

## Age-Specific Determinants of Stunting in Filipino Children<sup>1,2,3</sup>

Linda S. Adair<sup>4</sup> and David K. Guilkey

Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, NC 27516

**ABSTRACT** This study identifies age-specific factors related to new cases of stunting that develop in Filipino children from birth to 24 mo of age. Data come from nearly 3000 participants in the Cebu Longitudinal Health and Nutrition Survey, a community-based study conducted from 1983 to 1995. Length, morbidity, feeding and health-related data were collected bimonthly during home visits. Stunting (length  $>2$  SD below the WHO age- and sex-specific medians) occurred in 69% of rural and 60% of urban children by 24 mo of age. We used a multivariate discrete time hazard model to estimate the likelihood of becoming stunted in each 2-mo interval. The likelihood of stunting was significantly increased by diarrhea, febrile respiratory infections, early supplemental feeding and low birth weight. The effect of birth weight was strongest in the first year. Breast-feeding, preventive health care and taller maternal stature significantly decreased the likelihood of stunting. Males were more likely to become stunted in the first year, whereas females were more likely to become stunted in the second year of life. Because stunting is strongly related to poor functional outcomes such as impaired intellectual development during childhood, and to short stature in adulthood, these results emphasize the need for prevention of growth retardation through promotion of prenatal care and breast-feeding, as well as control of infectious diseases. *J. Nutr.* 127: 314–320, 1997.

**KEY WORDS:** • *stunting* • *linear growth retardation* • *human infants* • *developing countries* • *infant feeding*

Stunting is widespread among children living in environments characterized by poverty, poor nutrition and a high prevalence of infectious disease. Stunting is an important public health problem in developing countries because of its association during childhood with poor functional outcomes such as impaired cognitive development (Pollitt et al. 1995 and Brown and Pollitt 1996), increased susceptibility to infection (Tomkins 1988) and increased risk of mortality (Bairagi et al. 1985, Chen et al. 1980). Long-term consequences of childhood stunting include short stature, reduced work capacity (Spurr 1988) and elevated risk of poor reproductive outcomes. Recent research also suggests that stunting in childhood may increase later risk of obesity and chronic diseases (Barker 1994).

Most stunting occurs in the first 2–3 y of life. Although there may be some biological potential for catch-up growth through adolescence, especially when development is delayed, few opportunities for catch-up exist among children who remain in the poor environments that caused them to become stunted in the first place. Thus, once children have become stunted, they are likely to remain stunted into adulthood (Martorell et al. 1994). It is therefore imperative to understand the causes of stunting early in infancy and childhood, so that preventive measures can be taken.

Waterlow (1994) and Allen and Uauy (1994) emphasized that despite numerous studies, relatively little is known about the causes and mechanisms of stunting. There are strong associations between poverty and stunting; however, relatively few longitudinal studies have identified more proximate causal factors related to poverty. Furthermore, effects of such determinants at different ages have not been identified. The weaning period of 6–12 mo is assumed to be a particularly vulnerable time, but growth retardation continues among toddlers as well. Previous studies have examined the prevalence of stunting at different ages, but typically do not account for the fact that a child who is stunted at 24 mo of age, for example, was likely to have become stunted at a much earlier age. Thus age-specific etiology is not identified.

We used prospective data collected bimonthly on a large sample of children in Cebu, Philippines, to study stunting in the first 2 y of life. The highly detailed longitudinal data allowed us to make new and valuable contributions to our understanding of stunting through the following approaches: 1) examination of the incidence rather than prevalence of stunting, 2) modeling of the effects of age-specific proximate determinants of stunting, and 3) the use of longitudinal, multivariate statistical methods that correct for biases inherent in much of the previous work on stunting.

### SUBJECTS AND METHODS

**Sample and data collection.** The study was conducted in Metro Cebu, the area surrounding the second largest city in the Philippines. The survey area included the densely populated urban neighborhoods and squatter settlements of the central city, surrounding periurban neighborhoods, and rural communities in the nearby mountains and small islands. Following a census, 33 of the 243 barangays (administra-

<sup>1</sup> Presented in part at the International Congress of Dietetics, February 1996, Manila, Philippines, and Experimental Biology 96, April 1996, Washington DC [Adair, L. S. (1996) Causes of stunting in Filipino children. *FASEB J.* 10: A230 (abs.)].

<sup>2</sup> Supported by National Institutes of Health RO1 HD19983A and the Thrasher Research Fund.

<sup>3</sup> The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 USC section 1734 solely to indicate this fact.

