The Relationship Between Child Anthropometry and Mortality in Developing Countries: Implications for Policy, Programs and Future Research[1]

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SUMMARY

The prevention of child mortality is a commonly stated health goal in developing countries and the target of much international assistance in the health sector. Over the past decade the primary strategy for accelerating the reduction in child mortality has been the dissemination of simple, low-cost technologies, such as immunization, oral rehydration therapy and antibiotics, that target specific diseases (Huffmann and Steel 1994). This is done despite the knowledge that malnutrition and disease have a synergistic relationship (Scrimshaw et al. 1968) and that the optimal strategy may involve a combination of health and nutrition interventions. In the 1970s, for instance, it was estimated that malnutrition (notably protein-energy malnutrition—PEM) was the underlying or contributing cause of death for roughly half of all deaths to children aged 1–4 years in several Latin American countries (Puffer and Serrano 1973). Apart from this early study, however, there has been little effort to quantify the contribution of malnutrition to child mortality in other regions of the world in ways which are meaningful to policy. This paper reviews the results of 28 community-based, prospective studies, in 12 Asian and Sub-Saharan African countries, which examined the relationship between anthropometric indicators of malnutrition and child mortality. One purpose is to estimate the contribution of malnutrition to child mortality—distinguishing the effects of severe malnutrition from mild-to-moderate malnutrition—and to examine a number of related issues relevant to policy, programs and research in this area.

The accumulated results are consistent in showing that the risk of mortality is inversely related to anthropometric indicators of nutritional status and that there is elevated risk even in the mild-to-moderate range of malnutrition. This latter result contradicts the findings from an earlier, landmark study which suggested that mild-to-moderate malnutrition was not associated with an increased risk of mortality (Chen et al. 1980). The present results indicate that somewhere between 20% and 75% of child deaths are statistically attributable to anthropometric deficits, with most estimates falling in the range 25–50%. When taking account of the relative proportions of severe versus mild-to-moderate malnutrition in the population, the results show further than 16–80% of all nutrition-related deaths are associated with mild-to-moderate malnutrition rather than severe malnutrition. In most studies 46–80% of all nutrition-related deaths are in the mild-to-moderate category. This represents the proportion of nutrition-related deaths that would be missed by policies and programs focusing primarily or exclusively on the severely malnourished, a bias that does exist in many public health programs in practice if not by design.

Another important result is the confirmation that malnutrition has a potentiating (multiplicative) effect on mortality within populations, as predicted from the theory of synergism. This means that malnutrition has its biggest impacts in populations with already high mortality levels and that morbidity has its biggest impacts in the most malnourished populations. This finding has far-reaching implications for child survival policy and programs, suggesting that greater attention should be paid to nutritional improvement than at present.

A potential limitation of the above conclusion is the possibility that the relationship between mortality

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and malnutrition may be confounded by behavioral and socioeconomic factors (e.g., caretaker knowledge and practices and access to health care). Several studies have addressed this question by controlling for confounders (through proxy socioeconomic variables), and these studies reveal that the anthropometry-mortality relationship is not due to such confounding. Usually this has been applied to severe cases of malnutrition, where the link to mortality is strongest, but one study indicated similar results among mild-to-moderate cases.

The results further suggest the possibility that, for a given anthropometric deficit, child mortality in South Asian children is lower than in children from other regions. This result is consistent with the findings from earlier cross-national comparisons (Haaga et al. 1985). It may relate to small maternal stature in these populations, which contributes to the exceptionally high rate of low birth weight (31%) and relatively benign carry-over effects on the size of preschool children. It is hypothesized that child anthropometric deficits arising in this fashion may not have the same functional consequences as deficits arising from poor maternal health and nutrition during pregnancy or from similar conditions in early childhood, an area requiring further research.

On the basis of a priori expectations as well as the results from the few studies examining these issues, the review concludes that the child anthropometry-mortality relationship is likely to be modified by a number of other factors, a result with important implications for policy and programs. Some of these factors include the age of the child, possibly sex of the child in some settings, length of follow-up after measurement, seasonality and breastfeeding. The policy importance is well illustrated by the breastfeeding results, which indicate that the elevated risk of death among severely malnourished children (>12 mo) is made even worse (four times worse) by the absence of breastfeeding. The programmatic importance of effect modifiers relates to such issues as deciding the optimal interval between measurements for screening purposes, choosing the most efficient anthropometric indicator and deciding the priority to be given to children of different ages when using a single screening indicator. Most studies have been limited in their ability to examine these issues due to small sample sizes, variation in analytical methodologies and failure to collect and/or use ancillary data in the analysis. However, future research should include consideration of these issues and may benefit from pooling the data from previous studies.

Finally, it is suggested that observational (i.e., non-intervention) studies of the type reviewed here may be inherently limited in their ability to answer a central policy question, namely the extent to which *reductions* in child mortality through health sector interventions may be compromised by persistently high rates of malnutrition. A stronger approach would be through careful evaluation of on-going, large-scale intervention programs, especially those that have successfully controlled severe malnutrition and are shifting attention to mild-to-moderate forms.

INTRODUCTION

The prevention of child mortality is perhaps the most commonly stated goal of national health policy in developing countries and is a major focus of international and bilateral aid in the health sector. This is reflected in the health plans of individual countries and by the increased attention in recent years to the promotion and/or dissemination of simple, low-cost technologies for child survival (Grant 1983, USAID/CDC 1992). Huffmann and Steel (1994) have reviewed the impact of these technologies on child survival and questioned whether nutrition has been given appropriate attention in the overall strategies. They note that some of the life-saving technologies have the potential to simultaneously improve nutrition and were originally designed to do so, but in the implementation process this objective was typically overlooked in favor of the more easily implemented components of the strategies. They note that this not only represents a missed opportunity to improve nutrition, but may also limit the effectiveness of the technologies on saving lives because of the importance of malnutrition as a contributor to infant and child mortality.

The issues raised by Huffmann and Steel are important for deciding the appropriate mix of strategies to promote child survival at a national and international level. While the argument might be made that governments and external agencies should strive to improve nutrition *simultaneous* with efforts to improve child survival, the reality of resource constraints means that decisions must always be made concerning the relative weight to be given to nutritional improvement versus technological interventions. UNICEF notes that expenditures on direct nutrition activities represented only 13% of the amount spent on other health activities in 1990 (Parker and Jesperson 1994). In order to evaluate the appropriateness of this resource allocation at an international level or within a given country the first step is to assess the quantitative contribution of malnutrition to mortality.

The report of the Inter-American Investigation of Mortality in Childhood (Puffer and Serrano 1973) was the first systematic attempt to estimate the contribution of malnutrition to child mortality in one region of the world. It suggested that 35% of all deaths to children <5 years of age involved malnutrition (severe or mild-to-moderate) as an underlying or contributing cause in 13 Latin American countries. The corresponding estimate for children aged 12–23 month was 60% and that for 2–4 year-olds was 54%.

Shortly after the Inter-American Investigation, the nutrition-mortality linkage received important confirmation in a small number of studies in India (Kielmann and McCord 1978) and Bangladesh (Chen et al. 1980; Sommer and Lowenstein 1975). These studies were conducted prospectively over a long period of time (months to years) and were population based, in contrast to earlier studies that were either retrospective, employed short follow-up periods and/or were based on hospital admissions (Garrow and Pike 1967; Gomez et al. 1956; McLaren et al. 1969; Puffer and Serrano 1973). These studies also found that relatively simple anthropometric indicators of nutritional status are related to the risk of future mortality. At a policy level this has confirmed the potential importance of malnutrition for designing child survival strategies and at a programmatic level it provides one of the rationales for using growth monitoring in health programs to assist in identifying high-risk children.

Although the studies cited above have provided important documentation that anthropometric indicators of malnutrition are related to mortality risk, a number of important questions remain. The purpose of this paper is to draw on the existing literature to address these issues to the extent possible. Specifically, this paper reviews the results of prospective, population-based studies to examine the following questions:

- Are there any differences in the relationship between anthropometry and mortality across populations?
- What fraction of child mortality is attributable to malnutrition as measured by anthropometric deficits?
- What is the contribution of mild-to-moderate malnutrition versus severe malnutrition to this fraction?
- Does the relationship between anthropometric indicators and mortality differ according to the age or sex of the child?
- To what extent are these relationships confounded or modified by other factors such as household socioeconomic status, concurrent morbidity and feeding practices?
- How are these relationships affected by the length of follow-up of the child after measurement?
- Which anthropometric indicators are most closely related to mortality and how do static (one-time) measures compare with repeated measures on the same child (velocities)?

The paper concludes by identifying the implications for policy, programs and future research.

CONCEPTUAL AND METHODOLOGICAL CONSIDERATIONS

The available studies differ in a number of respects which have an important bearing on their interpretation and comparability. The studies and some of these important features are described in **Table 1** and elaborated upon below. Before describing the studies it is important to note the distinction between nutritional status and anthropometric indicators of nutritional status. Although the terms are sometimes used interchangeably the distinction is important in the present review. Nutritional status refers to the internal state of the individual as it relates to the availability and utilization of energy and nutrients at the cellular level. Protein-energy nutritional status, therefore, refers to the state of the individual in relation to these two "nutrients." Because this state cannot be directly observed, one relies on measurable indicators of this state. Anthropometrics provide relatively simple and convenient indicators of protein-energy status, but they are not synonymous with it. For instance, a child may have a moderately low weight-for-age because of recent diarrheal dehydration (though being well fed), because he is stunted from nutritional insults some years ago (though currently well nourished) or because of improper feeding practices in recent months (though presently free of disease). Alternatively, the child may have some combination of these conditions. Each of these scenarios has different implications for the child's *current* nutritional status (viz. nutrient availability and utilization at cellular level) and for the probability of mortality in the coming 1 month, 6 months or 12 months. These scenarios also mean that, in various circumstances, the use of anthropometric indicators may lead to underestimates or overestimates of the effects of nutritional status on mortality. Thus, the present review is concerned with *anthropometry*-mortality relationships in the first instance and only through cautious inference does it discuss *nutrition*-mortality relationships. A more direct approach to estimating *nutrition*-mortality relationships, and the one most meaningful when estimating the nutritionally preventable fraction, is through intervention trials. Although fewer in number, these studies have been reviewed elsewhere (Rose and Martorell 1992).

As shown in Table 1 there are 28 reports available in the journal literature (in two cases supplemented with dissertations). These studies were identified through computer-based searches (Medline and Agricola) from January, 1983 through December, 1992 and through secondary citations given in all of these papers. This list is thought to be complete with respect to prospective, population-based studies comparing one or more anthropometric indicators to risk of subsequent mortality. It excludes hospital or clinic-based studies.

As shown in the table, some studies are included in the list more than once, when reanalysis of the same data provided additional information bearing on the objectives of this paper. Thus, the 28 reports actually

TABLE 1

Description of population-based studies included in this review

Study No.[1]	Population	Age range	Effective length of follow-up[2]	Effective no. of deaths[3]	No. of children[4]	Gross[5] mortality rate	Citation
		mo	*mo*				
1a	Matlab, Bangladesh	12–23	24	112/48	2019	27.7	Chen et al. 1980
1b	Matlab, Bangladesh	12–23	24	112/48	2019	27.7	Bairagi 1981
1c	Matlab, Bangladesh	12–23	24	112/48	2019	27.7	Chowdhury 1988
1d	Matlab, Bangladesh	12–23	24	98/39*	1998	27.7	Cogill 1982
2a	Matlab, Bangladesh	12–59	18	154/35	3757	27.3	Sommer and Lowenstein 1975
2b	Matlab, Bangladesh	12–59	18	154/35	3757	27.3	Trowbridge and Sommer 1981
2c	Matlab, Bangladesh	12–59	6	49*	922	N/A[6]	Briend and Zimicki 1986
3a	Matlab, Bangladesh	6–36	1	52/29	4927*	21.1	Briend et al. 1987
3b	Matlab, Bangladesh	12–36	1	49/28*	4612*	21.1	Briend et al. 1988
4	Matlab, Bangladesh	12–59	12	23/4	961	14.3	Bairagi et al. 1985
5	Teknaf, Bangladesh	12–59	6	60/16	2625*	5.1	Alam et al. 1989
6a	C-M Hwy, Bangladesh[7]	12–36	1	69/47*	1087*	31.7	Briend and Bari 1989a
6b	C-M Hwy, Bangladesh	0–36	1	66/47*	1011*	32.6	Briend and Bari 1989b
7	Punjab, India	12–36	12	148/26	2808	52.7	Kielmann and McCord 1978
8	W. Java, Indonesia	0–59	18	151/20	3461	43.6	Katz et al. 1989[8]
9	Papua New Guinea	6–30	18	47/4	1147	27.3	Heywood 1982
10	Kasongo, Zaire	6–59	3	105	7092	4.2	Kasongo Project Team 1983
11	Kasongo, Zaire	6-59	3	52	4273*	3.5	Kasongo Project Team 1986
12	Iringa, Tanzania	6–30	12	88/7	2452	35.9	Yambi 1988
13	Guinea-Bissau	6–59	12	109/17	2228*	48.9	Smedman et al. 1987
14	Senegal	6–59	6	301	3153*	47.8	Briend et al. 1989
15	S. Malawi	0–59	12	84/18	1178*	28.5	Lindskog et al. 1988
16	N. Malawi	6–59	12	84/8	2883	29.1	Pelletier et al. 1994
17	SW Uganda	0–59	12	104/12	4320	24.1	Vella et al 1994
18	NW Uganda	0–59	12	21/8	1178	17.8	Vella et al. 1992
19	Yemen	0–84	12	47/6	2071	22.7	Bagenholm and Nasher 1989
20	Matlab	6–36	24	412/139	16276	12.7	Fauveau et al. 1990
21	Zaire	0–72	30	246/30	5167	19.0	Van Den Broeck et al. 1993

[1] Studies in a series (e.g., 1a–1d) represent analysis of the same data set.

