The Risk of Morbidity in a Stunted Child

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The general relationships between malnutrition and infection have been recognized for centuries, but the details of the links have been defined considerably only during the last few decades. Many of the studies examining the effect of malnutrition on the response to infection have been reviewed (1,2). However, most of the clinical, epidemiological, and experimental reports have emphasized the importance of severe protein-energy malnutrition (PEM), underweight, and thinness rather than shortness of stature or slow rates of height gain. It is the purpose of this chapter to review what is known about the relationship between short stature and subsequent risk of disease and death. The classification and causes of short stature are reviewed elsewhere (3), but for the purpose of this review the term "short" will be used for those children who are significantly different from standard height/age. Children who are less than 90% height/age will be called "stunted," as proposed by Waterlow and co-workers (4).

There are considerable differences of opinion among experts about the significance of malnutrition. Gopalan maintains that "to plead the virtues of 'smallness' is to acquiesce in the preservation of the status quo of poverty, ill health, under-nutrition, and socioeconomic status" (5), whereas Seckler and Sukhatme (6) maintain that "small is healthy." In recent years there has been considerable use of the term "nutritional adaptation" to describe some of the factors contributing to the slow growth rates of children in less developed countries compared with richer countries. A timely review of the topic (7) has clarified some of the confusion that surrounds the use of the term "adaptation."

A FRAMEWORK FOR ANALYSIS

Before infection can develop, the pathogen, whether bacteria, parasite, or virus, must first broach the body's immune system (Fig. 1). The nature of the impact of nutrition on the individual components of the immune response are comprehensively reviewed by Chandra (8). In general we are concerned with nonspecific host defenses, antibody formation, and the cellular immune system. Once the infection invades the superficial tissues or infiltrates into deep organs, there are various host
factors determining whether an abscess becomes a septicemia, a few intestinal parasites start to replicate prolifically, or whether a bacterial colonization syndrome develops.

The physiological response to infection may vary; intestinal secretion to an enterotoxin-producing enteric pathogen differs according to the nutritional state. Duration of the infection depends on a competent immune mechanism and adequate repair of damaged tissues. The outcome of the infection may be variable. Rapid recovery without residual deficit or complication is what we hope for, but persistent pathology such as the blinding malnutrition following precipitation of deficiency of vitamin A by severe measles is a lifelong complication of a transient infection and disorder of nutrition.

Death itself is more frequent among the severely malnourished, but community-based studies in developing countries have usually experienced difficulties in accurately ascribing a single cause of death in their analyses. Indeed, in all stages of the development and evolution of an infection, we have to consider the result of infection in an individual child to be influenced, to a greater or lesser degree, by the social and physical environment as well as by biological responses to pathogens. Thus, the availability of food and of somebody to prepare and feed it, perceptions of appropriate foods to be given for particular illnesses, and access to medical resources will all have an influence on the outcome of an infection.

IMMUNE DEFENSE SYSTEM

Previous studies in children with severe PEM (marasmus or kwashiorkor) showed a variety of disturbances of cellular immunity (CMI) with atrophy of the
thymus and thymus-dependent lymphoid tissues (9). These could be detected by in vivo tests of responsiveness to antigens injected into the skin or in vitro tests on a range of cell types. These findings have been implicated as the major reason why severely malnourished children have such a high incidence of tuberculosis and miliary infections.

There were also several studies of CMI in children with milder grades of PEM (10,11). Most of them showed intermediate responses in CMI among children with moderate PEM (weight/age 60–75%) and normal responses in those with mild PEM (weight/age 75–90%) compared with results in well-nourished children. However, the authors did not report the length or height of these children.

Studies of the humoral responses in severe PEM have shown relatively little evidence of impaired function. Several reports described lower levels of IgA in tears, intestinal secretions, and jejunal mucosa (12). However, seroconversion to measles vaccine appears to be satisfactory, albeit delayed, in some children (13). Again, none of these reports included information on height or length.

The nonspecific immune functions such as integrity of mucosal surfaces and gastric acidity (14) are all abnormal in severe PEM, but there is no information on gastric acidity in moderate or mild PEM and no data on the relationship between gastric acidity and height/age.

