The Importance of Infections and Environmental Factors as Possible Determinants of Growth Retardation in Children

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In this chapter, we examine patterns of children’s linear growth in Nepal and in Bangladesh. We then present an analysis of different variables associated with slowed linear growth; these include the child’s sex, the place where the child lives, the season, the economic status of the household, the ethnic group to which the child belongs, and illnesses to which the child is exposed. We finally consider the relative importance of different factors in precipitating slowed linear growth.

Our review draws on a number of studies of children’s growth and nutritional status undertaken in the Kingdom of Nepal between 1975 and 1985. Most of these studies were undertaken by or in partnership with His Majesty’s Government of Nepal. They are summarized in Tables 1 and 2.

PATTERNS OF LINEAR GROWTH IN CHILDHOOD

Adult Nepalis are short in stature. One finding from the first round of the Kosi Hill Area Rural Development Programme (KCHARDEP) Impact Studies is that in the seven sites studied, the mean height of adult men is 160 cm and that of adult women is 148 cm. The same studies reveal that adults from poorer families tend to be shorter than those from wealthier families (9,10).

The slowed linear growth of Nepalese children is noticeable even in infancy. Figures 1 and 2 show the mean values of children’s weights and heights at different ages. The values are averaged from the data collected in Chuliban over 2.5 years.
<table>
<thead>
<tr>
<th>Study title (ref.)</th>
<th>Date (year)</th>
<th>Responsible agency</th>
<th>Purpose of study</th>
<th>Location</th>
<th>Details of sample</th>
<th>Number of children</th>
<th>Ages (months)</th>
<th>Season</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nepal Nutrition Survey (1)</td>
<td>1975</td>
<td>HMGN, CDC</td>
<td>Description of national situation</td>
<td>Nationwide</td>
<td>Random two-stage cluster</td>
<td>6,500</td>
<td>0–72</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Nepal Nutrition Survey (1)</td>
<td>1975</td>
<td>HMGN, CDC</td>
<td>Comparison group</td>
<td>Kathmandu Patan</td>
<td>Children from better-off families</td>
<td>486</td>
<td>0–72</td>
<td>?</td>
<td>Families could afford to provide adequate nutrition for their children</td>
</tr>
<tr>
<td>Surkhet District Nutrition Survey (2)</td>
<td>1977</td>
<td>SCF with HMGN</td>
<td>Description of district situation</td>
<td>Surkhet District (W. Nepal)</td>
<td>Random two-stage cluster</td>
<td>1,000</td>
<td>0–59</td>
<td>Premonsoon</td>
<td></td>
</tr>
<tr>
<td>Baglung Nutrition Survey (3)</td>
<td>1979</td>
<td>SCF</td>
<td>Association of nutrition with HH wealth</td>
<td>Baglung District (W. Nepal)</td>
<td>All children in all HH from three Panchayats</td>
<td>1,490</td>
<td>0–71</td>
<td>?</td>
<td>Sufficiency of HH food production and milk buffalo as wealth indices</td>
</tr>
<tr>
<td>KCHARDEP Baseline Survey (4)</td>
<td>1979</td>
<td>HMGN, (KHCARDEP), SCF, BNMT</td>
<td>Base line for development program</td>
<td>Dhankuta Sankhuasabha (E. Nepal)</td>
<td>Random two-stage; 40 sites; 457 HHs</td>
<td>907</td>
<td>0–95</td>
<td>Early monsoon</td>
<td>Total land area cultivated as HH wealth index</td>
</tr>
</tbody>
</table>

*All abbreviations are defined in Table 2.*
### TABLE 2. Longitudinal studies of children's nutritional status discussed in this chapter*

<table>
<thead>
<tr>
<th>Study title (ref.)</th>
<th>Dates</th>
<th>Responsible agency</th>
<th>Purpose of study</th>
<th>Location</th>
<th>Details of sample</th>
<th>Number of children (midsurvey)</th>
<th>Ages (months)</th>
<th>Frequency of data collection</th>
<th>Extra data collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chuliban longitudinal study (5–7)</td>
<td>1977–82</td>
<td>SCF HMGN ODA</td>
<td>Influences of illness and season on child growth</td>
<td>Chuliban village, Dhankuta District</td>
<td>Children seen in a home visiting program</td>
<td>Approx. 200</td>
<td>0–71</td>
<td>Average 7 per year</td>
<td>Illness episodes; some detailed studies of individual HHs</td>
</tr>
<tr>
<td>KHRDEP Impact Studies (KIS) (9–10)</td>
<td>1980–85</td>
<td>HMGN (KHRDEP) ODA SCF</td>
<td>Impact of a rural development program</td>
<td>Dhankuta, Sankhua-sabha, Terathum, Bhojpur Districts</td>
<td>Stratified random at 7 sites; 760 HH</td>
<td>Approx. 450</td>
<td>0–71</td>
<td>2–3 per year</td>
<td>Socioeconomic; service use</td>
</tr>
</tbody>
</table>

