of the importance of stunting at birth, as a reflection of IUGR and as a future predictor of growth faltering, and of the potential benefit of nutritional interventions.

Most of the literature on IUGR relies on the measurement of weight. IUGR is usually defined as weight below the 10th centile for gestational age. A further grouping classifies IUGR infants into proportionate [infants with an adequate ponderal index (weight/height$^3$)] or disproportionate (infants with a low ponderal index). Proportionate infants are, in effect, short and round, meaning that they are stunted but not wasted, whereas disproportionate infants are long (less stunted) and wasted. The rationale given for distinguishing between proportionate and disproportionate children is that the etiology and the timing of growth retardation are thought to differ between the groups, as well as the characteristics of the infants at birth, their risk of neonatal morbidity and mortality, and their patterns of growth and development during childhood (20–22). In developing countries, the incidence of proportionate IUGR infants is highly predominant compared with the disproportionate type.

The problem with the proportionality classification is that it is a subcategory of IUGR, and therefore it excludes infants who have adequate weight for gestational age. In countries with high levels of postnatal stunting and no wasting, it is likely that linear growth faltering, as opposed to weight deficits, occurs in utero. With the current definition of IUGR, short chubby infants may not be identified as being growth retarded at birth, although they may also experience adverse outcomes such as severe stunting and related functional consequences during their postnatal life. These infants may also have increased potential for response to nutritional interventions because of their already existing pattern of growth faltering, but they may not be screened as being IUGR if they do not meet the weight cutoff points defined by conventional indicators.

We used data from Guatemala, a country with one of the highest prevalences of stunting in the world, to examine the issue of stunting at birth. First, we used data from five different samples to describe the anthropometric characteristics of
Guatemalan infants at birth, using various definitions of IUGR. Second, we used data from the longitudinal INCAP supplementation trial and its first follow-up (2) to examine the postnatal growth of infants born short and to compare various indicators of IUGR relative to their performance as predictors of risk of future stunting and of benefit from nutritional supplementation.

**Anthropometric Characteristics of Guatemalan Infants at Birth**

Various datasets from Guatemala were used to examine the anthropometric characteristics of Guatemalan infants at birth, two of which were collected specifically for this purpose. Although the datasets are not nationally representative, they include a variety of locations, ethnic backgrounds, and time periods. All data were collected by INCAP, using trained and standardized anthropometrists. A short description of the five datasets is presented below.

1. Urban hospital, Instituto General de Seguro Social (IGSS) (December 1994; n = 1,042). This dataset was collected in a hospital from the Social Security Services of Guatemala City, with the specific purpose of describing anthropometric characteristics of urban Guatemalan children at birth. All infants delivered on weekdays (between Monday and Friday) in the hospital during December 1994 were measured within their first 24 hours of life. Mothers attended at this hospital were all beneficiaries of the Guatemalan Social Services System, and hence at least one member of their family was employed in the formal sector. We do not have any information on the ethnic mix of this population, but it is likely to include a larger proportion of ladino than indigenous women.

2. Urban hospital, San Juan de Dios (January 1995; n = 384). This hospital in the capital city is a public hospital that provides a free service for the population. The data were collected for the purpose described above on weekdays within 24 hours of birth. This hospital is also likely to treat a greater proportion of ladino than indigenous women.

3. Oriente INCAP longitudinal study (1969–77; n = 400). This study is a longitudinal supplementation trial in four rural communities of Guatemala (2). Two villages were randomly assigned to receive a high-energy/high-protein drink (atole), whereas the other two villages received a low-energy/no-protein drink (fresco). Both supplements contained similar amounts of various vitamins. All pregnant and lactating women and children up to 7 years of age were invited to participate on a voluntary basis in the supplementation trial. Weight was measured at birth, but length was measured only at 15 days. Thus, except for birthweight, the information used for this sample is based on anthropometric measurements taken at 15 days of age. The population is predominantly ladina.

4. Oriente longitudinal follow-up 2 (1990–93; n = 409). This study is the second follow-up of the study participants from the original supplementation trial. A first follow-up carried out in 1987–88 collected information on the study subjects during adolescence and early adulthood (2). The data used here are a subsample from the
second follow-up that was initiated in 1990 to document the growth and development of the children of mothers who had been supplemented during the original trial. All children included in the analysis were measured within 72 hours of birth.

5. Quetzaltenango (1985–86; n = 1,664). This dataset is from a research study carried in both urban and rural areas of Quetzaltenango. All newborns were measured within 48 hours of delivery. The population is predominantly indigenous.

The analysis was restricted to children with a gestational age of ≥37 weeks. The indicators used to describe anthropometry at birth were as follows: weight (kg); percent low birthweight (<2,500 kg); IUGR (weight <10th centile for gestational age) (23); length (cm); HAZ scores: stunting (moderate = HAZ less than −1; severe = HAZ less than −2); weight-for-age z (WAZ) scores: underweight (moderate = WAZ less than −1; severe = WAZ less than −2); WHZ scores: wasting (moderate = WHZ less than −1; severe = WHZ less than −2). The WHZ scores could be computed for only approximately one-third of all the infants because the remaining ones were shorter than the minimum length available in the reference standards to compute weight-for-length z scores.