In view of the absence of any data on the impact of stunting on the immune response, it might be valuable to inspect the literature on immune response in fetal malnutrition. There are problems of interpretation in that neonates who are born with a low birth weight and shorter length have been exposed to different nutritional and nonnutritional stresses than those experienced by a child who becomes stunted postnatally. It is necessary to distinguish those neonates whose physical development is appropriate for the gestational age (AGA) from those that are small for gestational age (SGA). The latter group might have been born small as a result of maternal undernutrition, hypertension, preeclamptic toxemia, maternal infection, or placental insufficiency (8).

Studies of rosette-forming T lymphocytes and hypersensitivity reactions to skin antigens show abnormal results that persist for as long as 5 years after birth among SGA children, whereas the immune system of AGA children is either normal from birth or improves rapidly within a few weeks of delivery (8).

There is some deficit in transplacental transfer of antibodies to tetanus toxoid in SGA infants together with generally lower levels of serum IgA and IgG and a slight decrease in titers of antibody to polio vaccine following immunization (15), but, in general, most immunization responses are equally effective in SGA and AGA infants.

Neutrophil function, migration of polymorphonuclear leucocytes, neutrophil bactericidal activity, and opsonic activity are also decreased in SGA infants. Studies in Guatemala showed an increased mortality for SGA babies compared with AGA babies (16). An interesting feature of SGA babies is that many of them develop circulatory antibodies to food antigens (17). This may well set the scene for persistent diarrhea in which food intolerance is thought to play an important role.
INCIDENCE OF INFECTION

There is no doubt that infection rates among children are higher in those countries where malnutrition is also prevalent, whether the malnutrition is categorized as underweight (low weight/age), short stature (low height/age), or thinness (low weight/height). A crude global estimate suggests that only 6% of children less than 5 years of age in more developed countries (where stunting is rare) have an episode of diarrhea during a year, whereas the majority of children in less developed countries have more than two episodes annually (18). Thus, on international comparison, there is a link between stunting and incidence of infection.

However, longitudinal studies of nutritional status of individual children within a single community and their subsequent morbidity do not show a clear relationship between stunting and incidence of infection (19,20).

A total of 2,019 children aged 12 to 23 months were weighed, and their height was measured, in a rural area of Bangladesh. Their attack rate for diarrhea was then assessed by recording the number of visits to a diarrhea treatment center during the following 24 months (21). Frequency of visits was similar among taller and shorter children. Neither was there any greater frequency among those who were underweight or thin. A more recent study in the same area in Bangladesh (22) examined the relationship between nutritional status among 197 children less than 4 years of age and their attack rate for diarrhea as assessed by regular home visits by trained field workers in the subsequent 60 days. Again, there was no greater attack rate among the underweight, short, or thin than among the better nourished.

Three hundred forty-three children aged 6 to 36 months living in a rural area of Malumfashi District, northern Nigeria, were measured, and diarrhea morbidity was assessed by weekly home visits (23). As in the Bangladesh studies, there was no greater frequency of diarrhea in those who were shorter or more underweight, although there was a slight but significant increase in frequency among those who were wasted (<80% weight/height). Subsequent analysis showed that there was a bias in this group because many of the wasted children were just recovering from measles, and they may well have been more susceptible to the postmeasles diarrhea syndrome.

The factors affecting the incidence of diarrhea in an individual or community are reviewed in more detail elsewhere (24) and summarized in Table 1. The main

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point is that only two of these (gastric acidity and intestinal immunity) are likely to be influenced directly by nutritional status, and even then the data on gastric acidity (14) and intestinal immunity (12) are only available from severely malnourished children.

The complexity of biological, behavioral, and environmental factors affecting the frequency of episodes of diarrhea is such that they may well override any effect of malnutrition on incidence. In a recent longitudinal study of children aged 6 to 36 months in Bakau, an urban community in the Gambia, West Africa, there was a high attack rate for diarrhea among the community as a whole, but a quarter of the children managed to survive the rainy season, an unpleasant time with high humidity, environmental temperature, and contamination of food, without any diarrhea at all (25). When 38 variables, including style of house, water supply, sanitation, and education of parents were analyzed, there were only two that correlated significantly with attack rate. This may seem surprising at first, but a walk through the township soon reveals that young children do not stay within the social and environmental groups of their parents. Play activities in the streets, visits to a variety of other compounds, and snack eating are all major opportunities for the acquisition of enteropathogens that may not exist in their own homes. Thus, the data available do not eliminate the possibility that stunting has an important effect on disease incidence, but in the face of overwhelming environmental stress affecting the whole community, well nourished as well as undernourished, the hypothesis that stunting increases the incidence of infection is not yet proven.