*Notes for Tables 1 and 2: HH, household; KHRDEP, Kosi Hill Area Rural Development Programme; HMGN, His Majesty’s Government of Nepal; CDC, Center for Disease Control, Atlanta, Georgia, U.S.A.; SCF, Save the Children Fund, U.K. (works under an agreement with HMGN Health Ministry); BNMT, Britain Nepal Medical Trust (works under an agreement with HMGN Health Ministry); ODA, British Government Overseas Development Administration.
The growth patterns of children studied in the Chuliban Longitudinal Study (6) and the KARDEP Impact Studies (8,9) have been compared with the growth of children in the National Center for Health Statistics reference population (NCHS reference) (11). The average linear growth rate of the children (males and females combined) has been estimated from differences in their mean lengths or heights at different ages. These values, expressed as monthly linear growth rates (millimeter per month), are compared with the monthly growth rates derived from the NCHS figures. Weight gain figures, produced in a similar fashion, have been expressed in grams per month. They are compared with the reference-derived weight gain figures (Figs. 1 and 2).

Estimates for monthly linear growth rates and weight gains of children in the Chuliban longitudinal study and linear growth rates of children in the KARDEP study are shown in Table 3.

The weight gain of children in Chuliban is far lower than reference values between the ages of 3 and 24 months; it increases between 24 and 36 months and then starts to fall off again between 36 and 72 months. Length increments are less than expected from birth onwards. The deficit is most marked between the ages
FIG. 2. Chuliban Longitudinal Study: mean lengths or heights of children compared to the NCHS reference.

of 3 and 12 months; linear growth rates improve (approaching 80% of the reference values) after the first year of life. Data from the KHardep Impact Study

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Weight gain g/month</th>
<th>% ref.</th>
<th>Reference ht. gain mm/month</th>
<th>Chuliban mm/month</th>
<th>% ref.</th>
<th>KHardep mm/month</th>
<th>% ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–3</td>
<td>780</td>
<td>117</td>
<td>30.0</td>
<td>26.7</td>
<td>89</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3–6</td>
<td>350</td>
<td>66</td>
<td>21.7</td>
<td>15.0</td>
<td>69</td>
<td>15.8</td>
<td>73</td>
</tr>
<tr>
<td>6–12</td>
<td>173</td>
<td>43</td>
<td>15.0</td>
<td>10.4</td>
<td>69</td>
<td>9.6</td>
<td>64</td>
</tr>
<tr>
<td>12–24</td>
<td>136</td>
<td>58</td>
<td>9.2</td>
<td>6.9</td>
<td>75</td>
<td>6.7</td>
<td>73</td>
</tr>
<tr>
<td>24–36</td>
<td>167</td>
<td>100</td>
<td>7.7</td>
<td>6.2</td>
<td>80</td>
<td>5.8</td>
<td>76</td>
</tr>
<tr>
<td>36–48</td>
<td>116</td>
<td>82</td>
<td>7.0</td>
<td>5.8</td>
<td>82</td>
<td>5.6</td>
<td>81</td>
</tr>
<tr>
<td>48–60</td>
<td>100</td>
<td>67</td>
<td>5.4</td>
<td>4.8</td>
<td>89</td>
<td>4.5</td>
<td>83</td>
</tr>
<tr>
<td>60–72</td>
<td>67</td>
<td>45</td>
<td>5.0</td>
<td>4.0</td>
<td>80</td>
<td>4.1</td>
<td>82</td>
</tr>
</tbody>
</table>
support the Chuliban results and reveal that slowing of linear growth is maximum between 6 and 12 months. Taken together (Fig. 3), the results confirm that slowed linear growth starts soon after birth and becomes pronounced during the first 12 months of life.

Cross-sectional studies reveal that in Nepal stunting (lengths or heights less than 90% of reference) is prevalent from an early age. The results of the Nepal Nutrition Status Survey showed that 48% of children in the 13- to 24-month age group were stunted; the prevalence rate rises to 63% in the 49- to 60-month age group.