The results are presented in Table 2 for boys and girls combined, because there was no evidence of any differences between the two groups. Low birthweight affected between 9% and 16% of infants in the sample, but IUGR was much more common, affecting more than one-third of all newborn babies. Moderate stunting at birth was even more widespread, affecting more than half of the infants in the original Oriente trial and about 50% in the two hospitals from the capital city. Severe stunting (less than −2 SD) was found in almost 10% of the newborn babies in datasets from the 1980s onward and in 18% of infants in the original Oriente study. Mean HAZ values for all datasets were close to −1, and the whole distribution of HAZ was shifted to the left by approximately 1 SD for all five datasets (not shown). The prevalence of underweight paralleled that of stunting, but at consistently lower levels. Wasting, on the other hand, was much less prevalent, although it could be computed only for a subsample of infants. Thus, the levels of wasting are probably an overestimate of the real prevalence that would be obtained if all the short and (probably) proportional infants were included.

The consistency of the results between the different datasets was unexpected because of the heterogeneity of the samples and the time difference between the surveys. Although some improvement was apparent since the 1970s, the datasets did not indicate further improvement between the mid-1980s and the mid-1990s. Surprisingly, even urban mothers delivering in hospitals were giving birth to infants with significant levels of linear IUGR. Thus, these data show very high levels of shortness and stunting at birth among Guatemalan children, with little evidence of urban/rural differences or of improvement since 1985.

Postnatal Growth of Infants Born Stunted

This section follows up on the issue of stunting at birth and looks at the postnatal growth of infants born with evidence of IUGR. The analysis includes infants born
<table>
<thead>
<tr>
<th>Indicator</th>
<th>Capital city hospital (Social Services)</th>
<th>Capital city hospital (public)</th>
<th>Oriente longitudinal study</th>
<th>Oriente follow-up 2</th>
<th>Quetzaltenango</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight (kg)</td>
<td>2.90 ± 0.39</td>
<td>2.88 ± 0.41</td>
<td>3.08 ± 0.48</td>
<td>2.97 ± 0.41</td>
<td>2.88 ± 0.42</td>
</tr>
<tr>
<td>Birth length (cm)</td>
<td>48.02 ± 1.72</td>
<td>48.10 ± 1.90</td>
<td>49.38 ± 2.33</td>
<td>48.24 ± 1.89</td>
<td>48.57 ± 2.12</td>
</tr>
<tr>
<td>WAZ</td>
<td>0.81 ± 0.86</td>
<td>-0.87 ± 0.94</td>
<td>-0.67 ± 0.92</td>
<td>-0.61 ± 0.92</td>
<td>-0.81 ± 0.95</td>
</tr>
<tr>
<td>HAZ</td>
<td>-1.02 ± 0.77</td>
<td>-0.99 ± 0.85</td>
<td>-1.24 ± 0.98</td>
<td>-0.87 ± 0.84</td>
<td>-0.72 ± 0.94</td>
</tr>
<tr>
<td>Ponderal index (weight/length³)</td>
<td>2.62 ± 0.25</td>
<td>2.58 ± 0.28</td>
<td>2.76 ± 0.32</td>
<td>2.64 ± 0.24</td>
<td>2.51 ± 0.29</td>
</tr>
<tr>
<td>% low birthweight</td>
<td>12.8</td>
<td>15.6</td>
<td>8.8</td>
<td>11.0</td>
<td>16.3</td>
</tr>
<tr>
<td>% IUGR</td>
<td>37.7</td>
<td>35.4</td>
<td>27.8</td>
<td>29.1</td>
<td>36.5</td>
</tr>
<tr>
<td>Moderate stunting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% HAZ &lt; -1</td>
<td>50.7</td>
<td>47.5</td>
<td>57.3</td>
<td>31.1</td>
<td>41.3</td>
</tr>
<tr>
<td>Severe stunting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% HAZ &lt; -2</td>
<td>9.3</td>
<td>10.1</td>
<td>18.5</td>
<td>6.8</td>
<td>9.2</td>
</tr>
<tr>
<td>Moderate underweight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% WAZ &lt; -1</td>
<td>42.4</td>
<td>43.9</td>
<td>36.1</td>
<td>40.1</td>
<td>39.8</td>
</tr>
<tr>
<td>Severe underweight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% WAZ &lt; -2</td>
<td>11.4</td>
<td>11.4</td>
<td>8.6</td>
<td>8.4</td>
<td>7.2</td>
</tr>
<tr>
<td>Moderate wasting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% WHZ &lt; -1</td>
<td>17.7</td>
<td>21.1</td>
<td>-</td>
<td>13.8</td>
<td>36.3</td>
</tr>
<tr>
<td>Severe wasting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% WHZ &lt; -2</td>
<td>1.0</td>
<td>3.3</td>
<td>-</td>
<td>0.0</td>
<td>3.8</td>
</tr>
</tbody>
</table>