SEVERITY OF INFECTION

Many studies have emphasized the severity of illnesses such as measles, watery diarrhea, dysentery, and pneumonia among the severely malnourished (26). However, recent studies have shown that measles infection may be very severe, even among the well nourished, if it develops during the first year of life. Studies in South Africa (27) and Guinea-Bissau (28) showed that mortality rates were very high among urban children in whom severe measles occurred during infancy and early childhood. It seems possible that the severity may be caused by the poor host response in the younger child, whatever the nutritional status. The high-density living conditions for poor blacks in the slums of South Africa and Guinea-Bissau and for poor whites in the slums of Scotland earlier this century (29) may encourage a large inoculating dose of infection, which may account for the severe clinical picture (27). Unfortunately, there are no data on the attack rate or clinical severity of measles according to height/age.

DURATION OF INFECTION

There is more convincing evidence that stunting is associated with prolonged illness (Table 2). In the study of children in Malumfashi, northern Nigeria (23), episodes of diarrhea lasted 30% longer among the stunted than in the taller chil-
TABLE 2. Possible risk factors for prolonged duration of infection

- Delayed immune clearance of pathogen
- Delayed repair of damaged tissues (e.g., PEM, folate or zinc deficiency)
- Persistent mucosal damage (e.g., food intolerance)
- Carbohydrate malabsorption (e.g., lactase deficiency)

dren. A similar finding was noted in the nutrition/diarrhea study in Bangladesh (22), where, overall, there was a 26% longer duration in the shorter than in the taller children. A particularly valuable aspect of this study is that microbiological investigations were performed in a high proportion of diarrheal episodes. When those episodes attributable to enterotoxin-producing *Escherichia coli* only were analyzed, the diarrhea lasted about three times longer among the shorter than among the taller children. There was a similar threefold increase in duration of *E. coli* diarrhea among the most underweight and the thinnest compared with the better nourished in each category. In addition, there was a similar though less striking pattern in cases attributable to *Shigella*. In both the Nigerian and Bangladesh studies, the greatest association was between wasting and subsequent duration of diarrhea. It is interesting that such a relationship has also been found in Indonesian children (30), but unfortunately no data are available on the relationship between stunting and diarrhea incidence or duration in that country.

A more recent study has examined the relationship between anthropometric indices and subsequent disease patterns among children in Bakau, an urban community in the Gambia, West Africa (31). Although this study examined a wide range of illnesses (fever, respiratory infections, skin infections, and accidents as well as diarrhea), the data on morbidity were not collected as frequently as in the Nigerian or Bangladesh studies. Nevertheless, it was possible to calculate the prevalence of illness. For all the disease categories except diarrhea, prevalence was similar among the undernourished (whether more underweight, shorter, or thinner) and better nourished (32). The differences in prevalence of diarrhea between stunted and taller children were especially marked in the second year of life.

There are several possible biological reasons for prolonged symptoms in infection. Cellular immunity and hormonal immunity (especially secretory IgA) may be less active, thus limiting the speed at which a pathogen is cleared from the host. There is an abundant literature on the effect of dietary deficiency (nearly always severe by any standards) on the immune status and clearance of experimental pathogens in animals, but data on milder nutritional deficits are minimal, and the hypothesis that a stunted child has more persistent symptoms because of delayed clearance of intestinal pathogens is neither proven nor disproven.

Several studies have concentrated on the effect of nutritional status on function of the intestinal mucosa. Studies in protein–energy malnutrition (33), folate deficiency (34), and zinc deficiency (35) all show mucosal atrophy as a result of decreased rates of production of enterocytes in the crypts of the intestinal villi. This
MORBIDITY IN A STUNTED CHILD

may have a direct effect by decreasing surface area for intestinal absorption. It may also affect the type of cell that lines the intestinal villi; in nutritional deficiency the cells covering the villi are predominantly immature, cuboidal enterocytes with poorly developed microvilli and low levels of digestive enzymes (36). This may be especially important among children receiving milk (either human or cow’s), where secondary hypolactasia may contribute to the prolongation of diarrhea (37). A further nutritional factor affecting the gut is the secretory characteristics of immature enterocytes; they have high levels of Na$^+$ ATPase. This may well explain why there is a particularly increased secretory response of water and electrolytes when cholera toxin is perfused in the small intestine of animals with experimental zinc deficiency (35).