<sup>4</sup> To whom correspondence should be addressed.

tive units) of Metro Cebu were randomly selected. The initial cohort included all pregnant women from the selected barangays who gave birth during a 1-y period from 1983 to 1984. There were 3080 single live births in the cohort; the infants were followed bimonthly for 24 mo, then revisited in 1991 and 1995. Infant weight and length were measured during home visits at birth and subsequently by trained interviewers using regularly calibrated equipment, standard measurement techniques and periodic checks for interobserver reliability. All sociodemographic and health-related data were based on detailed interviews with the mother or primary caretaker. Information on infant feeding included frequency and intensity of breast-feeding, amounts of all foods and liquids given in the past 24 h and general feeding pattern 7 d prior to the survey. Morbidity data reflected the mothers' reports of episodes of diarrhea, respiratory symptoms and fever in the past week. The research protocol was reviewed and approved by the University of North Carolina School of Public Health Institutional Review Board for Research Involving Human Subjects.

**Variables.** *Dependent variable.* Stunting is defined as recumbent length more than 2 *sd* below the age and sex-specific WHO/NCHS median. Although the 2 *sd* cutoff point is somewhat arbitrary, it is widely used in comparing children around the world, and this level of growth retardation is known to be associated with increased risk for a variety of health conditions and behavioral deficits (Waterlow 1988). We identified the interval during which a child's length Z-score first dropped below  $-2$ . We restricted our focus to new cases of stunting, because we found in previous work that once a child becomes stunted, he or she is highly likely to remain stunted. This simplifying assumption ignores catch-up growth and recurrence of stunting, but allows us to explore age-specific determinants of the first incidence of stunting. We treated stunting as a discrete event, but recognized that it is ultimately the culmination of a longer process of growth retardation. Information about the timing of slowed growth in children who become stunted is presented in the Results section. Furthermore, children who are not identified as stunted under this method may have experienced comparable levels of growth retardation, but do not end up with Z-scores below  $-2$ . Finally, in a small number of cases, "becoming stunted" may reflect only a small decline in Z-score (e.g., from  $-1.9$  to  $-2.1$ ); for others, the change may represent more substantial declines. For incident cases of stunting, the mean change in length-for-age Z-score during a 2-mo interval ranged from  $-1.25$  from birth to 2 mo, to  $-0.58$  from 10–12 mo, to  $-0.35$  from 22–24 mo, suggesting that incident stunting represents a substantial recent decline in Z-score.

*Independent variables.* We assessed the contribution to the likelihood of stunting of a set of fixed factors (not varying with time) including mother's height to partially control for genetic factors, sex of the child, place of residence (urban or rural), and birth weight. More importantly, we included time-varying variables representing exposures in the interval prior to the measurement of stunting. Use of appropriately lagged variables strengthened our ability to infer causal relationships rather than to simply identify associations. The measured exposures included whether the infant was still breast-feeding, energy intake (per kg) from foods and liquids other than breast milk, use of preventive health care (visits to health care providers for well-baby care and immunizations), febrile respiratory infections, diarrhea and season. Morbidity data represented the mothers' reports of infant illness in the 7 d prior to each interview.<sup>5</sup> Age interaction terms allowed us to determine whether the effects of these exposures varied with age of the child. Based on preliminary empirical work with the data and to simplify the interpretation of results, age interaction terms were specified in 6-mo blocks. We tested for the presence of significant age interactions with all of the independent variables. Nonsignificant interaction terms were dropped from the final model, except to show the full range of effects of a single variable over the full 2 y. For example, in the case of birth weight, we assessed whether there was a persistent effect or whether the effect was lessened as the child became older by interacting birth weight with each 6-mo block,

using 18–24 mo as the omitted category. Further information about the independent variables and their interrelationships can be found in earlier work on the Cebu study (Adair and Popkin 1996, Adair et al. 1993, Briscoe et al. 1990, Cebu Study Team 1991 and 1992).

**Statistical methods.** We modeled the incidence of stunting using a structural discrete time hazard equation estimated simultaneously with reduced form equations for endogenous independent variables. Although a straightforward hazard framework is commonly used for analysis of mortality, the method of correction for bias associated with endogeneity was developed only recently and applied to estimation of mortality in the Cebu sample by Guilkey and Riphahn (1996). To our knowledge, this approach has not been applied to growth data before.

In the hazard framework, once a child becomes stunted, he or she is censored, and subsequent recovery and relapse are ignored.<sup>6</sup> We estimated the log odds that a child would become stunted during each 2-mo interval, conditional on not having been stunted at the previous time period (2 mo earlier), as a function of the set of independent variables described above. We used a discrete time framework because data were obtained on the children at bimonthly intervals, and it is not possible to ascertain the exact day on which a child became stunted. The analysis sample consisted of 2859 infants with length data at 2 mo of age. We began our analysis of stunting at 2 mo of age because the determinants of stunting at birth (intrauterine factors) are not comparable to those (such as feeding and morbidity) that the child encounters in the postnatal environment. However, the child's status at birth is represented in the model by birth weight.

The estimation of the model for stunting is likely to present a set of noteworthy statistical problems. Repeated length measures of each child are highly intercorrelated. Furthermore, there are underlying characteristics of the child which we cannot observe or measure, but which affect the likelihood that the child will become stunted. These characteristics, referred to as unobserved heterogeneity, reflect a child's genetic potential or resistance to disease, and represent complexities which our model had to address.