HAZ, height-for-age z score; WAZ, weight-for-age z score; IUGR, intra-uterine growth retardation; WHZ, weight-for-height z score.
stunted over the whole range of birthweights; that is, the sample is not restricted to IUGR infants. We also compared stunting at birth with other conventional indicators of IUGR in predicting both the risk of future stunting and the benefit from nutritional supplementation. We used data from the original supplementation trial (1969–77) and from the first follow-up study carried out in 1987–88 to document the status of the study subjects at adolescence and early adulthood (2). The sample includes children with a gestational age of ≥37 weeks and who had birthweight information and anthropometric data at 15 days, 36 months, and in young adulthood (n = 400).

The postnatal linear growth of infants born stunted (defined as length for age at 15 days of less than −1) is compared in Fig. 9 with the growth of infants not stunted at birth and with the WHO/National Center for Health Statistics (NCHS) reference standards. Only children who received the fresco supplement (low energy/no protein) are included. Compared with the reference standards, infants born stunted had a length deficit at 15 days of 4.3 cm compared with a deficit of only 0.7 cm among the nonstunted group. There was no evidence of catchup growth among stunted children, who tended to follow their growth channel during their first 3 years of life. The nonstunted children, on the other hand, were born with almost normal length, but their growth started to deviate from the reference standards as early as the first 3 months. By 6 months of age, the deficit in length among this group was already 3.24 cm, about four times greater than it was at 15 days. The deficit compared with the reference standards continued to widen for both groups of children until it reached 12.4 cm for the group stunted at birth and 9.7 cm for the group not stunted at birth. The gap between the two groups, however, remained constant from approximately 6 months to 3 years of age. The initial gap (at 15 days) was 3.6 cm, but it had already narrowed to 2.7 cm by 3 months of age, and it remained at this level throughout the first 3 months of life.

Thus, of the total 12.4-cm length deficit at 36 months of age among children who were born stunted, one-third (34%; 4.3 cm) was due to a deficit already acquired at birth as a result of IUGR. Among the nonstunted infants, the deficit at birth explained only about 7% of their 9.7-cm deficit at 36 months of age. Thus, it seems

![Fig. 9. Linear growth of Guatemalan children compared with the National Center for Health Statistics reference standards, by stunting at 15 days postpartum.](image-url)
like at least some of the "excessive" linear growth retardation found among Guatemalan children reflects linear growth faltering in utero. Because these children are raised in the same environment that led them to be stunted at birth, they do not tend to catch up postnatally, and thus the deficit acquired by the time of birth is not reversed. Postnatal growth is also responsible for a large proportion of the total deficit acquired by 3 years of age, as seen from the pattern of growth of the infants not stunted at birth. As documented in a previous report, the slowing down in growth (weight, in that case) among children from the Oriente communities was found to occur as early as the first 3 months of life, and growth faltering during this period accounted for up to 19% of the deficit acquired by the age of 3 years (24). An additional 19% of the total deficit at 3 years was accounted for by failure to thrive between 3 and 6 months of age and yet another 25% by growth faltering between 6 and 9 months of age. Thus, by 6 months of age, these infants had already accumulated 38% of their total weight deficit at 3 years of age, and by 9 months, they had accumulated >60% of this deficit (24).

Martorell and collaborators (25) also analyzed the contribution of prenatal and postnatal growth to stunting at 3 years of age in this population, using IUGR definitions based on birthweight. They show a gradual decrease in the contribution of the prenatal component to length at 3 years of age as birthweight increased. Because of differences in indicators and in the methodology used, the results of this study are not directly comparable with our findings. These investigators also used a different definition of catchup growth, which was based on the comparison of growth rates between IUGR children and a group of non-IUGR children as opposed to comparisons with reference standards. The results of the two studies, however, concur in showing that the contribution of prenatal growth faltering is large among IUGR infants, irrespective of the definition of IUGR, and that in spite of some degree of catchup during postnatal life (shown in the study of Martorell et al.), IUGR children remain smaller throughout childhood and through adult life.