There are also important social factors affecting the relationship between duration of disease and stunting. It is frequently observed that within a region or community, those children who are stunted are often from poorer socioeconomic environments (31) not only with respect to type and availability of food but also housing, sanitation, parental education, and access to health services.

The importance of obtaining treatment at an early stage of infection for diarrhea (especially dysentery, where antibiotics may be indicated) is obvious. Delays in initiation of treatment may result in prolonged illness. Despite the many evaluations of the utilization of health services by communities, none has examined whether stunted children obtain effective treatment at a later stage than better-nourished children. Thus, the suggestion that the increased duration of morbidity in stunted children is caused by delays in therapy is, although perfectly logical, unproven.

MORTALITY

Prospective studies in India (38) and Bangladesh (39) have demonstrated an important relationship between weight/age and subsequent risk of death. Other studies demonstrated a relationship between thinness, whether by midarm circumference (40) or by weight/height as in Bangladesh (39) and Papua New Guinea (41), and subsequent mortality. The detailed study in Bangladesh in which deaths were recorded during the 2 years following anthropometry showed an increase in mortality in relation to weight/age, weight/height, and height/age, but only among the severest by each anthropometric category (39). This threshold relationship has been highlighted by reviewers who have emphasized that high-risk children can be identified by anthropometric screening. However, a more recent analysis of these data, in which only deaths occurring in the 12 months after anthropometry were computed, suggests a more cautious interpretation (42). It appears that the relationship between anthropometry and risk of death varies considerably according to the season in which the measurement is made. If children are measured in June (at the beginning of the rains), the subsequent risk of death for the thinnest (assessed by weight/height) is eight times greater than that for the fattest. If, however, the children are measured in October (at the beginning of the dry season), the thinnest
have the same risk of death as the fattest. The predictive value of height/age is also much less if the child is measured in October compared with measurements in June or August.

Mortality during the 18 months following anthropometry among children aged 6 to 60 months in Papua New Guinea was almost twice as high in the thin (less than 90% weight/height) than in the better nourished (41). Among the thin children, mortality rates were nearly three times higher in the stunted children. A study in Africa showed no relationship between anthropometry and death in the 100 days after measurement among children aged 6 to 59 months (43). It is interesting that a high proportion (54%) of the children in this urban community in Zaire died of measles. It could be informative to analyze the nonmeasles deaths separately.

Considerable problems exist in drawing a unifying conclusion from these studies because they differ with respect to age of children, duration of follow-up for assessment of mortality risk, environment, climatic season, and cause of death. Nevertheless, the data indicate that under certain circumstances height/age is a strong predictor of subsequent mortality. The report from Narangwal, India, which only described weight/age, showed a stronger relationship during the first 6 months compared with the second 6 months after anthropometry (38). The data from Bangladesh (42) show a strong, graded mortality rate in relation to anthropometry for the first year, but the relationship with mortality showed a threshold pattern if children were followed for a further year (39).

COFACTORS

Any study of malnutrition and risk of infection or death should recognize that children who are malnourished usually come from underprivileged families with greatest difficulties in obtaining employment, food, potable water, decent housing and sanitation, or even access to the most basic medical care facilities (Table 3). Short stature often relates closely to social and economic deprivation. A recent study in urban Gambia (Bakau) showed a close relationship between stunting and variables such as type of housing, sanitation, water supply, and maternal education, each of which could have a major impact on the risk of infection and mortality (31). It seems likely that the severity of an infection, whether it be measles,

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<th>TABLE 3. Factors coexisting with stunting that may increase risk of morbidity and mortality</th>
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malaria, or diarrhea, is the end result of much more than a balance between host immunity and pathogen virulence. The management of the child by parents or guardians may be markedly affected by any of the above cofactors, which affect childcare. Thus, withholding breast milk, avoidance of certain nutritious foods, purging, and delays in obtaining medical assistance such as sound advice or drugs can all contribute to increases in severity, duration, and complicated outcomes of the illness (44). Some of these variables, particularly withholding of breast milk or food, seem more important in certain Asian communities than African communities and may contribute to variations in disease-specific mortality rates. Again, the lower socioeconomic status of families with stunted children suggests that parents may have less time and/or money to take a child for medical treatment.