[2] Refers to maximum possible length of follow-up used in the main analysis. Some studies using child-months as the denominator in the analysis often did so because the actual length of follow-up varied among children, with children only measured once at the beginning of the period: studies 5, 13 and 15; others measured children regularly and examined mortality in the following month or period: studies 3a, 3b, 6a, 6b, 11 and 14.

[3] Refers to number actually used in the main analysis and the number in the "severe" category of the anthropometric indicator (when available). *Studies that reported exclusion of accidental deaths.

[4] * Asterisk refers to studies in which child-months (or child-semesters) are used as the unit of analysis, which in all cases is higher than the number of children shown here.

[5] Defined as effective no. deaths/effective no. children, expressed per 1,000 per year; note that the effective follow-up period often differed from the period over which these deaths occurred, in which case the rate is standardized relative to the latter. The rate is intended here as a crude measure of completeness of death reporting.

[6] These represent incomplete samples from previously archived data (study 2a) and thus a mortality rate cannot be calculated.

[7] Chandpur-Comilla Highway, study area 70 km from Dhaka. Studies 6a and 6b refer to the same basic data set, but the two papers disagree on the age range, and one excludes 3 drowning deaths and 73 other (unspecified) children.

[8] This source provides results on weight-for-height and height-for-age; results on weight-for-age were obtained through personal communication with this research group for the purpose of this paper.

refer to 21 separate studies, representing 12 distinct - world populations. There is a clear bias in the literature in favor of Bangladesh, for which 14 reports exist based on 7 different studies. There are 10 reports from Africa, representing studies in 6 countries, and no reports from Latin America.

The studies differ in the range of children's ages, with some having fairly restricted ranges and others covering the entire underfives period. Variation in age may have an important effect on study results because of the well-known changes in anthropometric indicators, feeding practices, disease exposure and health care that take place during the period from birth through 5 years (Leslie and Gupta 1989; Martorell and Habicht 1986). It is relevant to note, for instance, that the study that has been most widely cited and debated in the literature (Chen et al. 1980) and has undoubtedly had a significant impact on

policies and programs is also the one with the most restricted age range (12–23 months). The few studies available for explicitly examining this issue are reviewed in a later section.

Length of follow-up also varies widely across the studies, ranging from 1 to 30 months. This may also have a significant influence on the predictive ability of various indicators, because those reflecting wasting (e.g., weight-for-height, various arm circumference indicators and, at younger ages, weight-for-age) are indicative of an acute condition. This may predispose to death in a short follow-up interval but such indicators typically resume normal values within weeks or mo and should have little or no carry-over effects on mortality in the longer term. Other indicators (e.g., height-for-age) reflect a lifetime of chronically poor health and nutrient intake, with little relevance to the child's current nutritional status and would not be expected to contribute *directly* to mortality in older children. They may nonetheless have good predictive value if they identify children and households with chronically poor environmental and behavioral characteristics, which are themselves responsible for greater mortality. Thus, variation across studies should be expected in the ranking of indicators from best-to-worse as a result of the length of follow-up employed and ages of study children.

Another important factor in many of these studies is the relatively small numbers of deaths available for analysis. The relatively low prevalence of severe (<60% weight-for-age) malnutrition (2–10% in most studies) results in small numbers of deaths in the severe category (with 6 of the distinct studies having <10 deaths in this category). This suggests that the estimates of mortality rates within categories of nutritional status have a fairly wide confidence interval and that conclusions regarding the existence, or lack thereof, of threshold effects, population differences, etc. should be made and interpreted with caution.

As indicated in Table 1, a calculation of approximate mortality rates based on the data provided in each report identifies several studies with unusually low mortality estimates and a high probability of incomplete death registration. In most cases this results in misclassification of the unreported deaths as survivors (because the default in most studies was probably to assume they survived) and significant loss of power to distinguish the deceased and the survivors on the basis of anthropometry.

Another feature of the mortality data that varies among studies is the way in which accidental deaths are treated in the analysis. Several studies (noted in Table 1) excluded these from analysis, whereas others included them. As reflected by the discussion over this point in connection with Chen's study (Chen et al. 1981; Mosley 1981), this can have a significant impact on estimates of sensitivity, specificity and (to a lesser extent) attributable risk. The reports excluding accidental deaths as indicated in Table 1 represent only those in which explicit mention was made.

Table 1 shows that the studies differ in how they express and analyze mortality. Most reports provide sufficient information to permit the calculation of mortality rates (deaths per 1,000 children per year) according to various levels of anthropometric indicators. Others quantified mortality as the number of deaths per 1,000 child-month of observation, which was necessitated by the design of the study (variation in length of follow-up among children in the study). The latter method results in rates that are numerically lower than standard mortality rates and cannot be readily converted or compared to such rates. However, the estimates of anthropometry-mortality relationships and differences in prediction across indicators should be very similar for these two methods. The present report emphasizes studies with a fixed follow-up period, but the results for the other studies are broadly similar and have been described elsewhere (Pelletier 1991).

Finally, it is important to recognize that the prospective studies included in this analysis cannot, by themselves, provide evidence of causality between malnutrition and child mortality. Strictly speaking, they can only provide evidence of association. It is possible, and indeed likely, that child malnutrition and mortality may cluster in the same households as a result of socioeconomic and behavioral factors that cause malnutrition *and* mortality. When such confounding is operating, the observed, bivariate association between malnutrition and mortality would tend to *overestimate* the strength of the actual relationship between the two. A substantial portion of this review is devoted to examining these potential confounding effects. The results of these analyses should be considered in light of evidence from other sources (community interventions clinical studies, and knowledge of biological mechanisms) to draw causal inferences. Thus, it is important to note that terms like "nutrition-related deaths," "population attributable risk," "the effects of malnutrition" and others used in this report are subject to these qualifications concerning causality versus association.

RESULTS

Anthropometry-mortality relationships and population attributable risk

Figures 1–4 illustrate the relationship between mortality and four indicators of nutritional status:

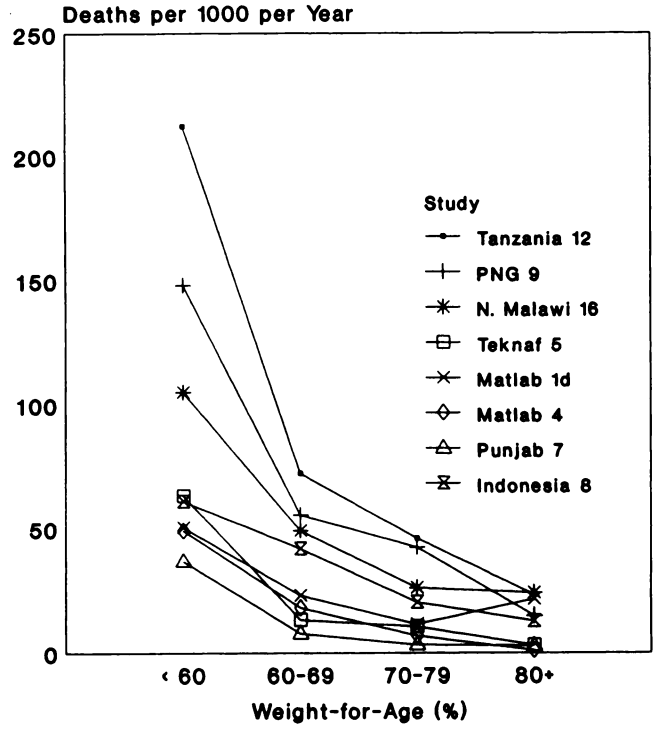


FIGURE 1 Relationship between child mortality and weight-for-age as a percentage of international median. Adapted from Yambi (1988), Heywood (1982), Pelletier et al. (1994a), Alam et al. (1989), Cogill (1982), Bairagi et al. (1985), Kielmann and McCord (1978) and Katz et al. (1989). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

weight-for-age (WA)[2], simple arm circumference (AC), weight-for-height (WH) and height-for-age (HA). These rates were either taken directly from the literature or calculated from data provided in the original reports and, in a few cases, estimated from figures in these reports.[3]

Figure 1 shows a consistent increase in child mortality with decreasing WA in all eight studies for which standard rates could be calculated. Mortality shows a basically linear increase as WA declines from 80% of median to 60% of median and a marked increase as WA declines below 60% of median. Similar results are seen in the studies expressing mortality relative to child-mo (Pelletier 1991).

The one notable exception is from Matlab, Bangladesh (study 1d), in which there is no consistent relationship between mortality and WA at levels >60–69%.[4] This study, which was one of the first to appear in the literature on this subject, suggested the existence of a threshold of WA (65% of median in the original report) above which there was no elevation in mortality risk. It is apparent from Figure 1 that this result has not been confirmed by subsequent results in the same study area (Bairagi et al. 1985), elsewhere in Bangladesh (Alam et al. 1989) nor in other populations such as India (Kielmann and McCord 1978), Indonesia (Katz et al., personal communication), Papua New Guinea (Heywood 1982), Guinea-Bissau (Smedman et al. 1987), Tanzania (Yambi 1988) and Malawi (Pelletier et al. 1994a). Although the gradient in mortality risk is greatest below 60% WA, these other studies demonstrate a consistent gradient even at levels above this point.

Given the far higher prevalence of mild-to-moderate malnutrition (60–80% WA) as compared to severe, this result has important policy implications as emphasized by the estimates of population attributable risk (PAR) provided in **Table 2.**[5] The PAR associated with total malnutrition (defined as WA < 80% of median) ranges from a low of 19% in N. Malawi to a high of 73% in one Bangladesh study,[6] with most other studies falling in the range 21–48%. Table 2 also reveals that mild-to-moderate malnutrition (60–80% WA) represents 46% to 93% of the total PAR across studies (except the Chen study). This represents a range of estimates of the percent of nutrition-related mortality that would be missed by

[2] Abbreviations used: AC, arm circumference; ACA, arm circumference-for-age; AC/HT, arm circumference/height; ACHT, arm circumference-for-height; AC/A, arm circumference/age; ARI, AIDS-related illness; d_n, normalized distance statistics; HA = height-for-age; MMM, mild-to-moderate malnutrition; MSS, maximum sum of sensitivity and specificity; ORS, oral rehydration solution; PAR, population attributable risk; PEM, protein-energy malnutrition; RR, relative risk; RRm, relative risk due to malnutrition; ROC, receiver operating characteristic; SES, socioeconomic status; WA, weight-for-age; WH, weight-for-height; W/H, weight/height; W/H^2, weight/height^2.

[3] These figures and many of the results that follow employ the "percent of median" classification system for anthropometry, rather than the Z-score system, because this review is based on the data as reported in the published papers.

[4] Note that the rates shown here by four categories of WA are based on data provided by Cogill (1982), which exclude accidental deaths. It differs from the original report (Chen et al., 1980), which used the Gomez classification and included all deaths. The form of the relationship is the same in the two versions, but the analysis of data in Cogill enhances cross-study comparability and is used in this and subsequent results.

[5] Note that with the exception of Cogill (1982) none of the WA reports shown in Tables 2 and 3 mention excluding accidental deaths. The re-analysis of Chen's data from that provided in Cogill (1982) was by necessity based on nonaccidental deaths; however, at a cutoff point of WA < 70% this only raises the PAR from 38% (with accidents) to 44% (without accidents).

[6] The Guinea-Bissau study suffered from the confounding effect of a measles epidemic during the follow-up period, which affected the urban portion of the sample where nutritional status is better than in the rural samples (90% versus 84% WA). Thus, the risk attributable to WA is likely to be an underestimate. The Bangladesh study with PAR = 73.5% had a follow-up period of only 6 mo, compared with 12–24 mos for all other studies. Similarly, the study with the next highest PAR (63.0%, based only on severe protein-energy malnutrition (PEM) also from Bangladesh), had a follow-up period of only 1 mo. Because most deaths to malnourished children occur in the first few mo after measurement, a longer follow-up period would probably have resulted in a narrower range of PAR estimates.