ANALYSIS OF DIFFERENT VARIABLES ASSOCIATED WITH SLOWED LINEAR GROWTH

Stunting is widespread in South Asia, and workers in community health programs want to know its significance. Does it indicate that a child is in danger? If so, what can be done to reduce this danger? The risks associated with being stunted have been examined in Bangladesh (12,13): the stunted child does have a higher risk of mortality compared to the child whose height is close to the reference value. Hence, personnel working in health and development programs in East Nepal have been involved in analyzing factors associated with the high prevalence rate of stunting.

![Graph](https://via.placeholder.com/150)

**Fig. 3.** Khardep Impact Study and Chuliban Longitudinal Study: estimated monthly length or height gain of children as a percentage of the expected gain (NCHS reference) by age group.
The Child's Sex

From the age of 3 months, female children in the Chuliban Longitudinal Study were an average of 1 cm shorter than males of similar ages. In the cross-sectional study of children under 5 years of age undertaken in Surkhet, the prevalence rate of severe stunting (height or length less than 85% of reference) was higher among girls (37%) than among boys (25%).

The Geographical Area

Results of the 1975 Nepal Nutrition Status Survey (1) revealed that 52% of children in rural Nepal aged less than 6 years were stunted. The prevalence rate of stunting in the hill terrain (55%) was higher than that in the lowland terrain (45%). Of all the geopolitical units in the country, the Eastern terrain had the highest prevalence rate of wasting and the lowest prevalence rate of stunting.

The Season

There is a pronounced seasonal variation in the climate in Nepal. Winters are cold; the early summer months (April and May) are hot and dry, though showers start toward the end of May. The late summer (July–September) is the monsoon season. The climate influences farming systems: harvests coincide with the end of the monsoon. The months before the harvest are associated with household food shortages, heavy demands on family members to undertake agricultural labor, and a high incidence of diarrheal disease.

Studies in rural Bangladesh have reported seasonal fluctuations in the nutritional status of preschool children; prevalence rates of wasting and stunting change with the seasons. The highest prevalence rates of mild and moderate wasting occur between July and December, the monsoon period that precedes the main rice harvest (14,15). There is a seasonal increase in prevalence rates of stunting between December and April, even though this is the period of maximum weight gain. Increased height gain and a fall in the prevalence rate of stunting occur about 3 months after the period of increased weight gain.

Data collected by several agencies reveal a similar seasonal variation in the prevalence rate of wasting among Nepalese children, with figures highest during the late monsoon months. The weight gain of children in the Chuliban study aged 12 to 35 months was greatest during the months from the middle of the monsoon, when the first cereal crops are harvested, until mid-January. Peak weight gains were recorded between mid-August and mid-October.

The length gain of children aged 12 to 23 months was greatest between mid-December and mid-April (Nepali months 9 to 12), 3 months after the period of maximum weight gain (Fig. 4).

Monthly prevalence rates for severe stunting in Chuliban (height or length less
FIG. 4. Chuliban Longitudinal Study: monthly level of height gain in cm among children in the 12- to 23-month age group. The seasons and month of the maize harvest are also shown.

FIG. 5. Chuliban Longitudinal Study: prevalence of severe stunting (< 85% height for age) among children aged less than 6 years, by month. The seasons and month of the maize harvest are also shown.
TABLE 4. Nutritional status (height/length for age) of children in SCF Baglung Nutrition Survey analyzed with respect to sufficiency of household food production during the previous year*

<table>
<thead>
<tr>
<th>Sufficiency of food production</th>
<th>Height/age &lt; 90%</th>
<th>Height/age &lt; 85%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Total</td>
</tr>
<tr>
<td>No food</td>
<td>29</td>
<td>39</td>
</tr>
<tr>
<td>3 months</td>
<td>58</td>
<td>89</td>
</tr>
<tr>
<td>3–6 months</td>
<td>251</td>
<td>394</td>
</tr>
<tr>
<td>6–9 months</td>
<td>200</td>
<td>372</td>
</tr>
<tr>
<td>9–12 months</td>
<td>308</td>
<td>595</td>
</tr>
<tr>
<td>Total</td>
<td>846</td>
<td>1489</td>
</tr>
</tbody>
</table>

*Chi squares: \( x^2 = 9.79, df = 4, p < 0.05; x^2 = 44.55, df = 4, p < 0.0001. 
Height/age 90% vs. food sufficiency 10.3, 4 df (p < 0.05). Height/age 85% vs. food sufficiency 48.1, 4 df (p < 0.001).