In the Bangladesh study analyzed by Bairagi (42), the greater mortality associated with stunting when the children were measured at the beginning (June) or middle (August) of the rains could be accounted for by the greater prevalence of infection and scarcity of food at this time of the year. Admittedly, there is no information on the months in which death was most frequent throughout the year, but from previous studies in this area, most occur during or immediately after the rains. The age of the cohort is such that mortality would be maximal during the months immediately after the anthropometry, thereafter declining as the children enter their third year of life. Perhaps a stunted child who becomes infected during the rains receives less food, parental time, and medicine than a stunted child who becomes infected during the dry season, when there is more food and less pressure on parents' time.

**CONCLUSION**

At the present time, there are many reports about the numbers of stunted children in less developed countries and how these numbers differ with respect to age, dietary patterns, previous illness, and the season of measurement. There is also reasonable evidence that stunted children have more prolonged infection and have a higher mortality from infection in certain communities. That the rate of linear growth is related to the type and amount of diet is incontrovertible, even though a concise, integrated, biological account of the nature of the deficits responsible for stunting is not yet available. It may be that the same nutritional inadequacy that limits the achievement of genetic potential for linear growth is also responsible for dysfunctions of host immunity and tissue repair. However, it is also clear that stunted children often come from homes with social, economic, and environmental deprivation. Those factors that may well explain why the dietary intake was not adequate to achieve the potential for linear growth in the first place may also influence the outcome of an infection in a stunted child.

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**DISCUSSION**

Dr. Martorell: I am not sure I understood you: which of the two is more related to duration of diarrhea, stunting or wasting?

Dr. Tomkins: The relationship between wasting and duration of diarrhea may differ considerably from one community to another. In the Nigerian study I showed, you may have noticed that there was an increased incidence of diarrhea among wasted children. This has not been corroborated by the Bangladesh study. I made an analysis of why the Nigerian children were wasted. For a very high proportion of them, it was because they had developed measles in the month before measurement. Anthropometric measurement was made at the end of the dry season, and postmeasles diarrhea (certainly in many parts of Africa and apparently in Bangladesh too) is a major feature and confounding variable in these studies. I don’t know whether measles epidemiology was tested for in the analysis of the Bangladesh study. So, one can say that in the Nigerian study diarrhea lasted 30% longer in stunted children but nearly 100% longer in wasted children. There was no association, however, between wasting and the prevalence of diarrhea in our Gambian study. The answer is therefore that there may well be differences in the relationship between wasting and risk of infection according to where you are.

Dr. Martorell: Do you think, in looking at the relationship between height for age, for example, and either the duration or attack rate of infection, that it is important to control for socioeconomic status? Could it be that shorter children are ill more often simply because they are poor and that it is a third factor, poverty, that is causing the association?
Dr. Tomkins: Is there perhaps a confounding variable whereby a child who is stunted is a child who is living in poor socioenvironmental conditions and therefore has a higher attack rate? I agree with you that it is what one would expect, but it doesn’t seem to be the case. That is why I spent some time in reviewing the studies. Neither the Chen and Black study nor ours has shown an increased incidence of infection in stunted children. With reference to the Gambian study, we have examined 36 variables; many of them were related to hygiene and feeding practices, and of the 36, we found only two that related to the incidence of diarrhea. Diarrhea, particularly in an urban community, is ubiquitous. Feces are indiscriminately excreted. Children are always running in and out of the compounds; they all live together. I think this explains why in certain societies this kind of study has not picked up these variables, because children don’t stay in their familial socioenvironmental background. It means that when we are looking at risk factors, we need to consider at least two variables: the first is the one that Dr. Nabarro referred to, which is a “family income and poverty index”; the second is a “microlevel family or mother–child behavioral index.” We have not gone into that in this analysis, but I think differences between the two may explain the failure for there to be an impact of height for age on incidence of diarrhea.

Dr. Guesry: As there is apparently a direct relationship between stunting and wasting on the one hand and duration and frequency of diarrheal episodes on the other, I thought both could be attributed to the same socioeconomic factors and that the apparent relationship between the two was only artifactual.