Stunting at Birth as a Predictor of the Risk of Stunting at 3 Years of Age and of Short Stature in Adult Life

Stunting at birth was compared to low birthweight and to the conventional definition of IUGR (low weight for gestational age) to determine which of the three indicators was more effective in predicting the risk of stunting at 3 years of age and of short stature at adulthood. Table 3 presents the sensitivity, specificity, positive predictive value, and percentages of false positives, false negatives, and misclassified children of the three indicators in predicting length at 3 years of age and stature in adult life. For the purpose of screening infants at birth with the intention of intervening in a timely manner to prevent further deterioration, sensitivity (the ability of the indicator to correctly identify infants at risk of poor growth) is more important than specificity (the ability of the indicator to correctly identify those who are not at risk). Thus, for this purpose, the indicator of highest sensitivity is the most desirable. Higher sensitivity, however, naturally results in lower specificity, which then causes greater
TABLE 3. Comparison of three indicators of intra-uterine growth retardation to predict risk of stunting at 3 years of age and of low stature in adult life (Institute of Nutrition of Central America and Panama longitudinal trial, 1969–77)

<table>
<thead>
<tr>
<th>Outcome/indicator</th>
<th>Sensitivity (%)</th>
<th>Positive Specificity (%)</th>
<th>False predictive value (%)</th>
<th>False positive (%)</th>
<th>False negative (%)</th>
<th>Misclassification (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length at 3 yr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW</td>
<td>12</td>
<td>96</td>
<td>81</td>
<td>15</td>
<td>51</td>
<td>52</td>
</tr>
<tr>
<td>IUGR</td>
<td>35</td>
<td>85</td>
<td>76</td>
<td>6</td>
<td>38</td>
<td>44</td>
</tr>
<tr>
<td>LAZ &lt;-1</td>
<td>66</td>
<td>59</td>
<td>69</td>
<td>17</td>
<td>19</td>
<td>36</td>
</tr>
<tr>
<td>Stature as adult (males)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW</td>
<td>14</td>
<td>97</td>
<td>85</td>
<td>1</td>
<td>43</td>
<td>44</td>
</tr>
<tr>
<td>IUGR</td>
<td>41</td>
<td>80</td>
<td>67</td>
<td>10</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>LAZ &lt;-1</td>
<td>60</td>
<td>59</td>
<td>64</td>
<td>18</td>
<td>22</td>
<td>40</td>
</tr>
<tr>
<td>Stature as adult (females)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBW</td>
<td>11</td>
<td>98</td>
<td>87</td>
<td>1</td>
<td>45</td>
<td>46</td>
</tr>
<tr>
<td>IUGR</td>
<td>26</td>
<td>85</td>
<td>64</td>
<td>1</td>
<td>37</td>
<td>38</td>
</tr>
<tr>
<td>LAZ &lt;-1</td>
<td>67</td>
<td>50</td>
<td>57</td>
<td>25</td>
<td>17</td>
<td>42</td>
</tr>
</tbody>
</table>

IUGR, intra-uterine growth retardation (weight <10th centile of weight for gestational age); LAZ, length-for-age z score; LBW, low birthweight; Sensitivity, percentage of all stunted children at 3 yr of age and in adult life, respectively, who were correctly identified as at risk by the indicator; Specificity, percentage of all non-stunted children at 3 yr of age and in adult life, respectively, who were correctly identified as not being at risk by the indicator; Positive predictive value, percentage of all children identified as at risk by the indicator, who are actually stunted at 3 yr of age or short in adult life; False positive, percentage of all children who were incorrectly classified as being at risk; False negative, percentage of children who were incorrectly classified as not being at risk.

leakage of the intervention to infants who may not be at risk (false positives). This in turn reduces the cost-effectiveness of the intervention. In spite of these caveats, in the case of IUGR, it is still more important to maximize sensitivity to ensure that as many of the at-risk children as possible will be identified correctly by the indicator and will be eligible to receive timely intervention.

Of the three indicators, stunting at birth had the highest sensitivity and the smallest percentage of false negatives (at-risk children who would go undetected) at all ages and for both boys and girls (Table 3). The difference in sensitivity between indicators was large. Low birthweight had a very low sensitivity (ranging from 11% to 14%) and IUGR had sensitivities between 26% and 41%. As a correlate, these two indicators had higher specificities than stunting at birth. Overall, however, stunting at birth was the best indicator to correctly identify infants with increased risk of stunting at 3 years of age and of short stature in adult life, because it had a high sensitivity and yielded a small proportion of false negatives.

The next question, then, which is addressed in the following section, is whether or not these infants, once identified as short at birth and at risk of future stunting, have the potential to catch up during their postnatal life if a nutritional intervention is provided.
Stunting at Birth as a Predictor of Differential Response to Energy/Protein Supplementation

Figure 10 is similar to Fig. 9, but it contains two additional growth curves: the growth curves of infants who received the protein/energy supplement (atole) according to whether they were stunted or not at birth. Mothers received the supplements during lactation, but the supplement was made available to infants only after their third month of life.

The growth curves show that stunted infants who received the atole supplement responded to the intervention, starting from the age of about 9 months. From that age onward, their growth curve started to deviate from the curve of short children who received the fresco supplement and remained slightly steeper until they reached 3 years of age. At that age, the stunted-at-birth infants who received atole were approximately the same length as children from the fresco group who had normal length at birth. Thus, by 3 years of age, the atole supplement had reversed the “excess” deficit (of approximately 3 cm) found among children who had suffered intrauterine linear growth retardation compared with those who were born of adequate length. The atole supplement, on the other hand, had little impact on the growth of children who were born of adequate length. The size of the difference in length at 3 years of age between the atole and fresco groups, adjusted for various socioeconomic and maternal characteristics, was 3.1 cm for the infants stunted at birth compared with only 1 cm for infants of normal length at birth. This indicates a differential response to the supplementation; that is, that infants who suffered linear growth retardation in utero responded more to the supplementation than those who did not. This is illustrated in Fig. 11, which presents adjusted mean length at 3 years of age, by supplementation group and by length at birth status.