TABLE 2

Estimates of population attributable risk for severe and mild-to-moderate protein-energy malnutrition (PEM) in relation to child mortality[1,2]

Population and study	Severe PEM			Mild-to-Moderate			Normal		Total PAR	PAR percent MMM
	MR	Prev.	PAR	MR	Prev.	PAR	MR	Prev.		
Weight-for-age[3]										
India (7)	36.7	4.2	26.1	4.8	61.2	22.5	2.8	34.6	48.6	46.2
Bangladesh (5)	63.2	5.1	25.2	11.6	70.2	48.5	3.2	24.7	73.7	65.8
Bangladesh (1d)	50.4	19.4	19.9	23.1	69.0	3.9[a]	21.5	11.6	23.8	16.5
Papua New Guinea (9)	148.1	1.6	7.8	44.2	35.5	38.1	14.8	62.9	45.9	83.0
Tanzania (12)	212.1	1.3	6.9	51.8	35.4	28.3	23.2	63.3	35.2	80.5
N. Malawi (16)	105.3	2.6	7.3	32.5	38.4	11.7	23.6	59.0	19.0	61.7
Indonesia (8)	61.0	1.4	3.0	38.4	35.2	40.4	12.7	63.5	43.4	93.0
Arm circumference[4]										
Bangladesh (5)	73.4	6.1	24.9	13.8	76.4	32.0	7.0	17.5	56.9	56.2
Bangladesh (1d)	104.7	5.3	19.1	26.3	43.9	19.4	15.3	50.8	38.5	50.5
Bangladesh (2c)	113.3	4.0	33.1	16.2	29.5	24.0	5.6	66.5	57.0	42.1
N. Malawi (16)	181.8	1.2	6.7	40.4	18.9	11.6	23.1	79.6	18.3	63.2
S.W. Uganda (17)	187.5	2.5	16.7	45.5	13.7	15.2	17.3	81.3	31.9	47.6
Weight-for-height[5]										
Bangladesh (1d)	29.8	24.4	7.6	23.2	48.4	2.0	22.2	27.1	9.5	20.7
Bangladesh (5)	30.0	7.4	11.7	10.6	40.4	0.0	10.8	52.2	11.0	0.0
Indonesia (8)	71.1	2.0	5.8	31.4	20.0	17.1	14.9	78.0	22.9	74.6
PNG (9)	148.1	1.6	7.5	44.5	19.6	17.1	20.6	78.8	24.6	69.7
Tanzania (12)	97.8	3.8	7.6	61.9	21.4	21.5	25.6	74.8	29.1	73.9
N. Malawi (16)	61	4.2	5.9	31.0	17.9	5.0	23.6	77.9	10.9	45.8
S.W. Uganda (17)	84.5	3.8	9.6	40.0	15.3	12.0	20.0	81.0	21.6	55.5
N.W. Uganda (18)	125.0	3.0	8.5	54.3	17.3	13.5	27.1	79.7	22.0	61.6
Yemen (19)	36.4	7.7	12.7	15.2	33.7	7.5	11.9	58.6	20.1	37.1
Height-for age[6]										
Bangladesh (1a)	56.6	23.2	33.5	19.4	67.8	7.1	16.5	9.0	40.6	17.5
Bangladesh (5)	30.7	15.2	29.4	10.4	42.8	11.3	7.2	42.0	40.7	27.7
Indonesia (8)	38.6	10.0	4.8	49.7	54.1	41.5	20.5	35.9	46.2	89.7
Papua New Guinea (9)	72.7	4.8	7.2	41.3	73.9	49.1	16.4	21.3	56.3	87.2
Tanzania (12)	56.8	3.6	2.8	37.2	72.3	17.1	28.7	24.1	20.0	85.9
N. Malawi (16)	52.9	13.0	16.4	24.8	64.0	14.2	18.8	23.1	30.6	46.4

[1] Adapted from Kielmann and McCord (1978), Alam et al. (1989), Cogill (1982), Heywood (1982), Yambi (1988), Pelletier et al. (1994a), Katz et al. (1989), Briend and Zimicki (1986), Vella et al. (1994), Vella et al. (1992), Bagenholm and Nasher (1989) and Chen et al. (1980). Study descriptions are provided in Table 1.

[2] MR = mortality rate per 1,000 children per year associated with each grade of PEM; PAR = population attributable risk of mortality associated with each grade of PEM; Prev. = percent of children in each grade of PEM; MMM = mild-to-moderate malnutrition; PAR = $[P_i(RR_i - 1)]/([P_i(RR_i - 1)] + 1)$, where P_i is the prevalence of malnutrition in a given grade and RR_i is the relative risk of mortality in the grade compared to those in the "normal" grade.

[3] Severe, <60% weight-for-age; mild-to-moderate, 60–79% weight-for-age; normal, >80% weight-for-age.

[a] The PAR is set at 0 because the MR for this grade is lower than the reference grade of PEM; total PAR is assumed to equal PAR for severe PEM.

[4] Severe, <110 mm; mild-to-moderate, 110–129 mm; normal, ≥130 mm (studies 1d, 2c). Severe, <121 mm; mild-to-moderate, 121–131 mm; normal, >130 mm (study 5, 16, 17).

[5] Severe, <80% or <−2 Z; mild to moderate, 80–89% or −1 Z; normal, ≥90% or ≥−1 Z.

[6] Severe, <85% height-for-age; mild-to-moderate, 85–94% height-for-age; normal, ≥95% height-for-age (except study 5 where mild-to-moderate is 85–89%; and, normal ≥ 90%.

nutrition programs if exclusive or primary attention were to be given to severe cases.[7]

Two notable exceptions exist in the literature concerning mortality and anthropometric indicators, both from Zaire. One is a study from Kasongo, Zaire (Kasongo Project Team 1983), which found no relationship between the two. This study generated some discussion concerning the possible population-specific nature of this relationship (Bairagi 1981, Chen et al. 1981). However, with the benefit of additional studies

[7] Note that PAR is used here simply to assess the extent to which malnutrition-related deaths are to be found in different grades of malnutrition. It is not a valid statistic for comparing across indicators because its magnitude is greatly influenced by the prevalence and, thus, the location of the cutoff point. More valid methods to choose among several indictors are described in a later section.

from Africa since that time,[8] it appears that methodological problems in the study may be a more likely explanation. For instance, the original study emphasized that the data, manpower and techniques used in the study are typical of what might be expected in a normal health clinic in Zaire. In addition, Table 1 shows that the 105 deaths analyzed in the study probably represent at most 20% of total deaths occurring in the sample during this period, because the gross mortality rate calculated from the report is only 4.2/1,000/year. Thus, this study by itself does not support the suggestion that the fundamental relationship between anthropometry and mortality differs in Sub-Saharan African populations.[9]

The second exception comes from Bwamanda, Zaire (Van Den Broeck et al. 1993). This study found no association between anthropometric indicators of nutritional status and mortality when extreme malnutrition cases are excluded (defined as kwashiorkor or extreme marasmus based on clinical diagnosis). This study does not appear to suffer from the methodological problems found in the Kasongo study. However, the authors note that the study area has been the target of an intensive integrated development project for the past 20 years, such that the population experiences less diarrheal burden, has high immunization rates and has access to effective curative health care. Under such circumstances, relatively rare in the developing world, it appears that the association between mild-to-moderate malnutrition and mortality may be attenuated. The authors further suggest that endemic malaria and severe anemia may have comparable case fatality rates across different grades of nutritional status. Because these are the dominant causes of mortality in this population, this would further attenuate the association between mild-to-moderate malnutrition and mortality.

Although the same fundamental relationship between anthropometry and mortality is seen in all populations studied, the results in Figure 1 give the appearance of possible population differences in the effects of malnutrition on mortality. Thus, Tanzania Papua New Guinea and N. Malawi have higher mortality rates than the four South Asian populations and Indonesia at any given WA, but especially for WA < 60%. This does not appear to be consistently accounted for by differential length of follow-up or age ranges, which are both intermediate between those of various South Asian studies (Table 1). The observation that (over the entire range of weight-for-age) mortality in the three African studies appears to be elevated relative to the South Asian rates is consistent with the clear trend observed along these lines in cross-national comparisons of data from 22 countries (Haaga et al. 1985). The differential response (slope) of mortality on malnutrition suggested here is taken up in a later section.

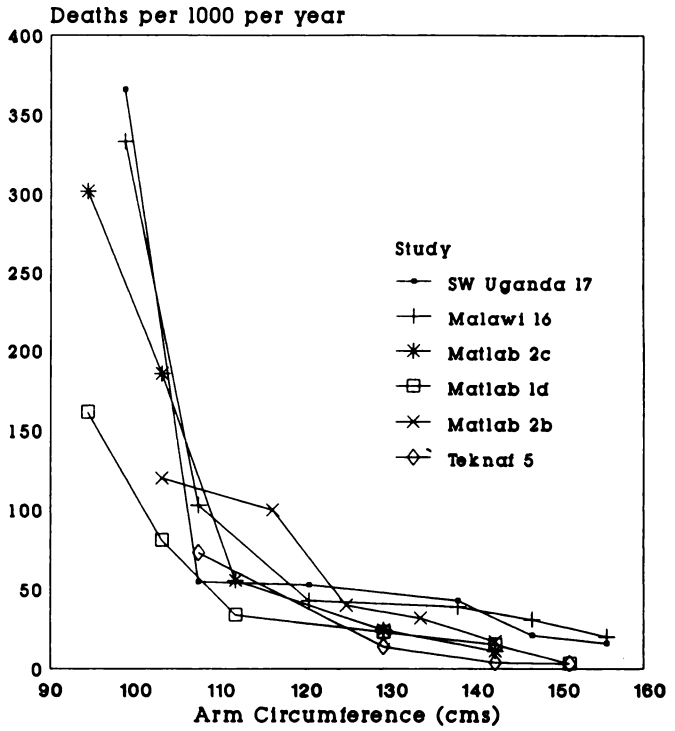


FIGURE 2 Relationship between child mortality and mid-to-upper arm circumference. Adapted from Vella et al. (1994), Pelletier et al. (1993), Briend and Zimicki (1986), Cogill (1982), Trowbridge and Sommer (1981) and Alam et al. (1989). Study descriptions are provided in Table 1.

Figure 2 shows the relationship between simple arm circumference and subsequent mortality. As with WA, mortality shows an exponential increase with declining AC, with a small but detectable elevation in risk at intermediate levels of AC (110–129 mm) and a marked elevation at the extreme levels (<110 mm). There appears to be far less interstudy variation than seen with WA. Because the length of follow-up varies from 6 to 24 months across studies, two studies excluded accidental deaths and two did not, and the measurements were taken under different conditions by different field teams, the similarity of results across these studies can be taken as an indirect indication of the robustness of the overall relationship. It is notable that the two African studies are similar to those from Bangladesh, unlike the divergence seen in WA (above).

The estimates of PAR for arm circumference (Table 2) vary from 18% to 57%. This is within the range of estimates based on weight-for-age. The fraction at-

[8] In addition to the studies represented in Figure 1 and Table 2, there are studies from Senegal, Guinea-Bissau and S. Malawi confirming the basic relationship, but all based on child-mos as the unit of observations.

[9] Due to the virtual absence of any statistically significant relationship in the Kasongo study, its methodological problems and, most importantly, the noncomparable method of presentation in the original reports (internal standards and no cell-specific sample sizes), this study is not considered further in this section.

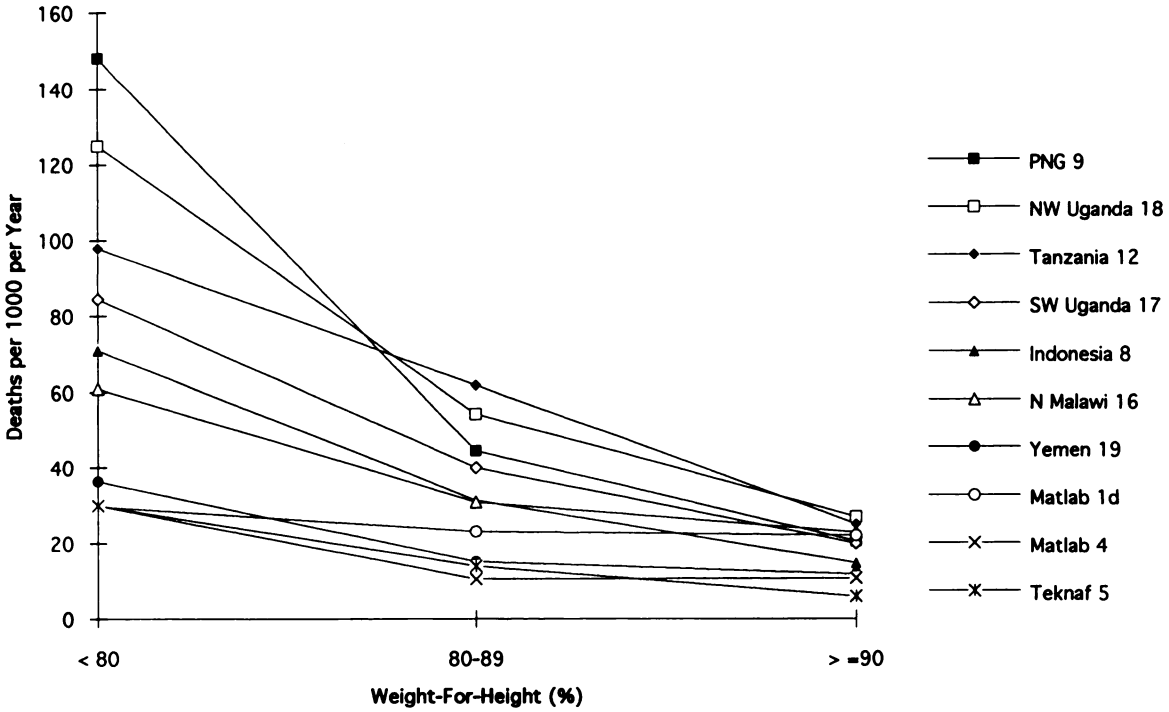


FIGURE 3 Relationship between child mortality and weight-for-height as a percentage of international median. Adapted from Heywood (1982), Vella et al. (1992), Yambi (1988), Vella et al. (1994), Katz et al. (1989), Pelletier et al. (1993), Bagenholm and Nasher (1989), Coghill (1982), Bairagi et al. (1985) and Alam et al. (1989). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