Dr. Tomkins: All I can say is that in the studies that I have reviewed, perhaps surprisingly, I have found very little evidence that malnutrition in its broadest terms, whether it be height for age or weight for age, is associated with an increased attack rate. I think the reason is that most of these studies have been done within relatively homogeneous communities with respect to the environment. In other words, the environment may be very dirty regardless of whether it is in a wealthy compound or in a poorer one. It is interesting that the rate of malaria, for instance, has no relationship whatsoever to socioenvironmental status in our community; indeed, one could postulate that a mosquito does not care how wealthy you may be before biting. Therefore, the relationships that I have put forward are predominantly on the duration of diarrhea. I have suggested that previous measles infection is one possible biological reason for the fact that wasting is associated with an increased duration of diarrhea. The question is: Why does a child who is shorter suffer longer episodes of diarrhea? I think the reason that the question has not been answered so far is that we have concentrated too much on one sector of the child–environment interaction with time. Shortness of stature is associated with increase in duration, but we don’t necessarily know why in each situation.

Dr. Valyasevi: Do you have data on the etiology of the diarrhea in the studies that you reviewed?

Dr. Tomkins: Black’s study, which showed a very interesting linear correlation between duration of diarrhea and height for age, concerned only enterotoxin-producing Escherichia coli. When he looked at all diarrheas in which the pathogen was not identified, the relationship was not so strong. One factor that emerges from this kind of study is that one has to be much clearer in defining what is actually causing the diarrhea biologically.

Dr. Aponso: I agree with you. Prevalence of diarrhea, I believe, depends on the etiological and environmental factors, which are the same whether you are short or tall.

However, at present we know that duration of diarrhea is related to early preventive treatment. That is the story of oral rehydration. I don’t know whether your study was carried out in the pre-ORT era or afterwards. We all remember that in 1978 the Lancet hailed the
discovery of oral rehydration. This was reported to be one of the most significant medical discoveries of the century. Maybe the mothers whose children were taller were also more educated, more motivated, and therefore able to make use of the services that were made available and accessible. Those variables should, I think, be considered. Were these mothers using ORT, and, if so, was it related to the duration of diarrhea?

**Dr. Tomkins:** These are very valuable points. To give you exact information on the Gambian study, the whole community had been very closely involved in promotion of oral rehydration therapy for about 2 years prior to the study; 88% of diarrhea episodes were treated with sugar-salt solutions made up in the home. There was no evidence that the management of diarrhea by better-educated mothers—we were using the criterion of how many years they had been to school, although I am not sure that that is necessarily an indication of education—was any different from the management by those who were less educated. In this community, early use of oral rehydration was incredibly popular; it was widely used at an early stage; but in general your point is a very valid one.

**Dr. Nabarro:** You were showing us, from your Bakau data, material mostly on diarrhea. Do you have anything on measles?

**Dr. Tomkins:** We had about 89% coverage with measles vaccination. We saw no cases of measles.

**Dr. Nabarro:** You studied six variables that you have not shown. You have discussed only diarrhea. Were there any interesting differences in any of the others? Accidents probably are not too relevant, but what about illness, fever, rash?

**Dr. Tomkins:** In fact, those six variables were the variables that related to stunting, not to the diarrheal epidemiology. When we look at the 36 in relation to diarrheal attack rate, which is a slightly separate study, only two of the variables actually related to diarrheal attack rate. One was referred to by Professor Aponso—maternal education, how many years she had been to school; the other was what the field workers felt the mother knew about feeding during illness and the cause of diarrhea. We have analyzed the variables in relationship to fever. There was no association.

**Dr. Nabarro:** So there was no association between the degree of stunting and the number or duration of episodes of fever, for example?

**Dr. Tomkins:** No, the only association that I mentioned briefly was respiratory infection. During the dry season, there was a higher prevalence of respiratory infection among those who were short. That was the only other nutrition-infection relation to be found.

**Dr. Waterlow:** I would like to explore the same line a bit further. The theme of the last few papers is the functional accompaniments, if any, of environmentally determined shortness in height or deficit in linear growth. What we are interested in, at this stage, is not so much what causes children to be stunted but whether that condition of the body has an effect on infection. You have shown rather small and not very consistent effects. You have also made the point of a very uniform environment for these children. Are the environmental factors totally swamping any possible effect of the state of the host?

**Dr. Tomkins:** You have raised a very interesting point regarding what is actually going on in this society, which is a relatively affluent one. It is an urban African situation where we do not see marasmus or kwashiorkor but where there is a very high prevalence of diarrhea and fever and quite a marked seasonal change in nutritional status. In this chapter we are not dealing with anything to do with the origins of the acquired height of those children. Your question was: Are environmental factors so strong in this area that they override any immunity? In this society I think they do, because there was no relationship between these variables and the diarrheal incidence.
Dr. Waterlow: Not in Bangladesh?