Interestingly, this differential benefit also remained into adulthood, as seen in Fig. 12. The same pattern of greater benefit from the atole supplement among subjects who were stunted at birth was observed on attained height as an adult. Even the magnitude of the difference between groups remained at the level it was at 3 years of age.

FIG. 10. Linear growth of Guatemalan children compared with the National Center for Health Statistics reference standards, by stunting at 15 days and supplement type.
For instance, girls stunted at birth who received the _atole_ supplement were 3.5 cm taller as adults than those who received the _fresco_ supplement, whereas among the nonstunted girls, the difference in height was only 0.94 cm. The findings in boys were similar. Thus, it appears that not only could the nutrition intervention during early childhood help reverse the effects of intrauterine linear growth retardation, but the beneficial effects also persisted into adult life.

In summary, in this severely stunted population, length at birth performed better than weight-based indicators to predict the risk of stunting at 3 years of age and of...
short stature by adulthood. Length at birth was also a good predictor of response to supplementation. The findings also highlight the fact that sustained protein/energy supplementation during the period of greatest vulnerability (0–3 years of age) was effective in reversing the growth faltering experienced by some Guatemalan infants during their prenatal life. Postnatal supplementation, however, corrected only about one-quarter (3 cm) of the total length deficit (12 cm) that Guatemalan children have when they reach their third birthday. Supplementation did not significantly improve the growth of infants who had not suffered from IUGR.

CONCLUSIONS

Considerable advances have been made in the last few decades in understanding the regional distribution and the age patterns of wasting and stunting. Our study corroborates the findings from previous reviews that Latin America has moderate levels of stunting but no wasting, that Asia has the highest levels of both stunting and wasting, and that Africa is split between countries with moderate stunting and high wasting (West Africa) and those with high stunting and moderate wasting (East and South Africa). The reason for these regional differences, however, is still poorly understood. Even the intriguing question of whether or not wasting and stunting share a unique, common etiology has not been specifically addressed in the recent past. Some researchers have provided a convincing explanation for the excessively high levels of malnutrition found in South Asia, emphasizing the issue of women’s status and its consequences through the life cycle (12). To our knowledge, however, no one has addressed the question of why some children in specific regions of the world become stunted, while others living in apparently similarly deprived environments become wasted and yet others develop both wasting and stunting. The stagnation in this area will prevent progress in identifying and implementing effective interventions to address the specific malnutrition problems faced by different countries and regions of the developing world.

This review challenges some of the previous assumptions related to the age patterns of malnutrition. First, it provides evidence that both wasting and stunting are prevalent during the early postnatal period (and probably at birth) and especially so in countries with the highest levels of childhood wasting and stunting, respectively. It appears that at least a proportion of the excessive levels of wasting and stunting found in some countries is explained by greater levels of IUGR.

Second, the hypothesis that growth during the first 3–4 months of life is generally adequate and comparable with the reference standards was also rejected in this review. Although this is true for some of the relatively better-off countries, it is definitely not the case in countries with the most severe malnutrition problems of either wasting or stunting. Significant growth faltering occurs in the very first months of postnatal life in these countries, and there is no evidence of any catchup relative to the reference standards before 24 months of age.

Another point highlighted by this review was the timing of peak wasting and stunting. Whereas previous research suggested that wasting was at its peak during the
second year of life, around 18–24 months, our findings indicate that wasting peaks at a much earlier age, even during the first year in many countries (between 9 and 18 months). Diarrhea also peaks during this period, which is earlier than most reviews suggest, whereas stunting peaks systematically at 18 months of age in all the countries studied.

The case study from Guatemala provides a convincing argument for the magnitude of the problem of stunting at birth in some countries. It also emphasizes the superiority of stunting at birth as an indicator of the risk of future stunting in a severely stunted population compared with the conventional weight-based indicators of IUGR. Stunting at birth also performed well as an indicator of the potential to respond to a nutrition intervention. There is extensive literature on the functional consequences of IUGR as defined by weight for gestational age. Little is known, however, about the growth patterns and functional consequences of being stunted during the prenatal period, in cases where the stunting is not detectable by a weight indicator. Clearly, there is significant overlap between the weight- and length-based indicators of IUGR, but, at least in Guatemala, the two indicators were not highly correlated. This indicates that they tended to capture different children. This also explains why they performed differently as predictors of risk and benefit.