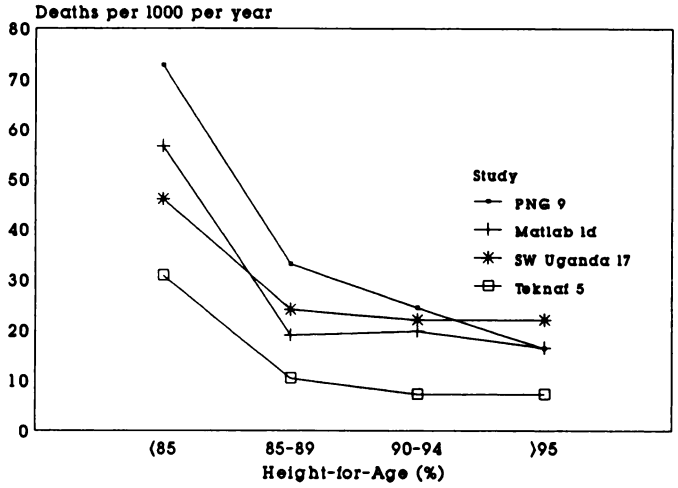
tributable to mild-to-moderate AC deficits (as defined here, 110–129 mm) is 42–63%. These percentages, of course, are highly sensitive to the choice of cutoff points delimiting these categories and largely reflect the cutoff points employed in the original reports.

Figure 3 shows the relationship between mortality and weight-for-height in the 10 studies with comparable methods. Most studies show the same basic pattern seen in the other anthropometric indicators, with a modest elevation in mortality among the mild-to-moderately malnourished and a more marked elevation among the severely malnourished. The only exception is Tanzania, which shows a linear increase across the entire range. This may reflect the effect of the Iringa Nutrition Program operating in the study area, which has been shown to reduce the prevalence of severe malnutrition and, by inference, mortality among the severely malnourished (GOT 1988). Table 2 shows that the total PAR for WH ranges from 10% to 29%. Mild-to-moderate malnutrition accounts for a small proportion of the total PAR in the two Bangladesh studies (0–20%), compared with the proportion seen in the other countries (39–75%). To a large extent this is due to the fact that the ratio of mild-to-moderate malnutrition (MMM) to severe malnutrition is far higher in the samples outside of Bangladesh.

Figure 4 shows the relationship between mortality and height-for-age in the eight studies for which adequate information was available for analysis. In four of the studies (top panel) the relationship is broadly similar to that seen for weight-for-age (Fig. 1), with modest rises in mortality risk in the mild-to-moderate range and a steep increase in risk below 85% HA.[10] However, four studies depart from this pattern (bottom panel), as do S. Malawi (Lindskog et al. 1988) and Guinea-Bissau (Smedman et al. 1987), which are based on child-month as the unit of observation and therefore not shown here (cf. Pelletier 1991). The reasons for these deviant results for HA in many studies are discussed below.

The Guinea-Bissau study was confounded by a measles epidemic that affected the urban (better-nourished) portion of the sample, thereby artificially elevating mortality among those with high values of HA. The southern Malawi study reported a significant association between HA and mortality (with the relative risk gradually rising to 4.99 in the most extreme HA category), but only after statistically adjusting for child's age and study period (a mixed cohort study over 2 years). The Indonesia study likewise found a significant association between HA and mortality; however, this is only found after controlling for weight-for-height, a step that was not necessary in the other studies shown in Figure 4. In the Tanzanian study the reason for the aberrant results at the extremes of the HA

[10] Note that lower mortality at all levels of HA found in Alam et al. (1989) reflects likely under-registration of deaths (or at least under-linkage of death records with anthropometry survey records); see last column of Table 1.



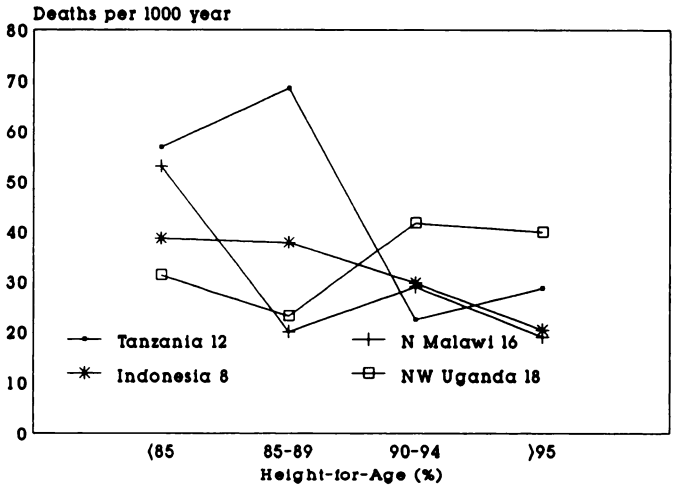


FIGURE 4 Relationship between child mortality and height-for-age as a percentage of international median. Adapted from Heywood (1982), Coghill (1982), Vella et al. (1994), Alam et al. (1989), Yambi (1988), Katz et al. (1989), Pelletier et al. (1993) and Vella et al. (1992). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

distribution appears to be small sample sizes at the lower end of the distribution (only 5 deaths among 88 children below HA < 85%) and confounding by age at the higher end (Yambi 1988).[11] Despite these aberrations at the extremes of HA in Tanzania, there is a significant difference ($P = 0.003$) in mean HA between all survivors and all deceased (Yambi et al. 1991).

As discussed by Yambi et al. (1991) the irregular, often U-shaped, response of mortality to HA has been found in a number of studies and may be due to confounding by age. This suggestion is supported by a reanalysis of results from northwest Uganda (Vella et al. 1992) as shown in **Table 3.** When all ages are combined, mortality shows a U-shaped response to HA, with the highest rate (41/1,000) actually being found in the least-stunted group. However, when the results are stratified by age, the expected negative association between mortality and HA is observed among those aged 0–11 months and 12–23 months. A U-shaped pattern persists among those older than 23 months. Inspection of the cell-specific sample sizes reveals that infants comprise 32% of the nonstunted children, 15% of the moderately stunted children, and only 7% of the severely stunted children. By comparison, the 23–59 month olds comprise 49%, 59% and 69% of these three HA categories, respectively. If the three categories of HA had identical age structure, the U-shaped pattern would be replaced by a curvilinear pattern as reflected in the age-adjusted rates in the table.

Thus, although the relationship between HA and mortality is less consistent across studies, each of the studies with aberrant results contain plausible explanations and/or have conducted additional analyses that support the existence of a significant relationship. It is noteworthy that the four studies with the weakest HA-mortality relationship (Guinea-Bissau, S. Malawi, N. W. Uganda and Indonesia) cover a wide age range (0/6 to 59 mo) and that the latter three report a significant relationship *after* age and/or weight-for-height are controlled. This reinforces the notion that there is likely to be some age-specificity as well as age confounding in the relationship between mortality and anthropometric indicators.

As shown in Table 2, the PAR estimates for HA range from 20% to 56%. Mild-to-moderate stunting accounts for 17–28% of the total PAR in the two Bangladesh studies, compared with 47–90% among the other populations. This reflects the fact that

[11] Many of the deaths to children with HA > 95% were below 18 mos, when stunting is not yet prevalent and were accompanied by illness and vomiting.

TABLE 3

***Effect of age confounding in the relationship between mortality (per 1000 per year) and height-for-age*[1,2]**

Child's age	Height-for-age Z-score: HAZ < −3 Mortality rate	HAZ −3 to −2 Mortality rate	HAZ ≥ −2 Mortality rate	Total *n*
	%	%	%	
0–11	231 (13)	105 (38)	90 (199)	250
12–23	43 (47)	29 (69)	9 (115)	231
>23	8 (132)	0 (153)	20 (300)	585
All ages	31 (192)	23 (260)	41 (614)	1066
Age adjusted	68 —	31 —	34 —	—

[1] Calculated from Vella et al. (1992). Values in parentheses are number of subjects.

[2] HAZ = height for age Z-score.

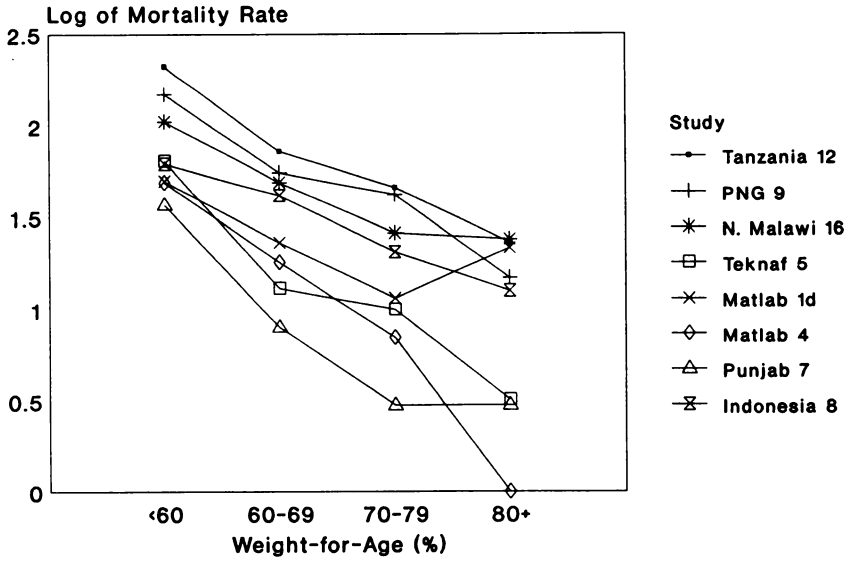


FIGURE 5 Relationship between log of mortality and weight-for-age. Adapted from Yambi (1988), Heywood (1982), Pelletier et al. (1994a), Alam et al. (1989), Cogill (1982), Bairagi et al. (1985) and Katz et al. (1989). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

in Bangladesh the mortality-HA relationship is stronger and extreme stunting relatively prevalent, whereas in the other populations the ratio of mild-to-moderate stunting to severe stunting is far higher.

The potentiating effect of malnutrition on mortality

One of the most important findings from the cross-study compilation of results is that the slope of mor-

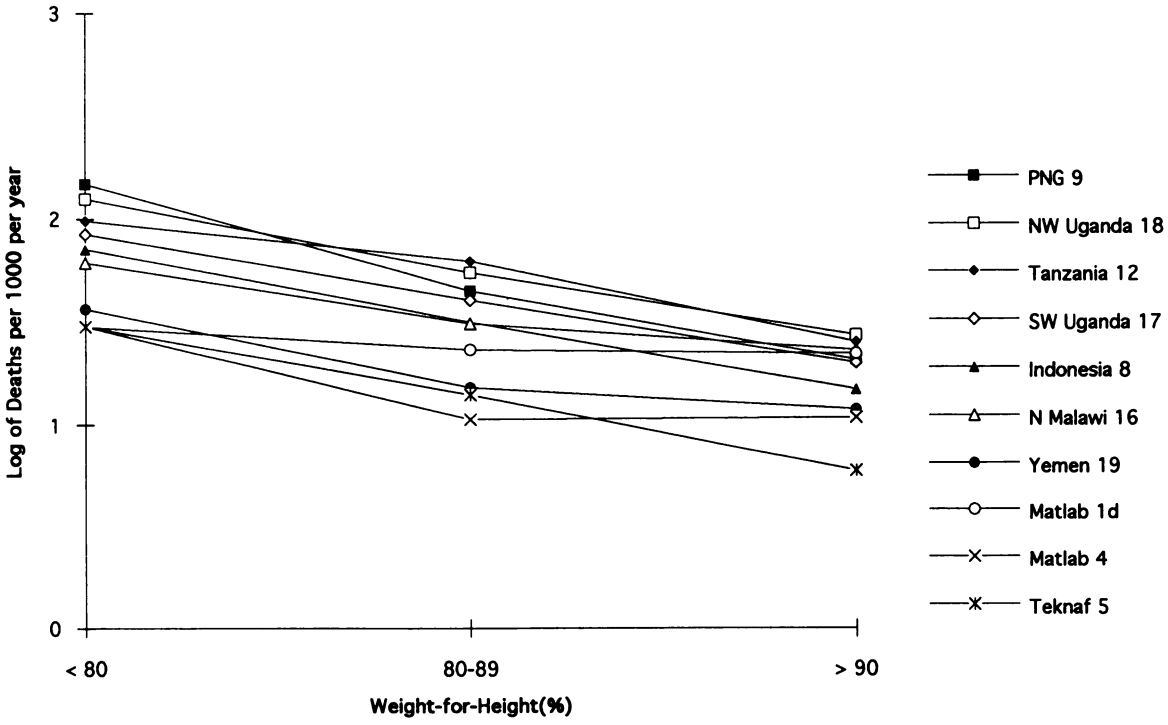


FIGURE 6 Relationship between log of mortality and weight-for-height as a percentage of international median. Adapted from Heywood (1982), Vella et al. (1992), Yambi (1988), Vella et al. (1994), Katz et al. (1989), Pelletier et al. (1994a), Bagenholm and Nasher (1989), Cogill (1982), Bairagi et al. (1985) and Alam et al. (1989). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

tality on malnutrition in a given population is positively related to the "baseline" levels of mortality as observed among the well nourished. This is illustrated with WA and WH in **Figures 5** and **6,** respectively, which use a log transformation of the mortality rates. As described in detail elsewhere (Pelletier et al. 1993), this finding confirms that malnutrition has a *potentiating* (i.e., multiplicative) effect on mortality in a population, rather than an additive effect. Specifically, populations with high levels of baseline mortality experience a quantitatively larger increase in mortality for a given prevalence of malnutrition than do populations with low baseline levels of mortality. Put another way, if these associations are causal, an increase or decrease in the prevalence of malnutrition will have a bigger impact on mortality in populations with already high mortality levels than in those populations with low mortality levels. Efforts to lower child mortality would therefore be most effective if attention is given to improving health and nutritional status simultaneously and if such efforts are targeted towards populations with the highest mortality levels.