Biases are likely to occur when the unobserved factors related to stunting are also associated with the independent variables in the model. For example, the innate healthiness or frailty of the child may influence how the mother chooses to feed the child and how much care she takes to avoid exposure to pathogens. If this is true, then the feeding and morbidity variables could be correlated with the unobserved factors that also influence stunting, that is, they are endogenous to the model. Unobserved heterogeneity confounds the relationship of stunting with feeding or diarrhea, potentially leading to a biased estimate of their effects (see Briscoe et al. 1990, Cebu Study Team 1991, VanDerslice et al. 1994 for examples and further discussion of the consequences of ignoring endogeneity). It is not possible to control for the effects of this potential confounding by including the confounder in the model, because in this case, the confounder is not observed.

One approach taken by economists to correct for endogeneity involves joint estimation of the main equation of interest (stunting) and equations for potentially endogenous independent variables (in this case, birth weight, feeding, health care and morbidity variables). The equations for the endogenous variables are specified only with exogenous household, community and individual variables, and are estimated using methods appropriate to their form (logit for categorical variables and linear regression for continuous variables). Correction for unobserved heterogeneity was achieved using the Heckman and Singer (1984) approach. Full details of this method and its application to the estimation of mortality in the same sample are presented in Guilkey and Riphahn (1996).

Once the stunting model was estimated, we simulated effects of different common scenarios on the likelihood of stunting. We calculated the cumulative predicted probability of stunting for the following: 1) males vs. females, 2) low birth weight (<2500 g) vs. normal birth weight infants, and 3) selected morbidity-feeding combinations.

<sup>5</sup> Because our statistical model incorporated instruments (predicted values) for morbidity, we actually assessed the effects of a child's usual morbidity experience over the entire 2-mo period.

<sup>6</sup> Recovery from stunting is more common in the first 6 mo of life than during the second year. For example, although about 20% of infants stunted at 4 mo are not stunted at 6 mo, after 1 y, fewer than 10% of infants recover in any 2-mo period. The number of recovering infants never exceeded 95 in any interval.

TABLE 1

Sociodemographic characteristics of CLHNS infants who formed the analysis sample<sup>1</sup>

		Percentage		
Child's household				
Urban				75.8
Has electricity				79.4
Has piped water inside				6.9
		Mean	SD	Range
Child's mother				
Height, cm	150.6	5.0		134.4–169.2
Education, y	7.06	3.3		0–17
Age, y	26.15	6.0		14–45
Child				
Birth weight, g	2996	425		907–4800
Birth length, cm	49.25	2.13		40.1–59.0
		Proportion		
% Male				53.0
% LBW <sup>2</sup>				11.5
% Breast-fed at 2 mo				84.2
% Breast-fed at 12 mo				61.7

<sup>1</sup>  $n = 2859$  infants.

<sup>2</sup> LBW, low birth weight.

Because inappropriate feeding practices and high morbidity tend to occur together, we developed risk profiles based on feeding and morbidity profiles characteristic of some communities of Cebu. The “high morbidity” profile is represented by the following: 1) a 50% chance of diarrhea and a 33% chance of febrile respiratory infections, levels which are about twice the mean prevalence rates in the sample; 2) inappropriate feeding, defined as not breast-feeding and consuming only 80% of the age-specific Filipino RDA for energy; and 3) no preventive health care. This set of conditions is common in urban squatter settlements of Cebu. In contrast, the “low morbidity” profile is represented by: 1) the mean prevalence of diarrhea among fully breast-fed infants for the first 4 mo of life (3% at 2 mo, and 6% at 4 mo) and a prevalence of 10% in the months thereafter, and a 7% prevalence of febrile respiratory infections, which is about half the mean prevalence in the population; 2) appropriate feeding which included full breast-feeding in the first 4 mo of life, followed by mixed feeding with energy intake from supplemental foods at levels estimated to meet the age-specific RDA; and 3) adequate levels of preventive health care. This profile, though infrequently seen in the Cebu sample, represents optimal health care and feeding practices, which in turn, would be associated with lower morbidity (Cebu Study Team 1991 and 1992).

## RESULTS

**Characteristics of the sample.** The health and demographic profile of the Cebu sample was similar to that of other developing areas of Asia. The sample was predominantly urban, but less than half of the households had electricity. Characteristics of the sample children, their mothers and their households are presented in Table 1. Short stature was common among the mothers, and it is impossible to know the extent to which this was a genetic characteristic vs. the outcome of the mothers' exposure to a growth-retarding environment during their childhood and adolescence. Low birth weight (LBW) occurred in 11.5% of Cebu infants, and the mean birth weight was about 3 kg. Stunting was observed in 31% of LBW infants. Among stunted LBW infants who sur-

vived to 2 mo, catch-up growth sufficient to bring length Z-score above  $-2$  sd was apparent in 57%.