Thus, we conclude that efforts should be made to develop screening and targeting indicators that address the specific nutritional problem found in a particular context. Although birthweight and weight for gestational age may be sensitive indicators for detecting malnutrition in wasted or both stunted and wasted populations, they may not be the most appropriate in populations experiencing mainly linear growth retardation.

Finally, the contribution of IUGR to overall malnutrition levels in Guatemala was large. Approximately one-third of the deficit in length observed at 3 years of age among infants stunted at birth was a reflection of linear growth faltering in utero. Continued protein/energy supplementation throughout the first 3 years of life was highly effective in reversing the deficit acquired in utero among infants born stunted but had surprisingly little effect on preventing growth faltering among infants not stunted at birth. Overall, the magnitude of the growth deficit among this latter group at 3 years of age was approximately 10 cm, and supplementation during the preschool years did little to prevent it. On the other hand, among those born stunted, the reversal of the fetal insult was achieved and persisted through to adult life. Thus, girls who were stunted at birth were approximately 3 cm taller by the time they were adults if they had received supplementation during the preschool years than if they had not received it. They were also of similar stature to those who were born with adequate length, irrespective of whether they had or had not received supplementation.

Traditionally, nutrition interventions have been compartmentalized to target one of the three key periods of a child’s early life: fetal life (pregnancy), early postnatal life (lactation), and the complementary feeding and preschool period. It is now becoming evident that the focus should be shifted to a life cycle and an intergenerational approach, which considers the different periods and generations as a continuum rather than as separate independent entities. Discussions about resource allocation for nutrition programs and policies should not focus on identifying the one period that will
offer the greatest measurable impact but rather should concentrate on the question of how to achieve consistent nutritional improvements throughout the life cycle and from one generation to the next. The evidence suggesting a large contribution of IUGR to overall childhood malnutrition, especially in countries with the most severe malnutrition problems, is yet another reminder of the importance of intervening before the damage is too pronounced to be reversed.

REFERENCES

DISCUSSION

Dr. Koletzko: Your impressive results with supplementation clearly show that part of the growth deficit was related to substrate availability. However, I’m puzzled by your figure of 50% stunting in the population, and I wonder whether the definition, −1 SD below the NCHS centiles, is adequate. Did you take into account the height of the parents? How many of those were more than 1 SD below the US reference of height? If part of the deficit reflects the growth potential of these children, it would also explain why the “stunting,” as you called it, was such a good predictor of future height and later stunting.

Dr. Ruel: The definition of stunting of less than −1 SD was only at birth. For a 3-year-old child, I was using the regular −2 SD definition. As stunting is a cumulative process, you can imagine that if they are −1 SD at birth, they will continue to deteriorate and will reach −2 SD quite soon. I admit this is not a conventional cutoff point, but the logic is that by 3 months of age, 20% of the infants are already −2 SD below the mean in this population, and prevalences increase linearly with age until 36 months of age. So, I gave a generous initial cutoff point and called it “stunting at birth.” As for the parents, maternal height was controlled for in the multivariate analysis—the adjusted means I showed controlled for mother’s height, which is always a very strong determinant of child nutritional status.

Dr. Arato: From what age do you suggest giving supplementary feeds to these children in developing countries? Do you oppose exclusive breast-feeding in these patients?

Dr. Ruel: These children were probably not exclusively breast-fed. In Guatemala, there is much use of sugar water and all sorts of teas during the period of supposedly exclusive breast-feeding. I am not providing any recommendations about supplementation. What I’m saying is that we need to find why those children are being breast-fed inadequately, and we need to intervene as soon after birth as possible to improve the situation, which does not necessarily mean by giving supplements. The particular study I described was carried out at the end of the 1960s, and a supplement was given from 4 months onward, which is earlier than would be done today. I’m certainly not recommending that supplements should be given that early, nor would I bypass the current recommendations for exclusive breast-feeding. Also, I don’t think that Guatemalan mothers are particularly malnourished—they are certainly not very skinny like in parts of Asia, where breast-feeding is likely to be inadequate because the mothers are so malnourished. In Guatemala, the problem is more likely to lie in the harm done by all the other things the mothers give their babies and in diarrheal
disease, which starts quite early. These children also have a bad start, with a high incidence of IUGR.

_Dr. Amigo:_ In studies we did in Chile, we found that short length at birth was the best predictor of stunting at 6 years of age. I believe that length measurement will have to become a standard public health procedure. The problem is the standardization of the measurements. This is a research problem and a challenge, but I'm sure length measurements must be included in our standard assessments.

_Dr. Ruel:_ I agree with that. I only presented research data carried out by trained anthropometrists, with attention to standardization. We did compare the measurements taken by our research anthropometrists with those done routinely in the hospital, and there was an amazingly large difference. The measurements done in the hospital totally underestimated the problem: They showed much less apparent stunting because there was a systematic error that exaggerated the length measurement. So, I agree: This is a real problem, and it's not clear at present what can be done about it. I'm not suggesting that these measurements should be done everywhere in baby clinics or by midwives, but what I am saying is that if we know there is a problem of stunting at birth, this should motivate us to do something to detect it. In many countries, people haven't begun to look at the 0- to 6-month-old children in near detail, and they certainly haven't looked at length at birth or at growth during the first few months. That's what the DHS datasets allow us to do. But we need even larger sample sizes and longitudinal studies, if possible, to document the problem better.