TABLE 5. Nutritional status (height/length for age) of children in SCF Baglung Nutrition Survey analyzed with respect to household ownership of a milk-producing buffalo*

<table>
<thead>
<tr>
<th>Ownership of buffalo</th>
<th>Height/age &lt; 90%</th>
<th>Height/age &lt; 85%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Total</td>
</tr>
<tr>
<td>No buffalo</td>
<td>413</td>
<td>683</td>
</tr>
<tr>
<td>Buffalo</td>
<td>440</td>
<td>806</td>
</tr>
</tbody>
</table>

*Asterisk indicates significant difference (p < 0.01).

than 85% of NCHS reference) are shown in Fig. 5. Between Nepali years 2035 and 2037 (1977–1979), the prevalence rates of severe stunting were highest between late August and mid-February (months 5–10, inclusive); this was the period of maximum weight gain.

The Economic Status of the Household

The association between growth patterns and household socioeconomic status was investigated in the Nepal Baglung Nutrition Survey in 1979 (Tables 4 and 5).

The results showed that the prevalence rates of both stunting and severe stunting among children from households classed as poorer was greater than those among children from wealthier households. Analysis of variance reveals that sufficiency of household food production is a highly significant predictor of height for age. This significance remains when the effects of age, sex, caste, and number of children aged less than 15 years in the family have been taken into account.
The 1979 KHARDEP Baseline Survey revealed that the prevalence rate of stunting varied both with the age of the child and with the socioeconomic status of the child’s household (Table 6).

The prevalence rate of severe stunting among children aged less than 8 years from households cultivating less than half a hectare was significantly greater than that among children from households cultivating more than 1 ha. However, when the prevalence rate of severe stunting was studied among children in different age groups, the differential was evident in the 12- to 35-month and 60- to 95-month group but not in the 36- to 59-month group.

It seemed possible that children from households cultivating more than 1 ha showed catch-up in linear growth rates after the age of 59 months. The prevalence rate of stunting is lower among these households’ children when they are aged 60 to 95 months than among children aged 36 to 59 months. This pattern, which suggests catch-up in linear growth, is not seen among children from the poorer households studied on the same occasion.

More recent results, obtained in the KHARDEP Impact Studies, show that it would be unwise to suggest that catch-up has occurred on the basis of cross-sectional data alone. Data obtained from approximately 400 children measured on seven occasions from year 2 (1982) to year 5 (1985) of the studies have been analyzed. Children were measured either in the dry winter season or in the monsoon. The prevalence rate of severe stunting at each visit shows considerable variation (Fig. 6) and is difficult to interpret, not least because the winter visits coincide with the likely period of maximum linear growth rates.

Food production data collected from the same households reveal that the harvest at the end of 1982 (year 2) was extremely bad. By the monsoon of 1983 (second visit, year 3), the prevalence rate of stunting in all age groups increased dramatically compared with the prevalence rate during the monsoon of 1982. The prevalence rate of wasting did not show a dramatic rise during the 1983 monsoon visit. Subsequent harvests were better, but the level of severe stunting in the 36- to 59-month age group remained high until 1985 (10).

Households were classified as poorer or wealthier on the basis of the area and type of land that they cultivated. Figure 7 shows that during all visits except 3.1, 174 INFECTIONS AND ENVIRONMENTAL FACTORS

TABLE 6. KHARDEP Baseline Survey (Nepal 1979): prevalence of severe stunting (< 85% height for age) among children by age group and area of land cultivated by household

<table>
<thead>
<tr>
<th>Household land area (ha)</th>
<th>Age group of children (months)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12-35</td>
<td>36-59</td>
<td>60-95</td>
<td>0-95</td>
</tr>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>0-0.5</td>
<td>25</td>
<td>24%</td>
<td>29</td>
<td>34%</td>
</tr>
<tr>
<td>0.51-1</td>
<td>42</td>
<td>19%</td>
<td>54</td>
<td>37%</td>
</tr>
<tr>
<td>Over 1.0</td>
<td>81</td>
<td>10%</td>
<td>71</td>
<td>32%</td>
</tr>
<tr>
<td>Total</td>
<td>148</td>
<td>15%</td>
<td>154</td>
<td>34%</td>
</tr>
</tbody>
</table>
the prevalence rate of severe stunting among poorer households' children was greater than that among the wealthy households' children. There was a marked increase in the prevalence rate of severe stunting among children from the poorer households following the bad harvest in 1982.

Taken together, the Nepal data suggest that the linear growth rates of Nepalese children are far slower than those of the children used to provide the NCHS refer-

**FIG. 6. KARDEP Impact Studies: prevalence of severe stunting (< 85% height for age) during different visits by age group.**

**FIG. 7. KARDEP Impact Studies: prevalence of severe stunting (< 85% height for age) among children aged 12–35 months by visit and household wealth.**
ence figures. The difference is most marked in infancy. There is definite evidence of an association between linear growth rates in childhood and household poverty. This is exacerbated by the effects of harvest failure. The suggestion of seasonal variation in linear growth rates can complicate the interpretation of longitudinal data.