Dr. Tomkins: Neither in Bangladesh. In Black's and in Chen's study, incidence was not increased in those who were short or even in those who were thin, but duration was. That is the point I am making.

Dr. Waterlow: This shows, then, that everybody is open to infection but that the underprivileged body is less able to get rid of it.

Dr. Tomkins: That is a very reasonable assumption. The point that I am making is that it is very easy to take a biological perspective and talk about clearance of enteropathogens, and it may well be right. We would really like to know what the diarrheal pathogen was, for how many days it was excreted in a short child, and for how many days it was excreted in a taller child. This would be a very interesting study to do. There are virtually no data that have rigorously analyzed this. However, children who are short are also short because they come from families with a variety of forms of social deprivation; often a child is not treated for dysentery with antibiotics because the mother has no time. We cannot assume that because a child is short, there is an immunological reason why he has got persistent diarrhea. We need a bigger framework in which the social interactions of the management of disease processes are addressed. I wish it were simple enough to think in pure biological terms. It would be so easy if we could just explain it in terms of shortness, immunity, and clearing of enteropathogens. However, it does not seem logical to do so in the face of the other confounding variables such as access to health services, use of antibiotics, etc.

Dr. Waterlow: My conclusion is that you need to find somewhere to carry out such a study, where there are perhaps fewer confounding variables. I don't remember exactly what Trowbridge found in his Haiti study.

Dr. Tomkins: I think you are referring to the analysis that was published in the American Journal of Clinical Nutrition (1) on the relationship between anthropometry and diarrheal prevalence. It is difficult to use that study as a reference point because he used retrospective morbidity data. He asked the children's mothers if they had been ill in the 1, 2, and 3 months before the measurement. All the studies emphasize the need for very regular and reliable morbidity measurements. In the study you referred to there was no account of the social constraints on maternal management of illness, and this is important. When one reviews the literature, one may see either some very clear biological explanation or some rather loose anthropological explanation but never an analysis of the interactions between the two. Your question is absolutely valid. However, I do not believe that these variables have been adequately controlled in any study that has been carried out. What you are asking for is an incredibly difficult study because you need to control about 30-odd variables of social behavior.

Dr. Waterlow: I would have thought that you and Dr. Nabarro could have answered that question from your seasonal data, if analyzed in the appropriate way.

Dr. Nabarro: It seems that there would be enormous problems to be faced if the outcome of the stunting and illness association were to be examined using cohort studies. An alternative approach is to use case-control methods, to examine children with infections of varying degrees of severity, and to study their antecedent weight-for-height status. We have used this when trying to study relationships between severe measles and nutritional status in Nepalese children.

Dr. Tomkins: A case-control study is extremely valuable. I didn't give you the results of the case-control study we did of mortality in relation to preceeding nutrition and social variables. It does help with the numerous problems that one has. The sample size, for instance, to be able to do these things would otherwise be quite enormous.
Dr. Mukherjee: What was the incidence of tuberculosis in the group that you studied?

Dr. Tomkins: Three children had tuberculosis on clinical diagnosis. Tuberculosis is not something that you can look at in relationship to stunting, as the prevalence is too low in a population of this size. It is an important public health problem, but the group in which it was diagnosed was too small to show any relationship between nutrition and tuberculosis.

To me, a short child is not only a child who is a different immunological subject but is also a child who is at increased risk of some of the variables affecting the outcome. That was why I analyzed the data in such a way.

Dr. Tanner: Just to give you some data about adults that appeared in a supplement of the *Acta Medica Scandinavica* last year (2), in 1954, 2 million Norwegians compulsorily had a mass mini-X-ray, and somebody had the bright idea of taking their heights and weights. Since then some 150,000 have died, and so now we have mortality rates for a substantial population. There is a close relationship between height and the incidence of death in the 30s, 40s, and 50s, and 60s; it gets less at 70, and it disappears at 80 because then you die however tall you are.

This is the only study I know of that indicates that in the long run taller people survive longer. It doesn’t, however, sort out the factors concerned, and I agree totally with what you said along those lines. But in the long run, height does seem to affect health. Growth is perhaps the sole and best bioassay of health.

Dr. Tomkins: Was there any control for the socioenvironmental background of those people who were shorter as opposed to taller?

Dr. Tanner: No, not in the publication, but I think there is a lot more to come.

REFERENCES