_Dr. Adair:_ In our study in the Philippines, where we were measuring this large cohort of children every 2 months, we found very dramatic differences in early postnatal growth depending on proportionality at birth. The children who were relatively long but underweight had very rapid weight gain velocities in the first 4 months and then they leveled off; in the long run, those children had greater potential for catchup growth throughout the first 2 years and then later. The children who were proportionate at birth tended to stay small. I wonder whether in the Guatemalan study, you were able to look at a differential responsiveness to supplementation in relation to whether there was proportionate or disproportionate intrauterine growth. I was struck by the fact that most of your growth retardation is proportionate, which is a different picture from in the Philippines where there are more children who are underweight for length.

_Dr. Ruel:_ I am not surprised by what you say. In Asia, it seems as though there are both stunting and wasting, before and after birth, so you will have more disproportionate children. In our study, we had only six children with disproportionate growth! With this small number, we couldn't analyze differences in their growth pattern. In Guatemala, no matter where you look, you never find much wasting.

_Dr. Stoltzfus:_ It was really impressive that you were able to show that stunting at birth is so much more predictive of later height growth than low birthweight or IUGR. I wonder whether that is because you are using length to predict length rather than weight to predict length. Perhaps that is more likely than your underlying assumption that the stunting reflects something happening in gestation. For example, if you did the same analyses to determine whether stunting at 1 year predicted later stunting, would there be similarly high correlations?

_Dr. Ruel:_ Some colleagues and I did this in an article published a few years ago (1). We were looking, from a public health point of view, at the times when we can measure children, which are the ages when we see them at primary health clinics. Measurements at 3 and 6 months showed that stunting at these early ages predicts
stunting at later ages, such as 3 years and even adulthood. But that was my point: Why use an indicator of IUGR based on weight in a country that has such severe stunting? If you need a screening indicator, you can screen by length measurement either at birth or at 3 months, if this is the first time a child has contact with the clinic, or even at 6 months. It is still more accurate in predicting future stunting than using weight.

**Dr. Pelletier:** I am intrigued by your results on the response to supplementation. It seems to me you have good news and bad news, and mostly bad news! The children stunted at birth who received Atole—the increased energy and protein supplement—seem to be able to catch up, at least to the level of those who were not stunted at birth and who received Fresco. That is the good news. But the catchup is quite small—only about 3 cm, I believe. On the other hand, your results suggest that you could not prevent stunting among a cohort of children who were not stunted at birth by giving them Atole. This suggests that we need to do many other things to the environment and nutrition of these children to prevent stunting, even in those born in good shape.

**Dr. Ruel:** This is exactly how I interpreted the results. I have been looking at these data for a long time, and so have a lot of other people, and I think this was the most negative finding we have seen. In fact, it is even worse, because the Oriente study not only provided supplements but also provided health care services. So, even the combination of food plus health services did not seem to have any effect on the children who were not stunted at birth.

**Dr. Martorell:** Maybe we should be asking at what age children are most responsive to nutrition interventions, whether it be supplementation or food fortification or nutrition education. I think this is an important question. What we found with Guatemalan data was that children were responsive to these supplements in the first 3 years of life, approximately (2), but between 3 and 7 years of age, there was no evidence of an effect in any subgroup. We should be asking why that should be. There are several possible reasons. One is that by about 3 years of age, the prevalence of diarrhea is quite low. From 3 to 7 years, the z scores are fairly flat, as you have implied; in other words, the children are not catching up relative to the NCHS reference data, but they are not falling behind either, which means they have average growth rates. So, at these ages, they are growing well and they are not responsive to supplements. The lesson from all this is that if you are going to improve growth, you have to intervene early, and in this population, this means under 3 years.

I have another comment on the impact of supplements. When I looked at the total population in the Atole and the Fresco villages, at the start of the study, the prevalence of very stunted children—more than 3 SD below the mean—was about 50% in both groups of villages. By 1977, when the study ended, the level of severe stunting in the Fresco villages was about the same, around 40–50%, but in the Atole villages, it had fallen to about 20–25%. So, that’s another way of looking at the same data: The prevalence of severe stunting had declined by half. That’s a more positive presentation.

**Dr. Grummer-Strawn:** Your analyses of the timing of linear growth retardation obviously depend on what you are comparing the children with. Implicitly, here you compared them with the Fels children in Ohio. It has been suggested that that sample may have been biased, particularly in length, the children being too long; that would change your assessment of how stunted these children are. I wonder if you have given any thought to comparing them against other references, such as the
Euro-Growth reference, for example, or the WHO breast-fed set, just to see how much variation there is in the estimates.