The Child’s Ethnic Group

The people of Nepal are drawn from a number of different tribal and caste groups: some are of Indo-Aryan origin; others are of Tibeto-Burman origin. Physically the members of these two ethnic groups look very different: the Indo-Aryans tend to be slimmer, the Tibeto-Burmans stockier. There are clear genetic influences on the final height and physical shape of Nepali adults.

In the Surkhet District Nutritional Survey (2), the children of the Magars and Gurungs (Tibeto-Burmans) had a higher prevalence rate of stunting than did those of the Tharus (Indo-Aryans). The socioeconomic status of the two groups is broadly similar, though their habitat (hills versus plains) and feeding practices are different. Results from the KHRDEP Baseline Survey (4) revealed similar prevalence rates of stunting for Indo-Aryan and Tibeto-Burman children.

Studies of privileged households in Kathmandu, undertaken as part of the Nepal Nutrition Status Survey (1) in 1975, confirm that under favorable economic circumstances and environmental conditions, the linear growth of Nepalese children aged less than 5 years is similar to that of Western children. However, it is likely that the growth patterns of children in adverse economic and environmental circumstances will be determined to some extent by genetic factors.

Preliminary results suggest that when incidence of infections is high and nutrient intake is low, the linear growth rate of Tibeto-Burman children is less than that of the Indo-Aryan children. These associations will be studied further.

Infection

Childhood infections, particularly those affecting the intestinal and upper respiratory tract, are extremely common in East Nepal. They have an adverse effect on the nutritional status of preschool children. This may be a result of several different mechanisms.

Children who are ill lose their appetite; this anorexia will result in a reduction in food intake. Food may not be offered to the child for cultural or compassionate reasons, and food that is consumed may be lost through malabsorption or vomiting. The presence of fever may increase the child’s energy needs above normal requirements. Of all these factors, anorexia and the mother’s reluctance to feed the child are the most important in determining the nutritional response to an infection (16).

Data from the Chuliban Longitudinal Study reveal that children with diarrhea
lose an average of 0.37 kg during the month after the episode compared with an average gain of 0.11 kg experienced by children who did not have diarrhea in the same month (6). Even greater differences are seen in children suffering from measles. This weight loss may be associated with a reduction in the rate of height gain. Preliminary results from the studies suggest that children who are less than 85% weight for height during the summer months gain less height than children whose weight is greater than 85% of the reference weight for height at this time.

The influence of infections on growth has been observed in many other parts of the world (17–19). Results suggest that frequent episodes of infection, particularly diarrhea, lead to acute weight loss and impaired linear growth. In one study in Bangladesh, each episode of prolonged diarrhea was associated with a 0.56-cm reduction in linear growth (20). Studies in northeast Brazil reveal that among children aged 1 to 2 years, the average increase in height of those with diarrhea during a 3-month period was 41% less than in children with no diarrhea during the same period (21).

Until recently, most investigators had been unable to determine the precise effect of infection on the growth of children. They needed to separate the effects of infection from other factors usually associated with the presence of both infection and malnutrition—lack of adequate food, sanitation, clean water, time for child care, access to health services, in other words, a poor social and economic environment. However, the group working in northeast Brazil has demonstrated that diarrhea remained a significant contributory cause of stunting even when age, socioeconomic status, and initial nutritional status were taken into account (21). Thus, though children may quickly regain weight lost during an episode of diarrhea, the long-term effect of the infection on linear growth persists.

THE ASSOCIATIONS AMONG INFECTIONS, ENVIRONMENTAL FACTORS, AND SLOWED LINEAR GROWTH

We have identified several factors that together account for the slowed linear growth of children in developing countries. We suspect that in young children there is an association between weight for height and the potential for subsequent linear growth. During the period of maximum height increment in seasonal environments, children who have been thin for long periods in the preceding months probably grow less in height than children who have been fatter.

Children are likely to have low weight for height as a result of infection and diminished food intake. The length of time for which a child remains below the expected weight for his height is likely to determine his linear growth during the succeeding months. The duration of low weight for height may be a more important determinant than the incidence rate of low weight for height. If this hypothesis is true, children who gain weight rapidly after an episode of infection will subsequently gain in height at the expected rate. Children whose catch-up growth is less satisfactory will, in turn, gain less in height.
Such a hypothesis explains the association between poverty and height gain rates, as poor families are less able to ensure rapid catch-up after their children's infections. It also explains why conditions that worsen the position of the poorest in a community may result in increased stunting prevalence rates with only a transient, if any, effect on wasting prevalence rates.