Another implication of this multiplicative effect of malnutrition is that epidemiologic parameters such as sensitivity, specificity and attributable risk are not constants to be applied across populations; the quantitative impact of malnutrition on mortality (as described by these parameters) will vary according to the prevailing burden of morbidity, and the impact of morbidity (as a whole or for specific diseases) will vary according to the prevalence of malnutrition. However, if the PAR estimates are interpreted in multiplicative (potentiating) terms rather than additive terms, it is possible to use the PAR estimates as an input into policy and planning decisions (Pelletier et al. 1994b).

One of the questions raised by these findings is the extent to which the relative risk of mortality due to malnutrition (RRm) varies according to the type of morbidity present in a given population. In other words, does malnutrition interact more or less equally with all common forms of morbidity, or is its effect stronger for some types of morbidity than others? Biological considerations would lead to the prediction that malnutrition has differential effects across different forms of morbidity, and one of the Zaire studies provides evidence to this effect (Van Den Broeck et al. 1993). If RRm does vary significantly by cause of death, then estimation of the PAR due to malnutrition would need to take account of the prevailing morbidity patterns and their response to malnutrition. Three studies in the literature provide some direct information relevant to this issue, as summarized in **Table 4.**

As shown, the studies vary somewhat in the categories used for cause of death and in their definitions of malnutrition, which limits interpretation somewhat. Moreover, one of the Matlab studies (Fauveau et al. 1990) defined malnutrition based on visible wasting or recent weight loss just before death and confirmed in a separate analysis that this roughly corresponds to an arm circumference < 110 mm. With these caveats, the results reveal that malnutrition potentiates the risk of death due to diarrhea, measles, fever, AIDS-related illnesses (ARI) and other infections. In the Uganda study, with the most detailed cause of death categories, the estimated RRm is weakest for ARI. The category "other infections" in the two Matlab studies probably includes ARI and febrile illness, among others, because these causes are not mentioned separately.

TABLE 4

***Relative risk of death due to malnutrition, by cause of death*[1]**

Cause of death	Study					
	Matlab (1a)[2] 12–23 mo WA	Matlab (20)[3] 6–36 mo Wasting	SW Uganda (17)[4] 6–59 mo			
			WA	WH	AC	HA
Diarrhea	3.7	16.8	7.1	3.8	7.5	1.4
Measles	2.3	4.2	4.6	8.6	4.9	1.2
Fever	—	—	7.3	3.4	8.2	4.7
ARI	—	—	1.9	1.9	9.4	2.3
Other Infections	7.0	2.1	—	—	—	—
Accidents	1.2	—	—	—	—	—
Other	4.1	1.2	1.5	0.9	0.7	1.7
All Causes	3.2	8.0	3.7	4.0	5.5	2.0

[1] Malnutrition defined as follows: Matlab study 1a: WA < 65%; Matlab study 20: visible wasting (approximately AC < 110 mm); SW Uganda Study 17: WA < −2.5 Z; WH < −1.5 Z; AC < 12.5 mm; HA, −3 Z. WA = weight-for-age; AC = arm circumference; HA = height-for-age; ARI = AIDS-related illness.

[2] Adapted from Chen et al. (1980).

[3] Adapted from Fauveau et al. (1990). This study defined malnutrition based on the presence/absence of visible wasting just before death and/or recent weight loss. It differs from the others, therefore, which used prospectively measured anthropometry to define malnutrition.

[4] Adapted from Vella et al. (1994).

Table 4 reveals several sources of variability in RRm estimates. These include cause of death, study and anthropometric indicator (the latter seen in the Uganda study). It is likely that child's age, anthropometric cutoff point and patterns of health care availability and utilization (specific to each symptom) also contribute to variation in RRm within and between populations. The evidence shown in Table 4 is not sufficient to quantify these effects. In particular, because of small sample sizes and variability in cause of death ascertainment, it does not provide a firm basis for testing whether the cause-specific RRm estimates are uniform and, therefore, applicable across populations with varying morbidity patterns. Unfortunately, the Zaire study by Van Den Broeck et al. (1993) did not provide results that would allow the RRm to be cal-

culated by cause of death. Such an analysis would permit a test of their hypothesis that the malnutrition-mortality association is attenuated in the case of malaria and severe anemia.

In contrast to the variability in results shown in Table 4, one of the implications of the results shown in Figures 5 and 6 is that RRm is, in fact, surprisingly uniform across studies. Analysis of the data in Figure 1 suggests a 5.9% compounded rate of increase in mortality for each percent decline in WA, with a standard error of 0.8% (Pelletier et al. 1994b). This constancy in RRm is rather surprising in light of the marked variation in environmental conditions and types of morbidity across these studies. For instance, the Papua New Guinea study took place in the highlands, where the author noted that ARI is the major cause of death (Heywood 1982); by contrast, ARI would be expected to assume lower *relative* importance in the three African populations (Tanzania, Uganda and Malawi) because of the importance of malaria and diarrhea in these environments (Feachem and Jamison 1991). In light of the larger sample sizes underlying Figure 5 and the more convincing evidence they provide concerning the constancy across the eight studies, the analysis related to Figures 5 and 6 represents rather firm empirical evidence that RRm is more or less constant across populations, at least over the range of environmental conditions represented in these eight studies. This is a rather surprising finding but one that is compelling on empirical grounds.

Effect modification and confounding

One of the questions that the early studies on this subject did not adequately address is the extent to which the observed anthropometry-mortality relationships either vary according to other factors (effect modification) or are actually accounted for by other factors (confounding). A simplified model for describing the causal relationships is shown in **Figure 7.** Of particular importance to the present discussion is the observation that child age, sex, socioeconomic status (SES) and seasonality may all affect child mortality through one or more of the following pathways: 1) energy/nutrient availability as affected by intake and body reserves; 2) disease exposure; 3) immune status and 4) treatment of illness. All of these pathways are expected to affect child anthropometry and mortality to some extent. It is theoretically possible, therefore, that some or all of the association between child anthropometry and mortality may be due to confounding by the distal factors (age/sex, SES and seasonality), which may create anthropometric deficits through one pathway (e.g., energy/nutrient availability) and mortality through other pathways (e.g., low immunization rates or inappropriate treatment and management of

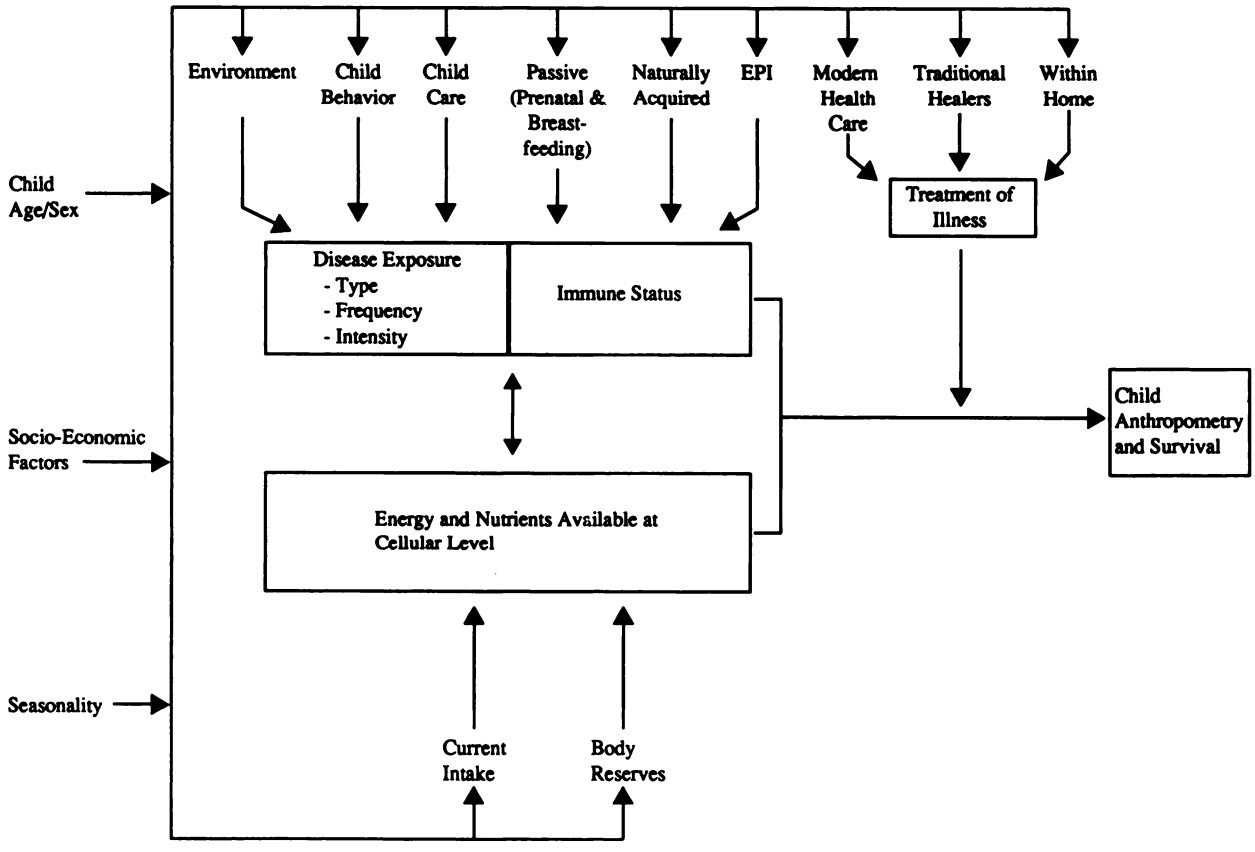


FIGURE 7 Biobehavioral determinants of child anthropometry and survival.

illness). This may be less relevant when the interest is in anthropometry as a screening tool, but one of the important policy implications is that the PAR estimates provided above may be grossly inflated by confounding. If this is the case, then reduction in child mortality may not be as dependent upon nutritional improvement (with all the implied multisectoral complexities) as the above PAR estimates would suggest.

In addition to its relevance for potential confounding, the causal framework shown in Figure 7 suggests the mechanisms that might underlie effect modification. Effect modification in this case would express itself as a stronger association between child anthropometry and mortality in some population groups than others (e.g., younger vs. older children or high SES vs. low SES). If effect modification is observed, it may indicate that one of the four proximal pathways is particularly strong in some subgroups (e.g., neonatal tetanus) or that multiple pathways are acting simultaneously in that group (e.g., weaning-aged children). Knowledge of effect modification is important for policy planning in order to identify the most vulnerable population groups and in order to design interventions that address the dominant proximal factors responsible for mortality in those groups. This information is also important from a programmatic perspective because it affects the efficiency of anthropometric screening.

Age effects. Most of the studies that considered the effects of age in the analysis conceptualized it as a potentially confounding main effect rather than as an interaction or effect-modifier. Thus, in 10 of the studies, age was entered into a logistic regression equation before examination of the effects of anthropometric indicators. This statistically accounts for the tendency for mortality rates to be lower among older children (among whom WA and HA are often lower as well), but does not allow for an examination of the extent to which the effect of the indicator itself varies with the age of the child. As suggested in Figure 7, all of the determinants of child anthropometry and mortality are known to vary with child's age, though not in a uniform fashion, thereby creating the possibility that various anthropometric indicators may have a stronger relationship to mortality at certain ages. Four studies provide sufficient information to permit an examination of relative risks and PARs by age group, and two others provided graphic evidence bearing on age effects. These are reviewed below.