**Dr. Ruel:** That would be worth doing, especially as the levels of stunting we document in Guatemala always seem so unreal. I have worked extensively in Guatemala and could never understand why children are so stunted, so much more than in many equally poor or even much poorer countries of Africa, for example. I have not found any answer to this question, but I wonder how much changing the reference standards would affect the finding. Dr. Ramakrishnan, have you started to do that?

**Dr. Ramakrishnan:** We haven’t, but it is a very good suggestion. In relation to your last remark, sometimes we may be looking at an intergenerational cycle of deprivation, which may have been going on for a longer time in certain subpopulations of Central America than it has in Africa, where things were not that bad until a couple of hundred years ago. Maybe an anthropologist could look into that.

**Dr. Ruel:** The data that we used were collected between 1969 and 1977. At a national level, more recent DHS studies show that there has been some improvement in Guatemala, with a reduction in stunting. So, our data show the growth patterns of children stunted at birth. The level of stunting found then may be more severe than those found 20 years later. However, the data from the two hospitals in the capital city show that the levels of stunting at birth were still very high in 1995.

**Dr. Frongillo:** I’m not sure that I see any added value in using a reference in this kind of analysis, because what you are really trying to show is where the children end up, given the way they start off. You don’t really need a reference to do that. You may get a clearer answer by thinking through what you are really trying to investigate, without the distraction of a reference.

**Dr. Ruel:** The reference is used only to classify children as being stunted or not; it is not used in the modeling of HAZ scores.

**Dr. Frongillo:** I was thinking about the sensitivity and specificity analysis you presented, which were using the reference data. I wonder if that gets in the way when you are trying to assess what it means to be smaller or larger in the context of Guatemala.

**Dr. Ulijaszek:** Just a comment on intergenerational effects. Michael Golden in the United Kingdom (3) has suggested that within these cycles of deprivation, you may have cycles of fetal imprinting, and it may take many generations before this process can be reversed.

**Dr. Stoltzfus:** What about the genetic experiments crossing big horses with little horses, where the birth size of the baby horses reflects the size of the mother? Perhaps the same concept of “maternal container” size operates in Guatemala, given the shortness of Guatemalan women. Maybe one reason for children being stunted at birth is that they were in a physically restricted environment; when they get out of that, and especially when they are provided with Atole, they quickly catch up to their genetic potential.

**Dr. Ruel:** In which case, maternal shortness should predict benefit from Atole, which it didn’t. I tested interactions with all the different factors that were included as main effects, including maternal height, and it was not found to be a predictor of response to supplementation.

**Dr. Stoltzfus:** But maybe it would if you took size at birth out of the model: Maternal height is much easier to measure than birth length. Another thing is that it might be easier to measure some other linear variable rather than birth length, such
as the length of the femur or any other part of the length component that is easier to measure than the whole infant.

Dr. Ruel: I am not sure how the length of the femur is related to crown–heel length, but it is probably a good suggestion to make the measurement simpler.

Dr. Martorell: Arm length is highly correlated with total length; for example, at 15 days, the correlation between arm length and total body length is 0.9 or so. Arm length is easy to measure.

Dr. Lejarraga: In relation to the experiments on big and little horses, does anybody know the mechanisms underlying in this relation? Is it heart rate, blood flow in the placenta, maternal size per se, or what?

Dr. Brunser: I can comment on that. About 25 years ago, some studies were done in the United Kingdom on sheep, in which they implanted additional eggs into the uterine cavity of pregnant sheep (4,5). The result was that the baby sheep were of smaller size than the offspring of ewes that carried a normal number of fetuses. The other thing they did was to restrict the size of the uterine cavity by suturing the uterus. The result was that the cavity could expand to a certain size and no more. Again, the result was that the baby sheep were much smaller than would be expected in a normal sheep whose uterus expanded normally. In a study done by Muzzo and Zvaighaft (6) at the Institute of Nutrition, also around 25 years ago, they found that when female rats were malnourished, the uterus of the female offspring had a reduced number of estrogen receptors. This continued into adulthood. These second-generation rats had baby rats that were much smaller in size because their uterus, when they became pregnant, did not grow to normal size. So, one factor limiting the size of the offspring appears to be the capacity of the uterus to expand. Small women tend to have small uteri, so this is probably one explanation for the results that Dr. Ruel has just presented.

Another explanation for some of the data is the high incidence of infection in Guatemala. I went to Oriente in 1978–79, and you didn’t have to take cultures to detect environmental contamination; you just had to jump around it to avoid soiling your shoes. When you have such conditions, the children are likely to become infected even when they are in the uterus. Children may gain weight for about 2–3 months, and probably maternal milk will provide some protection; but the system breaks down when sugar water or teas are given, and then you allow the massive contamination of the environment to gain access to the intestine, with ensuing diarrhea and malnutrition.

REFERENCES