These hypotheses need to be examined further in order that the linkages among poverty, illness, food intake, wasting, catch-up growth, height gain rates, and stunting can better be elaborated in the children of the developing world.

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We acknowledge financial assistance from the Overseas Development Administration Nutrition and Health Research Funds (Grant No. R3625, Child Nutrition in Nepal; Grant No: R4143, Assistance with data collection in Nepal), and from the Save the Children Fund. Some of the data quoted were collected while D.N, C.C., M.P., and A.W. were contracted to assess the impact of the Kosi Hills Area Rural Development Programme.

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DISCUSSION

Dr. Tanner: You are absolutely right in saying that the rate of growth, not only now but also in the last century during the course of industrialization, has been used by economic historians as an indicator of the living conditions of the working class and that around the world the correlation between height and social conditions, for means of populations, is very close. I would just like to add a professional auxologist's warning about getting far away from the data, turning it into percentages, using references, etc. For example, people sometimes forget that the NCHS has a big break at 3 years old: the first 3 years are from a totally different group than the rest. The reason that your children appeared smaller in the first 3 years is that they were being compared to a highly privileged Northwest European Caucasian stock measured at the Fels Institute. At 3 years old the NCHS shifts gear totally, and it is then a proportionate sample, theoretically at least, of all children in the United States.

There is another problem; I don’t know if it would affect your data, but I think it may have done. If you take a percentage of the median, the coefficient of variation isn’t the same as the child gets older. So 85% of median at one age gives you lots more children undernourished than 85% at another age.

Dr. Rappaport: Dr. Tanner, how would you express the results in this type of study?

Dr. Tanner: I would quite simply look at the means and variances of the heights in the various groups.

Dr. Nabarro: Would you compare the heights that we have obtained with the reference values and express them as standard deviation units, or would you be particularly interested in the means and variances of the actual heights?

Dr. Tanner: I'd be particularly interested in the means and variances of your heights, but if I were then going to put them in relation to any reference value, which is a very popular thing to do, then indeed I would do that in terms of SDs.

Dr. Nabarro: We would very much like to have data from a reference population show-
ing means and variances of length and height increments. That was what we were really after when doing that work, but we couldn’t find any data.

*Dr. Tanner:* That is exactly what our present paper is about. There is a paper (1), again from the Fels Institute, on 6-month increments only. However, 6 months is perhaps too long. You would prefer 3 months; Prof. Waterlow wanted 1 month. So we have gone down to 1 month or 2.

*Dr. Waterlow:* Dr. Nabarro, referring to Bangladesh you said they calculated that if the children had no diarrhea, they would have grown an extra 0.4 cm each year, and if worm-free, 1.2 cm. That is in children of what age? How do those figures relate to the expected annual increment?

*Dr. Nabarro:* The first data were obtained by Black and colleagues in Matlab, Bangladesh (2). They studied 177 children aged between 2 and 48 months over 12 months. The second set were obtained by Evans and colleagues in Jamalpur as part of a collaborative project involving the Save the Children Fund (3). They studied 467 children aged 2 to 5 years over 12 months. Examination of the NCHS reference figure reveals that children aged between 12 and 24 months would be expected to gain about 9 cm a year.

*Dr. Waterlow:* The impression I get is that the differences are not very great, and I have been arguing for some time that the effects of infection are often exaggerated. When you compare the growth in either weight or height between highly infected children and much less infected children, the differences are not very large (4). The problem is to relate the duration as well as the prevalence of illness over fairly long periods, say a year, to the growth during that year. I don’t believe there’s going to be a uniform pattern everywhere.

*Dr. Nabarro:* Our evidence suggests very strongly that there are 3 or 4 months in the year in Nepal when children aged between 12 and 23 months gain on average about 1.5 to 2 cm in length. For the rest of the year, they appear to gain very little. This suggests that, just as with weight gain, there are circumstances under which length increment can be extremely “good.” It seems very unlikely to us that the poor linear growth during the other 9 months of the year can be accounted for on the grounds of infection alone. I think that the processes are more complex and that we have insufficient information to permit us to suggest causes of retardation in linear growth. There are only certain months in the year when both weight gain and length increment are sufficient for children to achieve their maximum growth potential.

Perhaps the relative importance of infection or inadequate nutrient intake in retarding linear growth will not be of great relevance to national development policy makers in Nepal. The most important finding is that households that cultivate less than half a hectare of land are most likely to have children who are severely stunted; these same households are also likely to have children who are suffering from a wide variety of disabilities and risks to their health.