The prevalence of stunting was very high among Cebu children, with rural children worse off than their urban counterparts. By 12 mo of age, 37.7% of rural and 35.1% of urban children were stunted. By 24 mo, 68.8% of rural and 61.9% of urban children were stunted. Overall, only about one third of sample children never had a length for age Z-score less than  $-2$  at any point during the first 2 y of life. Furthermore, the sample children were short relative to a representative national sample of over 25,000 Filipino children. For example, at 12 mo of age, about 17% of Cebu males and 10% of females were below the Filipino 5th percentile of length-for-age (Florentino et al. 1992). Stunting tends to be highly persistent. Of children stunted by 6 mo of age, 96% were also stunted at age 2.

The age distribution of new cases of stunting is presented in Figure 1. The peak in new cases occurred at 8 mo in males and females, with a decline, among males, in the number of new cases thereafter. During the first year, the number of new cases in males consistently exceeded that among females. During the second year, however, there were more new cases of stunting among females, with a peak at 16 mo of age.

In an attempt to pinpoint when linear growth retardation occurs relative to our measurement of incident stunting, we compared the length velocities of infants who became stunted in each 2-mo interval with those of infants who did not become stunted in that interval or earlier. Results are presented in Table 2. In each case, length velocities were lower for incident cases of stunting only in the interval immediately prior to our identification of stunting. For example, compared with infants not stunted up to age 8 mo, those whose first became stunted between 6 and 8 mo had comparable length velocities from birth to 6 mo, but dramatically lower velocity from 6 to 8 mo.

**The hazard model.** We obtained very robust, consistent results using the discrete time hazard model. We found evidence of a large effect of unobserved heterogeneity, emphasizing the importance of correcting for this factor in our models. The coefficients, standard errors and  $t$  statistics for the fully corrected model are presented in Table 3. A positive coefficient indicates that the variable increases the likelihood of stunting. Given the large number of interaction terms in the model, it is important to interpret coefficients by examining their net (main plus interacted) effects. The age interaction

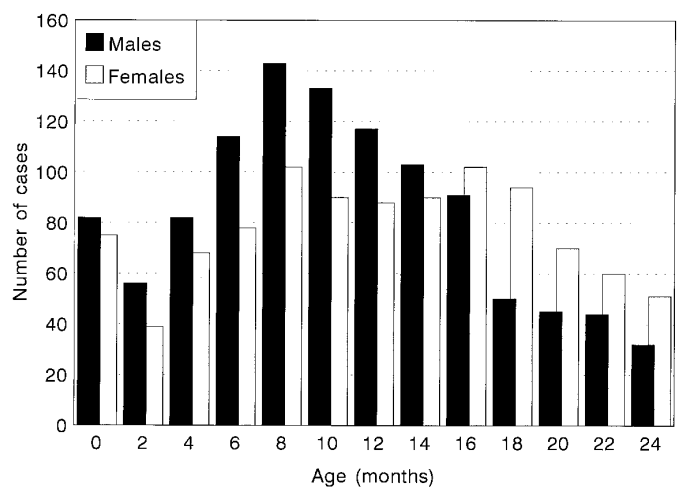


FIGURE 1 Distribution of new cases of stunting in male and female Cebu children, birth to 24 mo of age.

TABLE 2

Length velocities of Cebu infants, birth to 12 mo of age: a comparison of incident cases of stunting with infants not stunted by specified ages

Length velocity, <sup>1</sup> cm/d	n	Months					
		Birth-2	2-4	4-6	6-8	8-10	10-12
Stunted at 2 mo	95	0.072					
Not stunted at 2 mo <sup>2</sup>	2633	0.123					
Stunted at 4 mo	144	0.113	0.046				
Not stunted at 4 mo	2474	0.122	0.080				
Stunted at 6 mo	206	0.112	0.077	0.028			
Not stunted at 6 mo	2252	0.123	0.078	0.055			
Stunted at 8 mo	268	0.177	0.074	0.051	0.021		
Not stunted at 8 mo	1989	0.122	0.078	0.053	0.044		
Stunted at 10 mo	257	0.115	0.079	0.051	0.0412	0.016	
Not stunted at 10 mo	1770	0.122	0.078	0.053	0.042	0.036	
Stunted at 12 mo	262	0.120	0.076	0.055	0.040	0.035	0.013
Not stunted at 12 mo	1561	0.122	0.078	0.053	0.042	0.034	0.031

<sup>1</sup> Length velocity = [Length at time (t) – length at time (t – 1)]/interval in days from t to t1. The mean interval was 62 d.

<sup>2</sup> Not stunted at that point in time or prior to that point in time.

term describes the additional effect of a variable for a child of the age specified in each interaction term. For example, to interpret the effect of birth weight, we first noted that it has a significant negative main effect (as birth weight increases, the likelihood of stunting decreases). We then noted the large negative coefficient of the “birth weight by first 6 mo interaction term,” and concluded that there was an even larger effect of increasing birth weight on stunting in the first 6 mo than at any other age. We found significant age interactions with birth weight, sex of the infant, non-breast milk energy intake and breast-feeding.