Figure 8 shows the relative risk (RR) of 1-year mortality in Punjab, India according to the degree of deficit in WA and initial age of the child (where WA ≥ 80% is the reference group). The supporting statistics and related calculations are shown in **Table 5.** The relative risk of mortality peaks in the interval 6–12 months in the two most underweight groups (<60% WA and 60–69% WA), but the relative risk for moderately underweight children (70–79% WA) has no such peak. Between 6 and 12 months, severely underweight children are nearly twenty times more likely to die within 1 year than the reference group (≥80% WA), a relative risk not equalled in any other study. In all three underweight groups, the relative risk is much lower in the 12–36-month interval than in the 6–12-month interval. Thus, for WA the Punjab study suggests particular vulnerability during the 6–12-month period, when WA primarily reflects wasting, however, the restricted age range limits inferences beyond 36 months of age.

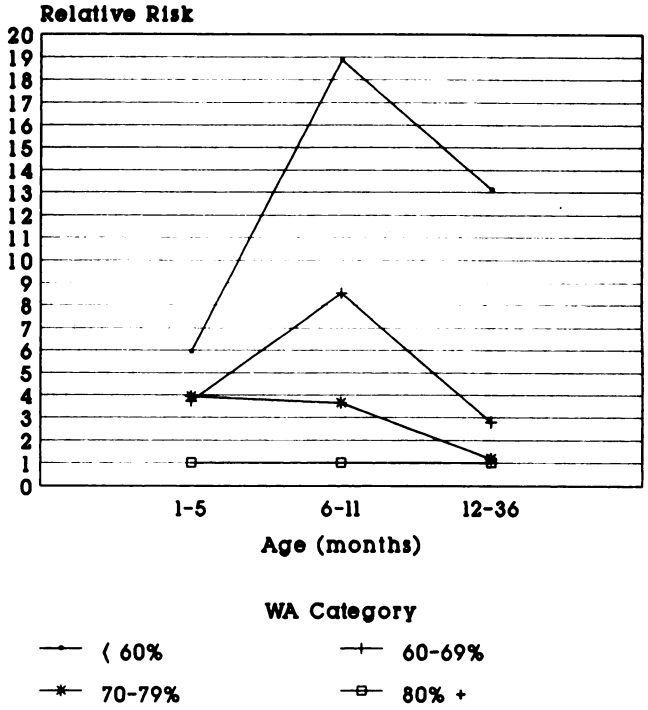


FIGURE 8 Punjab, India (study 7): relative risk of mortality according to child's age and weight-for-age. Data from Kielmann and McCord (1978). WA = weight-for-age.

The only other study with the potential to confirm this finding for WA is from Uganda (**Figure 9** and **Table 6**). This study employed less extreme cutoff points (<−3.0 Z-scores corresponding to "moderate deficits" and −3.0 to −2.0 corresponding to mild deficits) and suggests that the highest relative risks for WA occur among infants (<12 months). Beyond 12 to 24 months, the "mild deficits" category carries a lesser elevation in risk of mortality, confirming the results from Punjab. This may reflect the contribution of low HA to "underweight" at older ages, such that low WA is not particularly indicative of current nutritional deficits at these ages. By contrast, more severe deficits in WA are associated with elevated mortality at all ages in both studies, probably due to the existence of low HA as well as some deficits in WH.

If the above interpretation is correct, one would expect to see that deficits in WH are associated with elevated mortality in older, as well as younger, children,

TABLE 5

Interaction between child's age and weight-for-age in relation to subsequent mortality, Punjab, India (study 7)[1,2]

	Child's age in months		
Weight-for-age	1–5	6–11	12–36
>60%			
Rate	145.8	177.4	36.7
N	7/48	11/62	8/218
RR(age)	3.97	4.83	1.00
RR(WA)	5.93	4.83	13.11
60–69%			
Rate	91.7	80.2	7.8
N	10/109	17/212	8/1025
RR(age)	11.76	10.28	1.00
RR(WA)	3.73	8.53	2.79
70–79%			
Rate	96.9	34.3	3.3
N	25/250	15/437	7/2126
RR(age)	29.36	10.39	1.00
RR(WA)	3.94	3.65	1.18
≥80%			
Rate	24.6	9.4	2.8
N	28/1138	7/748	5/1776
RR(age)	8.79	3.36	1.00
RR(WA)	1.00	1.00	1.00
PAR(WA)	45.4%	72.6%	48.6%

[1] Calculated from Kielmann and McCord (1978).

[2] Rate = mortality rate per 1,000 children; N = number of deaths/number of children; RR(age) = relative risk for age, where the reference group is 12–36 months old; RR(WA) = relative risk for weight-for-age, where the reference group is ≥80% weight-for-age; PAR(WA) = population attributable risk for weight-for-age, where normal is ≥80% weight-for-age.

whereas low HA may be only associated at younger ages. Two studies, one from Indonesia (**Figure 10** and **Table 7**) and another from Uganda (Figure 9 and **Table 8**), permit this expectation to be tested. The Indonesian results provided the clearest support for this prediction, in that moderate HA deficits are associated with elevated mortality only below 23 months, and severe HA deficits have elevated mortality below 35 months. Neither category of HA deficits have elevated mortality above 35 months. By contrast, children with severe WH deficits have modest elevations in mortality below 23 months and marked elevations above 23 months, with some of the highest relative risks reported in the literature (RR = 9.6 and 16.9 for moderate and severe wasting in the older ages, respectively).[12] When moderate WH deficits (80–89% of median) are considered, the elevation in mortality is surprisingly modest (RR < 2.0) after 12 mo of age. There is no excess mortality associated with moderate WH deficits in the 36–60-months age group. The results from Uganda (again considering the less extreme cutoff points) are broadly similar, showing strong effects of HA at younger ages and strong effects of WH at all ages.

In light of these results, one would expect indicators based on arm circumference (AC) to perform similar to WH, in showing associations with mortality at all ages. **Figure 11** (and **Table 9**), based on AC-for-height, which provides some standardization for age, shows that this is indeed the case. Children with severe deficits have strong elevations in mortality at all ages, and those with moderate deficits have modest elevations at all ages. There is the suggestion of stronger effects with severe deficits above 36 months. In the Uganda study (Figure 9 and **Table 10**), which used simple AC with no standardization for age, mortality is elevated at all ages and the trend is most marked above 23 months. Part of the explanation for these findings is that arm circumference does increase with age through about 18 months of age in a normal population (Jelliffe 1968), such that the low values of arm circumference in this study are concentrated among the younger children who have higher mortality for many reasons. This is shown in Table 10 in that the mortality of infants aged 1–5 months is three times higher than those above 24 months, even among those in the highest AC category. Although this age confounding may account for the exaggerated age trends in the performance of AC in Figure 9, AC corrected for age (or height) is nonetheless associated with mortality at all ages, as shown in Figure 11 and other studies employing logistic regression analysis (Alam et al. 1989; Briend et al. 1987).

An important point, well-illustrated by the Indonesian results, is that the behavior of these indicators vis-a-vis relative risk is not a reliable guide concerning which of them has the greatest policy relevance. As shown in **Table 11** severe wasting (with an overall prevalence of only 2% in the sample) has a PAR of only 7.7% at its maximum (among 3–5-year-olds) and 4.2% over all ages. By contrast, severe stunting (with a comparable statistical definition and an overall prevalence of 33%) has a PAR of 20.1% over all ages. Even moderate plus severe wasting combined have a PAR of only 15.5% over all ages. Thus, the proportion of the population falling below various cutoff points must be taken into account when interpreting the policy relevance of indicators with a high relative risk. Tables 5–10 provide corresponding estimates for the other studies for which data are available, as well as the relevant sample sizes required to calculate PARs for a variety of cutoff points and age groups with which one may be concerned.

Another factor that makes it difficult to evaluate the effect of age on the anthropometry-mortality relationship is that all four studies reviewed above are based on fixed cutoff points for the indicator and, thus,

[12] Part of the explanation for the pronounced effects at older ages may be that 80% WA represents a more extreme cutoff point (when compared to Z-scores) at older than younger ages, due to the increase in variance with age.

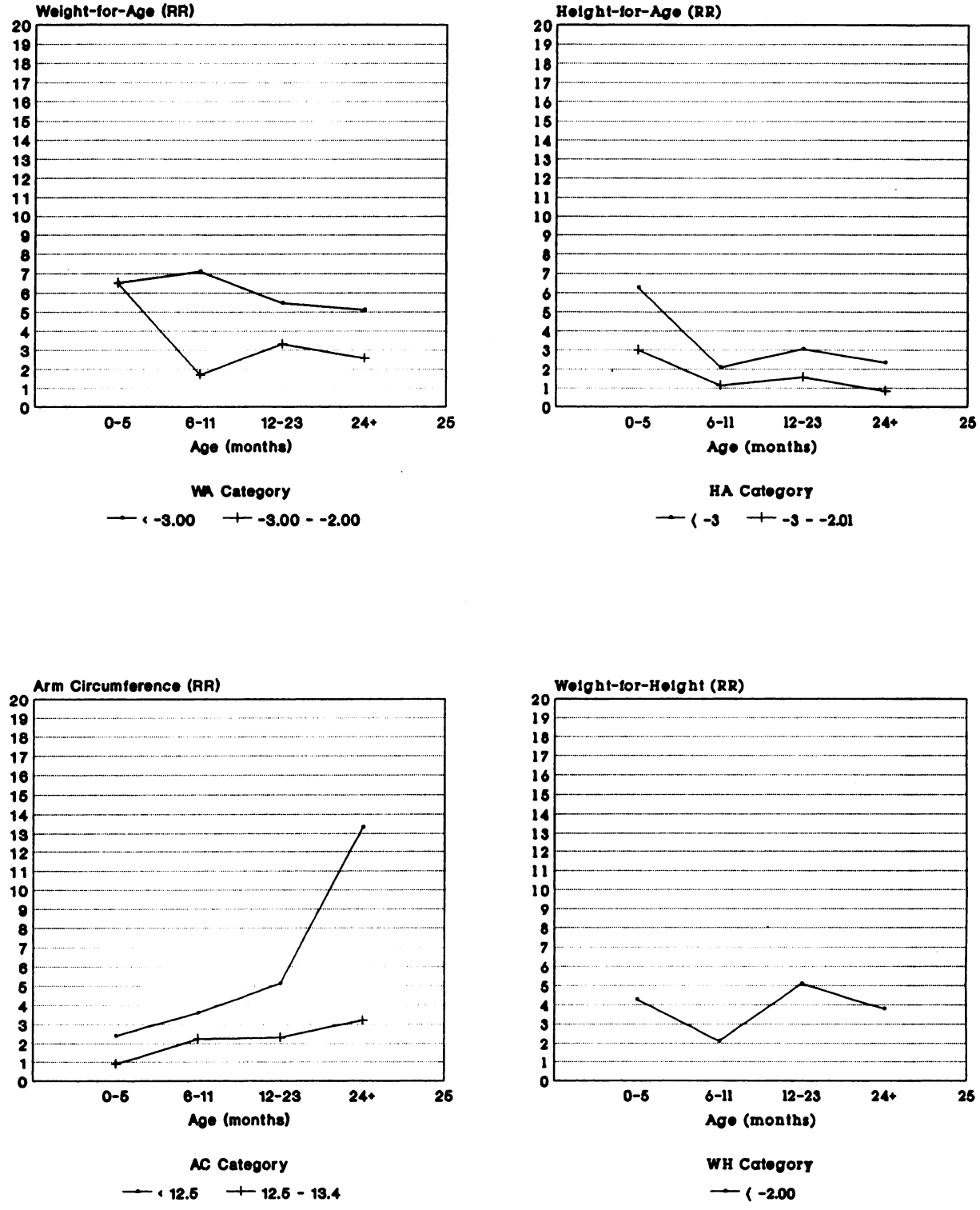
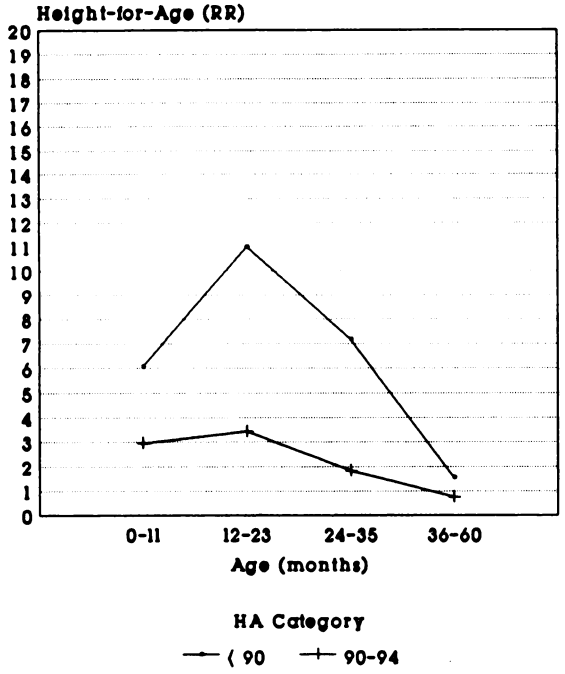