*Dr. Tanner:* To follow up what Professor Waterlow was saying, if you lose a centimeter every year, then it becomes a lot. One of the good ways to end up very small is to grow not at the 50th centile but consistently at the 40th. A one-time response to deparasitization would be fine, but if it’s something that continues, even that centimeter can be very important.

*Dr. Tomkins:* You’ve made a very interesting observation, that there was a dramatic increase in stunting and not in wasting at a certain period in the year. You also observed that there were two genetic groups in your population and that there may be genetically different responses to an infection. Were the increases in stunting and wasting the same in both ethnic groups?

*Dr. Nabarro:* No. It’s still difficult for us to give consistent answers. If we analyze chil-
children from households with broadly similar economic status but belonging to these two ethnic groups, we find in some of our studies that there are substantial differences in the prevalence rates of both stunting and wasting between the groups. We have not yet been able to demonstrate differences in the linear growth rates of children belonging to the two groups. Perhaps we should do more than consider the maximum potential growth of children belonging to different ethnic groups. We should also consider how genetic factors influence the way in which children respond to deprivation.

**Dr. Rappaport:** Is there any other model in which you could sort out genetic factors that might control catch-up growth?

**Dr. Nabarro:** It would be possible, I am sure, in parts of northern India. However, it may be that disaggregated data are not yet available to make this kind of comparison possible.

**Dr. Kraisid:** Do you find any difference in terms of feeding practices, especially in supplementary feeding, among these different ethnic groups? In Indian populations, complementary feed usually starts at 6 to 12 months. Among Burmese or people in southeast Asian countries, supplementary foods are given earlier. This may have some effect.

**Dr. Nabarro:** There are certainly differences in feeding practices; there may also be differences in rates of infection. As you say, these may be relevant.

**Dr. Waterlow:** Dr. Nabarro made a point that I think is extremely important, that the increase in length when things improve in the course of the seasonal effects follows the increase in weight. From a physiological point of view, this seems to be most interesting. I wonder whether Dr. Golden has similar information about the way children behave when they are being rehabilitated in hospital. I certainly have the impression that gain in length in our recovering children followed gain in weight. I would like Dr. Nabarro and Dr. Golden to comment on this.

**Dr. Nabarro:** We observed this 3 years ago when we compared children who had shown good height gain and children who had shown poor height increment over an interval of 6 months. We looked at the weight for height of these children at the start of the 6-month interval. We found that the amount of height gained was highest in the children with the greatest weight for height values. We did not follow this up. However, in view of Dr. Rappaport’s report, it seems to be highly relevant. Similarly, our observation made some years ago that seasonal differences in weight gains and height increments came 3 to 4 months apart now appears to be highly relevant too. We would like to reexamine some of our longitudinal data to study these observations in more detail.

One possibility will be a study of the very high prevalence rate for stunting in the third year of the KHRDEP Impact Studies in 1983. Was there a detectable decrease in the rate of weight gain of the children who became stunted during the 3 or 4 months before they should have increased in height?

**Dr. Golden:** We have only started to look at the question of what happens during recovery from malnutrition. About 8% of the children start to gain height as soon as they enter the ward; about 15 to 20% don’t gain any height at all while they’re in the ward, and we don’t know what differentiates them. The vast majority of children suddenly begin to gain height when they have reached a median of 85% weight for height; then, they get their first half-centimeter increment in height. At the moment we are trying to look at the variance of their weight per height at the time when they get their first height spurt. It seems at first sight to be quite narrow and certainly centered around 85% weight for height. It also seems at the same time to be that their somatomedin-C levels show a sharp increase, but this is very preliminary data based on a small number of children.
Dr. Nabarro: Perhaps the different timing of weight gain and height increment in the children we studied would be reflected in their growth hormone and somatomedin-C levels. A careful study of the associations between seasonal growth and hormone levels might explain these differences. Clearly, such a study would not be easy to undertake under field conditions.

Dr. Rappaport: That should probably be interesting, but it is a difficult approach. Dr. Golden, could you tell us what is more important? Is it the amount of weight that is gained when the child is in the ward or the percentage of weight for height reached during the same period?

Dr. Golden: We don't have body composition studies, but I think it's much more important to know the lean tissue mass in relation to height rather than the total weight for height. The absolute amount of weight gain by the children does not seem to be very important. The children who start off very low in weight for height will fill out, and when they reach 85% then they start to gain height. The children who come in much less stunted, or the children with kwashiorkor who just lose their edema and then gain up to 85% weight for height, will then have a reasonable height. Thus, the absolute amount of gain does not seem to be related, nor does the absolute amount of height deficit that the children come in with. The age of the children, however, is related to the height gain. The younger children show this height spurt at reaching 85% weight for height very much more regularly than the older children.