The cumulative predicted probability of stunting calculated from the means of all independent variables, and under selected morbidity and feeding scenarios is graphed in Figures 2 and 3. Results are grouped into categories representing constitutional factors present at birth, morbidity, feeding and combined feeding-morbidity effects.

**Constitutional factors.** The likelihood of stunting was inversely related to mother’s height and infant birth weight. The effects of birth weight were greatest in the first 6 mo, then declined so that in the second year, birth weight no longer significantly increased the likelihood of stunting. Figure 2

TABLE 3

Coefficients, standard errors and t statistics associated with independent variables in the stunting hazard model<sup>1</sup>

Independent variable <sup>2</sup>	Coefficient	SE	t
Mother’s height, cm	-0.4009	0.0291	13.7668**
Sex of the infant <sup>3</sup> × birth-12 mo	0.1870	0.0339	5.5097**
Sex of the infant × birth-12 mo	-0.1519	0.0429	3.5443**
Birth weight, g	-0.2897	0.0460	6.2942**
Birth weight × birth-6 mo <sup>4</sup>	-0.7061	0.0791	8.9210**
Birth weight × 6-12 mo	-0.1199	0.0271	4.4273**
Birth weight × 12-18 mo	0.0184	0.0271	0.6784
Diarrhea	0.0988	0.0350	2.8179**
Febrile respiratory infection	0.1119	0.0367	3.0525**
Preventive health care	-0.1042	0.0445	2.3425**
Breast-fed	-0.1522	0.0551	2.7639**
Breast-fed × birth-6 mo	-0.1679	0.0978	1.7340*
Energy intake, <sup>5</sup> kJ/kg	-0.0353	0.0210	1.6816*
Energy intake × birth-6 mo	0.0970	0.0262	3.6970**
Energy intake × 6-12 mo	0.0665	0.0236	2.8144**
Energy intake × 12-18 mo	0.0050	0.0252	0.1991
Urban residence	-0.0781	0.0310	2.5166**
Season of measurement <sup>6</sup>	0.0613	0.0267	2.2973**
Time term (first 6 mo) <sup>4</sup>	1.4321	0.2485	5.7641**
Constant	5.9023	0.4549	12.9754**

<sup>1</sup> Dependent variable = incident stunting, n = 2859 individuals.

<sup>2</sup> Age interaction term coefficients should be interpreted as the additional effect of the designated variable in that time interval.

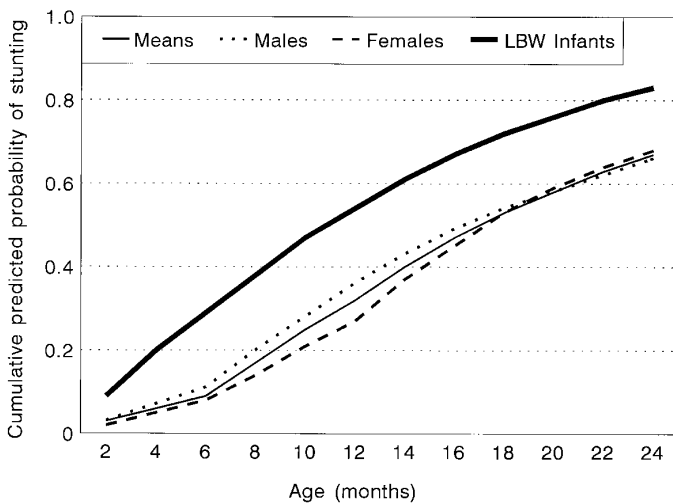
<sup>3</sup> Male = 1.

<sup>4</sup> Age of the infant was specified using dummy variables representing 6-mo blocks. When the infant is 0-6 mo old, the age “dummy” for that time period equals 1.

<sup>5</sup> Based on 24-hour dietary recall, energy intake from all foods and liquids EXCEPT breast-milk.

<sup>6</sup> Wet season (May-October) = 1.

\*P < 0.05; \*\*P < 0.01.



**FIGURE 2** Cumulative predicted probability of stunting among Cebu children. Results from simulations of the effects of sex of the infant and low birth weight (LBW) on the likelihood of stunting.

shows the estimated cumulative probability of stunting for low-birth-weight infants (<2500 g at birth) compared with normal weight infants. The likelihood of stunting was dramatically higher for low-birth-weight infants throughout the first 2 y.

The effect of the sex of the child was highly significant, with opposite signs on the coefficients for  $y_1$  and  $y_2$  of life. This is consistent with incidence data presented in Figure 1, and is also shown by the convergence in the likelihood curves estimated for males and females in Figure 2.

**Feeding.** Breast-fed infants had a reduced likelihood of becoming stunted. The protective effects of breast-feeding were most evident in the first 6 mo. There were also age-related effects of energy intake from supplemental foods. In the first year, the risk of stunting increased as intake increased. Higher intake from supplemental foods was associated with decreased levels of breast-feeding.