FIGURE 9 SW Uganda (study 17): relative risk of mortality according to child's age and four anthropometric indicators. WA = weight-for-age; HA = height-for-age; RR = relative risk; AC = arm circumference; WH = weight-for-height. Data from Vella et al. 1994.

cover a limited range of the sensitivity-specificity (Se/Sp) distribution. As elaborated elsewhere (Habicht et al. 1982), comparisons across indicators, or in this case across age groups for a single indicator, can lead to different conclusions depending on the region of the Se/Sp distribution being examined. This is illustrated in one of the Matlab studies (Briend and Zimicki 1986), which found that arm circumference has a slightly *higher* sensitivity for older children (36–59 months) than for younger children (12–23 or 24–35



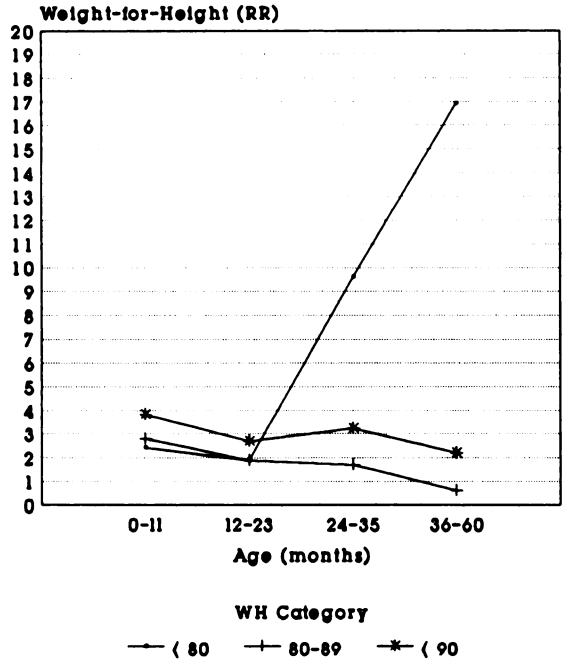


FIGURE 10 Indonesia (study 8): relative risk of mortality according to child's age and two anthropometric indicators. Adapted with permission from Katz et al. (1989). RR = relative risk; HA = height-for-age; WH = weight-for-height.

months), but only at extremely high levels of specificity (>95%). At lower levels of specificity the older children have a *lower* level of sensitivity. To the extent that the cutoff point for WH in the Indonesian study corresponds to the very high region of the specificity

TABLE 6

Interaction between child's age and weight-for-age in relation to subsequent mortality, SW Uganda (study 17)[1,2]

Weight-for-age (Z-scores)	Child's age in months			
	1–5	6–11	12–23	24+
< −2.50				
Rate	375.0	111.1	61.2	64.4
N	3/8	4/36	6/98	13/202
RR(age)	5.82	1.72	.95	1.00
RR(WA)	8.00	4.40	5.26	4.68
−2.50 to −1.51				
Rate	173.9	50.5	19.5	16.9
N	4/28	3/74	4/187	6/432
RR(age)	10.29	3.00	1.15	1.00
RR(WA)	3.71	2.00	1.67	1.23
> −1.50				
Rate	46.9	25.3	11.6	13.8
N	15/313	11/311	7/511	23/1410
RR(age)	3.40	1.83	.84	1.00
RR(WA)	1.00	1.00	1.00	1.00
PAR(WA)	25%	35%	40%	28%

[1] Calculated from Vella et al. (1994).

[2] Rate = mortality rate per 1,000 children; N = number of deaths/number of children; RR(age) = relative risk for age, where the reference group is 12–36 months old; RR(WA) = relative risk for weight-for-age, where the reference group is ≥80% weight-for-age; PAR(WA) = population attributable risk for weight-for-age, where normal is ≥80% weight-for-age.

spectrum (which seems likely), they are consistent with the results just described from Matlab, suggesting that measures of extreme wasting may have more mortality discriminating power among older than younger children.

The final study that provides some information on effect modification by age comes from Guinea-Bissau (Smedman et al. 1987). In this case visual inspection of survival curves based on synthetic cohorts of children suggests that all of the difference in cumulative probability of survival between wasted and nonwasted children arises in the first 6 months of life, whereas for stunting these differences become greater through ≥36 months of age. Although this would appear to be contrary to the results described above for wasting in Indonesia and Matlab, they are based on a single cutoff point (90% of median) and thus not strictly comparable. The results with respect to stunting (based on a 95% of median cutoff) are in better agreement with the Indonesian study, in demonstrating that discriminatory power exists only among younger children (<36 month). It is unclear to what extent the aforementioned measles epidemic in the better-nourished urban portion of the sample may have influenced these results.

By way of summary, **Table 12** shows the age-specific relative risks associated with anthropometry for all the studies permitting quantification. Although inter-

TABLE 7

***Interaction between child's age and anthropometric indicators in relation to subsequent mortality, Indonesia (study 8)*[1,2]**

Indicators	Child's age in months			
	0–11	12–23	24–35	36–59
Height-for-age				
<90%				
Rate	173.9	149.7	85.1	18.8
N	4/23	28/187	20/235	13/693
RR(age)	9.25	7.96	4.53	1.00
RR(HA)	6.06	11.01	7.15	1.55
90–94%				
Rate	84.8	46.8	21.9	9.1
N	14/110	20/285	8/244	6/440
RR(age)	9.32	5.14	2.41	1.00
RR(HA)	2.95	3.44	1.84	.75
≥95%				
Rate	28.7	13.76	11.9	12.1
N	26/605	4/196	3/168	5/275
RR(age)	2.37	1.12	.98	1.00
RR(HA)	1.00	1.00	1.00	1.00
PAR(HA)	21.6%	67.1%	63.2%	19.2%
Weight-for-height				
<90%				
Rate	118.5	108.8	78.2	23.3
N	16/135	26/239	14/179	5/2158
RR(age)	5.09	4.67	3.36	1.00
RR(WH)	3.82	2.69	3.23	2.20
≥90%				
Rate	31.0	40.4	24.2	10.6
N	28/603	26/429	14/179	5/215
RR(age)	2.92	3.81	2.28	1.00
RR(WH)	1.00	1.00	1.00	1.00
PAR(WH)	26.8%	31.4%	31.2%	11.3%

[1] Calculated from Katz et al. (1989).

[2] Rate = mortality rate per 1,000 children; N = number of deaths divided by number of children; RR(age) = relative risk for age, where the reference group is 36–59 months old; RR(HA) = relative risk for height-for-age where the reference group is ≥95% height-for-age; PAR(HA) = population attributable risk for height-for-age where normal is ≥95% height-for-age; RR(WH) = relative risk for weight-for-height where the reference group is ≥90% weight-for-height; PAR(WH) = population attributable risk for weight-for-height where normal is ≥90% weight-for-height.

pretation is made difficult by the use of different indicators, cutoff points and age ranges in various studies, some general conclusions are suggested by these data. The major caution is that the conclusions should be restricted to within-study comparison of relative risks, rather than between-study comparisons. The table shows that mild deficits of WA are not associated with elevated mortality among older children, though they are associated in younger children.

Moderate and severe deficits have elevated risks in all age groups. This is consistent with the knowledge that much of the weight deficit in older children is due to height deficits that accumulated earlier and may not pose any current danger to the child. The results for WH reveal that moderate and severe deficits are associated with modest elevations in mortality risk through 23 months of age, as do moderate deficits among older children (>23 months). However, in both studies, there is a marked elevation in risk among older children when the deficit is severe. The relative risks among older children (>12 months) are similar in magnitude to those found for AC/HT in Bangladesh, which represents an alternative indicator of wasting and also corrects for child's height. These results sug-

TABLE 8

***Interaction between child's age and anthropometric indicators in relation to subsequent mortality, SW Uganda (study 17)*[1,2]**

Indicators (Z-Scores)	Child's age in months			
	0–5	6–11	12–23	24+
Height-for-age				
< −3				
Rate	300.0	74.1	41.7	37.7
N	3/10	2/27	5/120	13/345
RR(age)	7.96	1.97	1.11	1.00
RR(HA)	6.26	2.09	3.04	2.31
−3–−2.01				
Rate	142.9	40.5	21.4	13.9
N	4/28	3/74	4/187	6/432
RR(age)	10.28	2.91	1.57	1.00
RR(HA)	2.98	1.15	1.56	.85
> −2.0				
Rate	47.9	35.4	13.7	16.3
N	15/313	11/311	7/511	23/1410
RR(age)	2.94	2.17	.84	1.00
RR(HA)	1.00	1.00	1.00	1.00
PAR(HA)	24%	9%	30%	15%
Weight-for-height				
< −1.5				
Rate	166.7	86.2	42.4	76.0
N	3/18	5/58	5/118	12/158
RR(age)	2.19	1.13	.56	1.00
RR(WH)	3.06	3.02	2.64	5.47
−1.5–−1.01				
Rate	95.2	76.9	13.0	17.5
N	2/21	3/39	1/77	4/228
RR(age)	5.44	4.39	.74	1.00
RR(WH)	1.75	2.69	.81	1.26
> −1.0				
Rate	54.5	28.6	16.1	13.9
N	17/312	9/315	10/623	25/1801
RR(age)	3.92	2.06	1.16	1.00
RR(WH)	1.00	1.00	1.00	1.00
PAR(WH)	13%	31%	18%	26%

[1] Calculated from Vella et al. (1994).

[2] Rate = mortality rate per 1000 children; N = number of deaths divided by number of children; RR(age) = relative risk for age, where the reference group is 36–59 months old; RR(HA) = relative risk for height-for-age where the reference group is ≥95% height-for-age; PAR(HA) = population attributable risk for height-for-age where normal is ≥95% height-for-age; RR(WH) = relative risk for weight-for-height where the reference group is ≥90% weight-for-height; PAR(WH) = population attributable risk for weight-for-height where normal is ≥90% weight-for-height.

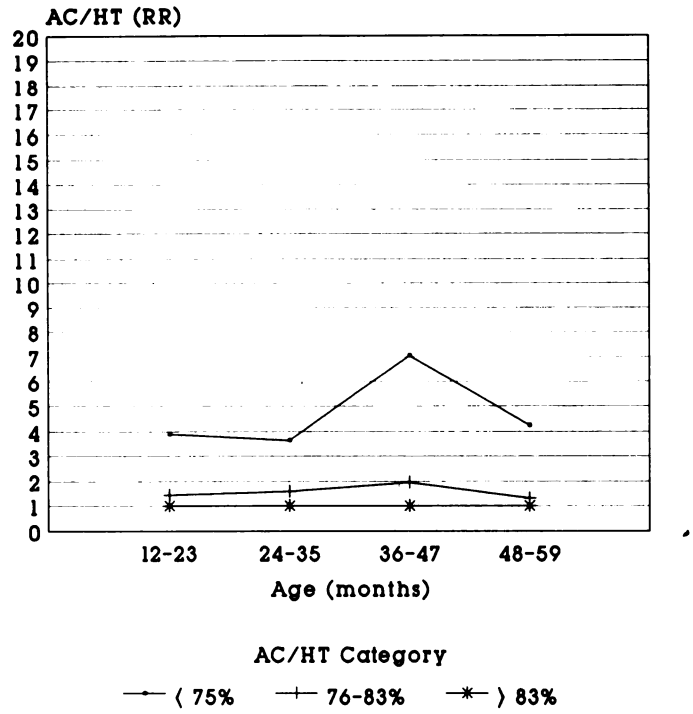


FIGURE 11 Matlab, Bangladesh (study 2a): relative risk of mortality according to child's age and AC/HT. Data from Sommer and Lowenstein (1975). AC/HT = arm circumference/height; RR = relative risk.

gest that wasting (when defined with a common percent of median cutoff across all ages) is more strongly associated with mortality among older children than among younger children. Some of this may be due to the increase in variance with age, but the present analyses do not permit this to be quantified. Finally, these results reveal that mortality is elevated among those with moderate height deficits before 24 months of age, but much less so beyond that age, and that severe height deficits are associated with elevated mortality at all ages. The findings pertaining to HA at older ages confirm the interpretation related to WA, that moderate stunting poses no immediate threat to survival when it occurs among older children.

Gender effects. Considering the disproportionate number of studies coming from South Asia (Table 1) and the evidence for sex differences in dietary intake, nutritional status, health care and mortality in this part of the world (Abdullah and Wheeler 1985; Chen et al. 1981; Das Gupta 1987; Kielmann et al. 1978), it is surprising to find that only two studies have undertaken an explicit analysis of effect modification due to sex of the child (Cogill 1982). Several studies appeared from verbal descriptions in the text to have tested or controlled for main effects due to sex (all finding that gender does not account for the anthro-

TABLE 9

***Interaction between child's age and arm circumference in relation to subsequent mortality Bangladesh (study 2a)*[1,2]**

Arm circumference-for-height	Child's age in months			
	12–23	24–35	36–47	48–59
<75%				
Rate	118.6	136.4	177.4	60.6
N	7/59	9/66	11/62	4/66
RR(age)	1.96	2.25	2.93	1.00
RR(ACHT)	3.89	3.64	7.04	4.24
76–83%				
Rate	44.1	59.7	49.3	18.7
N	18/408	26/435	20/406	6/321
RR(age)	2.36	3.19	2.64	1.00
RR(ACHT)	1.45	1.59	1.96	1.31
≥83%				
Rate	30.5	37.5	25.2	14.3
N	15/492	19/507	13/515	6/420
RR(age)	2.13	2.62	1.76	1.00
RR(ACHT)	1.00	1.00	1.00	1.00
PAR(ACHT)	26.7%	30.0%	43.7%	27.9%

[1] Calculated from Sommer et al. (1975).