Dr. Martorell: It's an interesting finding that when the situation improves, children first put on weight for height and afterwards grow in length. To me, this suggests that stunting and wasting are caused by the same factors and that a child, when faced with a nutrient deficiency that is not severe, may cope simply by slowing down in growth; we don't know much about effects of physical activity. If the deficiency is severe, cessation of growth alone is not enough, and in addition the child has to subsist on his own tissues. When the situation improves, the child first puts on weight for height and then starts growing in length. Could stunting and wasting be caused by the same factors, the difference simply being a reflection of the severity, acuteness, or suddenness of the causal factors?

Dr. Nabarro: This is our explanation for what we have seen, with the proviso that children from different ethnic groups do not respond to the causal factors in the same way. Thresholds at which they slow their growth and start to become wasted may vary in the different ethnic groups. This hypothesis needs to be tested.

Dr. Colombo: First a short comment. A 6-year follow-up study of about 400 malnourished children has been conducted in Chile; a very good correlation was found between weight for height after discharge—these children were hospitalized for about 5 months—and subsequent growth. I have also a question. Have you done any psychomotor studies in the children you were working with?

Dr. Nabarro: No, we have not done this.

Dr. Ousa: Our experience in treating children with severe protein-calorie malnutrition (PCM) is that those who come in with severe PCM gain weight and height more quickly than the ones who come in with less severe PCM. Wasted children with similar intakes, e.g., 4 g of protein and 175 kcal/kg, gain weight better than stunted children. Some children may appear very healthy although their weight is far below their expected weight for age, down to 60%. For that reason, we always rely on the weight for height. The cut-off point for discharge is 85%; when the children leave the hospital earlier, the relapse rate increases substantially.

Dr. Nabarro: Do you have any length or height increment figures for these children?

Dr. Ousa: Yes, we do.
Dr. Nabarro: It would be interesting to examine the height increment of children less than, say, 85% weight for height during succeeding months and then their height gain once they pass that particular cut-off figure. Of course, it may not be 85% in your situation.

Dr. Tanner: It seems to me important to know the family structure, the number of children in the family, the interval between pregnancies, and also when these children between 6 and 12 years old actually go out to work and the sort of work they do. Have you gone into that?

Dr. Nabarro: Yes, we have. What I was showing here were three indicators that we finally identified to disaggregate the children we studied into meaningful socioeconomic groups. We looked at very large numbers of variables before deciding to use the area of land cultivated by the household, the number of months for which food production is sufficient, and the presence or absence of a milk-producing buffalo. I agree that if we were to require a more precise indication of the actual wealth of a household, we would need much more detailed information about household assets and to relate it to household size (dividing the assets by the number of 'adult consumption units' in the household). We should also take into account the amount of 'off-farm' income coming into the household. The methods we used to indicate household wealth will inevitably have led us to misclassify certain households. However, it has enabled us to draw some broad conclusions about associations between household wealth and child nutritional status.

Dr. Waterlow: I don’t want to see the point made by Dr. Martorell—that he regarded stunting and wasting, if I may still use these words, as the same condition—to go unchallenged. I totally disagree with him, even more after the previous chapters. That does not, of course, mean that I am right and that he is wrong. But, first of all, I disagree with him on the statistical evidence that was produced by Dr. Keller, and, second, I think we should bear the physiological points in mind that have been raised by Dr. Rappaport and Dr Milner: the body consists of different kinds of tissues—very roughly, let us say, the visceral tissues, peripheral or muscle tissue, and the skeleton. There is no question that these tissues respond to malnutrition and to rehabilitation in different ways: albumin and liver regenerate quickly, muscle less quickly, and bone, from what we have heard, perhaps still less quickly. It seems to me a very unphysiological attitude to look on these tissues as the same. However, it is probably wrong to open such a discussion at this point; it may come up again in the general discussion.

Dr. Nabarro: The kind of observations that we are producing suggest that the two processes—slowed weight gain and linear growth—are not necessarily caused by the same factors. They also suggest that children will only show a substantial increment in linear growth if they have had satisfactory weight gain during the preceding months. Retardation in weight gain and linear growth are not necessarily results of the same process, but there appears to be a definite permissive relationship between the two processes. The annual increase in a Nepalese child’s length or height may well depend on postharvest weight gain.

REFERENCES