**Morbidity.** Diarrhea and febrile respiratory infections both significantly increased the likelihood of stunting. We found no significant morbidity-age interactions, suggesting that the effects of morbidity were similar in younger and older infants, despite the fact that diarrhea was more prevalent in the first year. Additional information on patterns of morbidity in the Cebu sample is presented in Adair, VanDerslice and Zohoori (1992), the Cebu Study Team (1991 and 1992) and Popkin et al. (1990). The magnitude of the effects of diarrhea and respiratory infections was quite similar.

Preventive health care protects infants from stunting. The major components of preventive care include maternal education, and immunization against diphtheria, pertussis and tetanus (DPT) and measles.

**Combined effects of morbidity and feeding.** Results from the two feeding-morbidity simulations are presented in Figure 3. Note the dramatic difference in the cumulative probability of stunting associated with these two profiles. For example, at 12 mo, the likelihood of stunting was 0.25 for the low morbidity, optimal feeding group, whereas it was twice as high (0.51) in the high morbidity, inappropriately fed group.

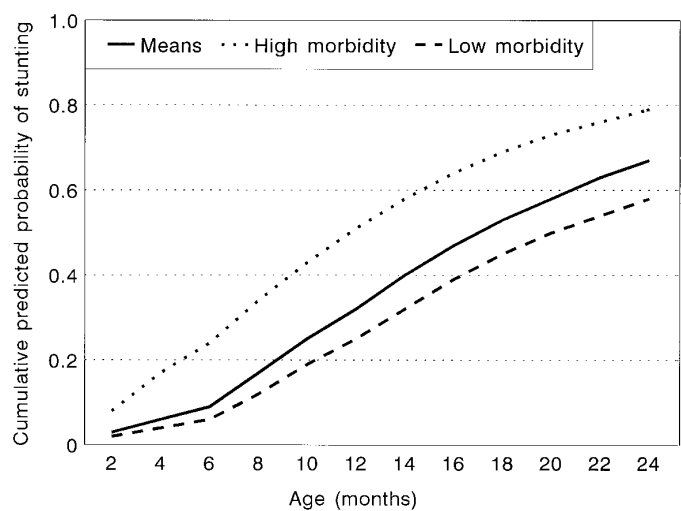
**Other factors.** Urban children were significantly less likely than rural children to become stunted. There was also a significant seasonality effect. If the measurement interval occurred during the wet season, the likelihood of stunting was significantly increased.

## DISCUSSION

We defined stunting relative to U.S. reference data and found it to be highly prevalent in Cebu children. There remains a controversy over whether U.S. reference data are appropriate for Asian children, because several authors concluded that Asian children have a different genetic potential for growth (Davies 1988, Ulijaszek 1994). If this is the case, one might argue that the Cebu children are following a population-specific normal trajectory, leading to shorter adult stature. However, the  $-2$  SD cutoff represents a deficit in length exceeding what might be expected based on between-population differences. Finally, our comparison of length velocities between cases of incident stunting and infants who did not become stunted by the same age strongly suggests that incident stunting is a short-term response to recent insults, rather than continuous slowed linear growth. This finding provides a solid rationale for examining incident stunting as a dependent variable, in spite of some of the limitations we have described. The profound effects of environment-related risk factors on stunting suggests that the Cebu children are not reaching their genetic potential for growth.

Second, we have singled out children whose process of growth retardation leaves them "stunted" and have ignored those children who may be experiencing growth retardation, but who do not end up with length-for-age Z scores less than  $-2$ . It is highly likely that the same growth-retarding influences affect infants of all starting sizes, and thus our results should be generalizable to less severe cases as well. However, this assumption will require empirical testing.

**Determinants of stunting.** Our model assessed the effects of factors manifested at birth, but that have persistent effects throughout childhood. These include mother's height, sex of the child and birth weight. We called these constitutional factors to differentiate them from the time-varying inputs such as morbidity and feeding that were measured every 2 mo in our study. In light of our finding that length velocity of infants with incident stunting was lower only in the 2 mo preceding our identification of stunting, we suggest that the constitutional factors set the limits of growth potential, and more acute insults such as morbidity or inadequate feeding produced the shorter-term decelerations in growth that resulted in stunting.



**FIGURE 3** Cumulative predicted probability of stunting among Cebu children. Results from simulations of the effects of high morbidity-poor feeding and low morbidity-optimal feeding on the likelihood of stunting.

Specific effects of sex of the infant, birth weight, feeding and morbidity are discussed in turn below.

**Constitutional factors.** Among well-nourished children, sex differences are attributed to a normal pattern of dimorphism, with males tending to be taller and heavier than females. The Cebu children showed less pronounced dimorphism in weight and height than the reference population. Males were more likely to become stunted in the first year of life, but females were more affected in the second year. Differences in unmeasured factors such as parental care-giving behaviors may partly account for this result. In previous work, we found that males are given supplemental foods earlier, are fed larger quantities of supplemental foods and have higher rates of diarrhea compared with females (Popkin et al. 1990). Our hazard model accounts for morbidity and energy intake, but other characteristics of the weaning diet such as protein and micronutrient content may be important. Popkin and Solon (1976) found sex differences in diet among Filipino children, with males having higher intakes of protein-rich foods. Females were given more vegetables which provided them with precursors of vitamin A, but fewer calories. Sex-related differences in diet and child care require further exploration, especially in light of the fact that in other Asian countries, females are more likely than males to be stunted.

The effects of birth weight on stunting were strongest in the first 6 mo, then diminished. This is consistent with the structure of the hazard model, and the observed persistence of stunting in the sample. Once an infant becomes stunted, he or she is censored, and makes no subsequent contribution to the estimates. LBW infants have an increased likelihood of becoming stunted early on, and are no longer in the sample "eligible" to become stunted at a later age. However, when we look at the cumulative probability of stunting, we see a strong persistent effect of LBW. This is consistent with earlier work, which presents length curves for LBW vs. normal weight infants in the first year of life, and shows significant differences in length at 12 mo among infants who were LBW vs. normal birth weight (Adair 1989). The initial low probability of stunting related to LBW (despite the high percentage of stunting among LBW infants at birth) occurs for several reasons. First, length velocities of LBW infants are significantly higher than normal weight infants (Adair 1989); thus many LBW infants stunted at birth are not stunted at 2 mo of age. Furthermore, the model examines the effects of LBW, controlling for a number of other factors also related to LBW. We recently showed significant effects of LBW on infant feeding patterns, with LBW infants less likely to initiate breast-feeding and more likely to be weaned early (Adair and Popkin 1996).

**Feeding and morbidity.** The present study shows a protective effect of breast-feeding, strongest in the first year, and a negative effect of energy intake from supplemental foods during the first year. Breast-feeding provides important nutrients needed for growth; it also protects against diarrheal morbidity through immune factors present in milk and reduced consumption of weaning foods potentially contaminated with pathogens. Among breast-fed infants, a higher energy intake from supplemental foods is related to a reduced level of breast-feeding. Thus, high quality nutrients from breast milk are replaced with lower quality nutrients from weaning diets based on corn or rice. Lack of breast-feeding, poor quality weaning foods and high levels of infectious disease typically occur together in poor environments. Their net effect in the Cebu children was seen in the dramatic difference in the cumulative probability of stunting among infants with the high morbidity-poor feeding profile, compared with those that were optimally fed and had a low prevalence of infectious disease.

Past literature has been quite consistent in showing a negative effect of diarrhea on growth (Adair et al. 1993, Black et al. 1982 and 1984). In addition to the effect of diarrhea, we also showed a strong effect of febrile respiratory infections on growth. Febrile infections may increase energy and nutrient needs, and suppress appetite, thus reducing growth. The lack of any significant age interactions with respiratory and diarrheal infections suggests that morbidity is equally important at all ages.

**Implications.** The persistence of stunting suggests that early interventions are vital. LBW is a highly significant risk factor for stunting, especially in the first 6 mo. Lack of breast-feeding and early introduction of weaning foods increases the risk of stunting in the early postnatal period. Thus, prevention of stunting requires prenatal interventions to optimize birth weight and promotion of full breast-feeding in the early postnatal months, followed by continued breast-feeding supplemented with high quality weaning foods. Throughout the first 2 y, diarrheal and respiratory infections increase the likelihood of stunting, and preventive health care has a protective effect. Effective strategies to reduce febrile respiratory infections are hard to define because they are very dependent on the person-to-person contact so typical in small dwellings with a high density of inhabitants found in Cebu. Other than the presence of smoky cooking fires in the home, we were unable to identify other modifiable factors related to respiratory infections (Cebu Study Team 1991 and 1992). In contrast, our earlier work with the Cebu data showed that diarrheal disease was increased when infants were not breast-fed, when water quality and excreta disposal were poor, and when weaning foods were prepared in an unhygienic manner (Cebu Study Team 1991 and 1992, VanDerslice et al. 1994). Given the importance of diarrhea as a determinant of stunting, effective interventions should include improvement of water quality and sanitation, coupled with promotion of breast-feeding.

## LITERATURE CITED

- Adair, L. S. (1989) Growth of Filipino infants who differ in body proportions at birth. *Am. J. Human Biol.* 1: 637-682.
- Adair, L. S., Popkin, B. M., VanDerslice, J., Akin, J. S., Guilkey, D. K., Black, R., Briscoe, J. & Fieger, W. (1993) Growth dynamics during the first two years: a prospective study in the Philippines. *Eur. J. Clin. Nutr.* 47: 42-51.
- Adair, L. S., VanDerslice, J., Zohoori, N. (1992) Urban-rural differences in determinants of growth and health of Filipino infants. In: *Urban Ecology and Health in the Third World* (Schell, L. M., Watts, E., Smith, M. & Bilsborough, A., eds.). Cambridge University Press, Cambridge, UK.
- Adair, L. S. & Popkin, B. M. (1996) Low birth weight reduces likelihood of breast-feeding among Filipino infants. *J. Nutr.* 126: 103-111.
- Allen, L. H. & Uauy, R. (1994) Guidelines for the study of mechanisms involved in the prevention or reversal of linear growth retardation in developing countries. *Eur. J. Clin. Nutr.* 48: S212-S216.
- Bairagi, R., Chowdhury, M. K., Kim, Y. J. & Curlin, G. T. (1985) Alternative anthropometric indicators of mortality. *Am. J. Clin. Nutr.* 42: 296-306.
- Barker, D.J.P. (1994) Mothers, babies and disease in later life. *B M Journal Publishing Group*, London, UK.
- Black, R. E., Brown, K. H. & Becker, S. (1982) Longitudinal studies of infectious diseases and physical growth of children in rural Bangladesh—patterns of morbidity. *Am. J. Epidemiol.* 115: 305-315.
- Black, R. E., Brown, K. H. & Becker, S. (1984) Effects of diarrhea associated with specific enteropathogens on the growth of children in rural Bangladesh. *Pediatrics* 73: 799-805.
- Briscoe, J., Akin, J. S. & Guilkey, D. K. (1990) People are not passive acceptors of threats to health: endogeneity and its consequences. *Int. J. Epidemiol.* 19: 147-153.
- Brown, J. L. & Pollitt, E. (1996) Malnutrition, poverty, and intellectual development. *Sci. Am.* 274: 38-43.
- Cebu Study Team (1991) Underlying and proximate determinants of child health: the Cebu longitudinal health and nutrition study. *Am. J. Epidemiol.* 133: 185-201.
- Cebu Study Team (1992) A child health production function estimated from longitudinal data. *J. Dev. Econ.* 38: 323-351.
- Chen, L. C., Chowdhury, A.K.M. & Huffman, S. L. (1980) Anthropometric assessment of energy-protein malnutrition and subsequent risk of mortality among preschool aged children. *Am. J. Clin. Nutr.* 33: 1836-1845.

- Davies, D. P. (1988) The importance of genetic influences on growth in early childhood with particular reference to children of Asiatic origin. In: *Linear Growth Retardation in Less Developed Countries* (Waterlow, J. C., ed.), Nestle Nutrition Workshop Series Volume 14. Raven Press, New York, NY.
- Florentino, R. F., Magbitang, J. A., Flores, E. G., Mendoza, T. S., Santos-Ocampo, P. D. (1992) FNRI-PPS anthropometric tables and charts for Filipino children. Food Nutr. Res. Inst. and Philippine Pediatr. Soc. Manila, Philippines.
- Guilkey, D.K.G. & Riphahn, R. (1996) The determinants of child mortality in the Philippines: estimation of a structural model. Working paper, Carolina Population Center, Chapel Hill, NC.
- Heckman, J. & Singer, B. (1984) A method for minimizing the impact of distributional assumptions in econometric models for duration data. *Econometrica* 52: 271–320.
- Martorell, R., Kettle Khan, L. & Schroeder, D. G. (1994) Reversibility of stunting: epidemiological findings in children from developing countries. *Eur. J. Clin. Nutr.* 48: S45–S57.
- Pollitt, E., Gorman, K. S., Engle, P. L., Rivera, J. A. & Martorell, R. (1995) Nutrition in early life and the fulfillment of intellectual potential. *J. Nutr.* 125: 1111S–1118S.
- Popkin, B. M., Adair, L. S., Akin, J. S., Black, R., Briscoe, J. & Fieger, W. (1990) Breast-feeding and diarrheal morbidity. *Pediatrics* 86: 874–882.
- Popkin, B. M. & Solon, F. S. (1976) Income, time, the working mother, and child nutrition. *J. Trop. Pediatr. Environ. Child Health* 11: 156–166.
- Spurr, G. B. (1988) Body size, physical work capacity, and productivity in hard work: is bigger better? In: *Linear Growth Retardation in Less Developed Countries* (Waterlow, J. C., ed.), Nestle Nutrition Workshop Series Volume 14. Raven Press, New York, NY.
- Tomkins, A. (1988) The risk of morbidity in a stunted child. In: *Linear Growth Retardation in Less Developed Countries*. (Waterlow, J. C., ed.), Nestle Nutrition Workshop Series Volume 14. Raven Press, New York, NY.
- Ulijaszek, S. (1994) Between population variation in pre-adolescent growth. *Eur. J. Clin. Nutr.* 48: S5–S10.
- VanDerslice, J., Popkin, B. M. & Briscoe, J. (1994) Drinking water quality, sanitation, and breast-feeding: their interactive effect on infant health. *Bull. WHO* 72: 589–601.
- Waterlow, J. C. (1988) *Linear Growth Retardation in Less Developed Countries*, Nestle Nutrition Workshop Series Volume 14. Raven Press, New York, NY.
- Waterlow, J. C. (1994) Summary of research needs in the area of linear growth retardation. *Eur. J. Clin. Nutr.* 48: S211.