[2] Rate = mortality rate per 1000 children; N = number of deaths divided by number of children; RR(age) = relative risk for age, where the reference group is 48–59 months old; RR(ACHT) = relative risk for arm circumference-for-height (ACHT) where the reference group is ≥83% ACHT; PAR(ACHT) = population attributable risk for ACHT where normal is ≥83% ACHT.

TABLE 10

Interaction between child's age and arm circumference in relation to subsequent mortality, SW Uganda (study 17)

Arm circumference	Child's age in months			
	1–5	6–11	12–23	24+
cm				
<12.5				
Rate	106.2	90.9	71.4	200.0
N	12/113	4/44	3/42	6/30
RR(age)	.53	.45	.36	1.00
RR(AC)	2.39	3.60	5.13	13.33
12.5–13.4				
Rate	40.0	56.8	32.3	48.2
N	4/100	5/88	4/124	4/83
RR(age)	.83	1.18	.67	1.00
RR(AC)	.90	2.25	2.32	3.21
>13.5				
Rate	44.4	25.3	13.9	15.0
N	6/135	7/277	9/646	31/2066
RR(age)	2.96	1.69	.93	1.00
RR(AC)	1.00	1.00	1.00	1.00
PAR(AC)	30%	35%	29%	20%

[1] Calculated from Vella et al. (1994).

[2] Rate = mortality rate per 1000 children; N= number of deaths divided by number of children; RR(age) = relative risk for age, where the reference group is 48–59 months old; RR(AC) = relative risk for arm circumference where the reference group is ≥13.5 cms ACHT; PAR(AC) = population attributable risk for AC where normal is ≥13.5 cms AC. AC = arm circumference.

TABLE 11

Mortality rates, relative risks, and population attribute risks for weight-for-height and height-for-age, stratified by age of the child, Indonesia (Study 8)[1,2]

Age	Nutritional status[3]	Weight-for-height				Height-for-age			
		N	Rate	RR	PAR	N	Rate	RR	PAR
mo									
<12	Normal	28/603	31.0	1.0	—	26/605	28.7	1.0	—
	Moderate	14/116	80.5	2.8	12.9	14/110	84.8	3.2	14.0
	Severe	2/19	70.2	2.4	1.6	4/23	173.9	6.1	7.6
	Total				14.5				21.6
12–23	Normal	26/429	40.4	1.0	—	4/196	13.6	1.0	—
	Moderate	23/212	72.3	1.9	13.0	20/285	46.8	3.6	18.2
	Severe	3/27	74.1	1.9	1.7	28/187	149.7	11.0	48.9
	Total				14.7				67.1
24–35	Normal	17/468	24.2	1.0	—	3/168	11.9	1.0	—
	Moderate	10/164	40.7	1.7	8.7	8/244	21.9	1.9	7.9
	Severe	4/15	177.8	9.6	7.4	20/235	85.1	7.2	55.5
	Total				16.2				63.4
36–59	Normal	19/1193	10.6	1.0	—	5/275	12.1	1.0	—
	Moderate	2/201	6.6	0.6	0.0	6/440	9.1	0.7	0.0
	Severe	3/14	142.9	16.9	7.7	13/693	18.8	1.6	19.3
	Total				7.7				19.3
0–59	Normal	90/2693	22.4	1.0	—	38/1244	20.4	1.0	—
	Moderate	49/693	47.1	2.2	11.3	48/1079	29.7	1.5	6.6
	Severe	12/75	106.7	5.5	4.2	65/1138	38.1	1.9	20.1
	Total				15.5				26.7

[1] Calculated from Katz et al. (1989).

[2] N = number of deaths divided by number of children; Rate = deaths per 1000 children per year; RR = relative; risk; PAR = population attributable risk.

[3] Normal ($Z > -1$): weight-for-height (WH) > 90%; height-for-age (HA) > 95%; moderate ($-1 > Z > -2$): WH 80–89%; HA 90–94%; severe: ($Z < -2$); WH < 80%; HA < 90%.

pometry effect); however, they did not appear to have undertaken interaction analysis. This is unfortunate in light of the markedly different results which Cogill (1982) and Fauveau et al. (1992) obtained for males and females as described below.

The analysis by Cogill (1982) compared the performance of seven anthropometric indicators in predicting mortality within 2 years of measurement. **Table 13** shows these indicators, their normalized distance statistics (d_a)[13] and their ranks for males, females and sexes combined. The results shown here are very similar to those found with other criteria of indicator performance in Cogill's work. With the exception of WH-based indicators, which are low in both sexes, the mortality discrimination of all other indicators is greater among females than males, especially in the case of WA and HA. The results for combined sexes tend to be intermediate or to resemble those for females. The effect on the ranks is such that mortality is best discriminated by HA, AC and WA among females, and by arm circumference-for-height (ACHT), arm circumference-for-age (ACA) and AC in males. The two best discriminators among females (HA and WA) are ranked fourth and fifth among males.

The overall results by Cogill have since been confirmed by a study in the same area of Bangladesh (Fauveau et al. 1990). This study, which determined malnutrition based on visible wasting and recent weight loss just before death, found that the RR of death for girls is 1.8 times that for boys. The corresponding RR is 2.1 for deaths "due to wasting" and 2.6 for deaths due to wasting in combination with persistent diarrhea. Both sexes experienced an increase in mortality during the preharvest season, but this was especially marked for girls (i.e., the number of deaths per month increased from 2.3 to 4.4 for boys, and from 3.3 to 10.5 for girls).

In attempting to explain the gender differences in the anthropometry-mortality relationship, Cogill notes that females in the sample have a mortality rate that is twice that of males and significantly lower levels of nutritional status as reflected in anthropometric indicators. The latter is consistent with evidence of male preference in food allocation (in terms of quality and

[13] d_a is calculated for each indicator as the difference between the means of dead and surviving children, divided by the square root of the average variance of the indicator in dead and surviving children (Brownie et al. 1986).

TABLE 12

Summary of age-specific relative risks of mortality by indicator[1]

Study	Indicator	Severity	Age[a] 0-11	12-23	12-36	24+
Punjab[2] (study 7)	WA	Normal (>80%)	(19)	—	(3)	—
		Mild (70-79)	3.1	—	1.2	—
		Moderate (60-69)	4.5	—	2.8	—
		Severe (<60)	8.8	—	13.1	—
SW Uganda[2] (Study 17)	WA	Normal (>−1.5 Z)	(42)	(12)		(14)
		Mild-Mod (−1.5 to −2.5)	1.65	1.7		1.2
		Mod-Sev (<−2.5)	3.82	5.3		4.7
Indonesia[4] (Study 8)	WH	Normal (>90%)	(31)	(40)		(22)
		Moderate (80-89)	2.8	1.9		1.5
		Severe (<80)	2.4	1.9		11.1
SW Uganda[3] (Study 17)	WH	Normal (>−1 Z)	(45)	(16)		(14)
		Moderate (−1.5 to −1.0)	1.9	.8		1.3
		Severe (>−1.0)	2.4	2.6		5.5
Bangladesh[5] (Study 2a)	AC/HT	Normal (>83%)	—	(30)		(37)
		Moderate (76-83)	—	1.5		1.6
		Severe (<75)	—	3.9		3.6
Indonesia[4] (Study 8)	HA	Normal (>95%)	(29)	(14)		(18)
		Moderate (90-99)	3.0	3.4		1.1
		Severe (<90)	6.1	11.0		2.0
SW Uganda[3] (Study 17)	HA	Normal (>−2)	(42)	(14)		(16)
		Moderate (−2 to −3)	1.6	1.6		.8
		Severe (<−3)	3.2	3.0		2.3

[1] WA = weight-for-age; WH = weight-for-height; AC/HT = arm circumference/height; HA = height-for-age.
[2] Adapted from Kielmann and McCord (1978).
[3] Adapted from Vella et al. (1994).
[4] Adapted from Katz et al. (1989).
[5] Adapted from Sommer and Lowenstein 1975.
[a] Figures in parentheses are the mortality rates for the reference groups.

quantity) in this population (Chen et al. 1981). Yet, these observations alone are insufficient to explain why the relationship between these indicators and mortality is stronger *within* the female subsample as compared to males. Part of the answer may lie with the fact that, despite similar morbidity attack rates, female children are brought to free health facilities only 60% as often as males (Chen et al. 1981). Thus, greater utilization of curative health services by males may partially uncouple the linkage between nutritional status and mortality as compared to females. This is supported by more recent analysis of data from the same area of Bangladesh (Fauveau et al. 1991) and in South Asia in general (Basu 1989).

Morbidity. The existence of a synergistic relationship between malnutrition and infectious disease has been widely recognized for several decades (Scrimshaw et al. 1968). In the case of PEM the relationship is such that recurrent morbidity has a negative effect on nutritional status; poor nutritional status may, depending on the disease, increase either the incidence, severity and/or duration of morbidity; and the combination of poor nutritional status and morbidity increases the risk of death. The classic model of this interaction is with measles (Morley 1973), but the evidence relating malnutrition to immunocompetence suggests similar interactions with a variety of diseases (Chandra 1991; Martorell and Ho 1984).

Given these interactions it is not meaningful in any practical sense to attempt to separate the effects of nutritional status from those of morbidity as they relate to risk of mortality. This was discussed earlier with reference to Figure 5. Given the ubiquity of disease in developing countries only a very minor fraction of child deaths result directly and exclusively from malnutrition. Most deaths result from disease made worse by malnutrition.

As distinct from the above question, a number of investigators have raised the possibility that children with low weight-for-age (for instance) may appear to

TABLE 13

Normalized distance statistic and ranks for seven anthropometric indicators in Bangladeshi males, females and combined sexes (study 1d)[1]

Indicators	Normalized distance			Ranks		
	Males	Females	Combined	Males	Females	Combined
Height-for-age	0.2790	0.7306	0.5797	5	1	1
Weight-for-age	0.3644	0.6628	0.5793	4	2	3
Arm Circumference-for-age	0.4438	0.6156	0.5623	1	3	4
Arm Circumference	0.4180	0.5970	0.5886	3	4	2
Arm Circumference-for-height	0.4196	0.5320	0.5440	2	5	5
Weight/height^2	0.2657	0.2037	0.2936	6	6	6
Weight-for-height	0.1942	0.0540	0.1198	7	7	7

[1] Adapted with permission from Cogill (1982).

have an elevated risk of mortality simply because the disease that killed them resulted in acute weight loss during the weeks immediately preceding death. Thus, their low weight-for-age may be only secondary to the real cause of death and the entire association between WA and mortality may be due to statistical artefact.[14] The four studies bearing on these possibilities are reviewed below.

In their 1-year follow-up of children from Punjab, India, Kielmann and McCord (1978) recognized that the effect of low weight-for-age on subsequent mortality may be overestimated because concurrent infection is likely to decrease weight-for-age in the few weeks preceding death. Because children in that study had been weighed monthly, the investigators were able to minimize this effect by trying to confirm their overall results on a subsample. Specifically, they analyzed mortality in relation to the weight-for-age of each child as measured 2 months before death rather than the 1 month immediately preceding death. This eliminated 37 of the children (34%) from the analysis, representing those who died in the first month of the study and could not be assigned to a weight-for-age category.

The analysis by Kielmann and McCord shows that in each of the three age groups the relationship between weight-for-age and mortality is the same in the two samples. Thus, the authors concluded that the effect of concurrent illness on weight-for-age is unlikely to account for the association between the latter and mortality. It is possible that acute morbidity may affect weight-for-age for more than 1 month before death, thereby invalidating this approach for "controlling" these effects. However, the close correspondence between the controlled and uncontrolled results suggests that confounding of weight-for-age by intercurrent morbidity is unlikely to account for the association in its entirety. Moreover, if morbidity persists for more than 1 month, it is likely to have significant effects on nutritional status itself, not simply the weight-for-age proxy for nutritional status.

In a study from rural Bangladesh, Briend and Bari (1989b) used multivariate logistic regression to control for concurrent morbidity, while examining the association between severe weight-for-age and mortality of 0–36-month-olds. Children were classified as being above or below 60% WA and according to the presence or absence of diarrhea, respiratory infections, measles and edema. The mortality odds ratio for the WA variable was reduced from 14.7 (unadjusted) to 9.7 after adjusting for the presence/absence of these illnesses (with a 95% confidence interval of 5.7 to 16.6 for the adjusted ratio). Because there was no association between mortality and diarrhea in this sample [which the authors attribute to an intensive oral rehydration solution (ORS) program in the area], the association between mortality and WA is unaffected by diarrhea. This study therefore suggests that the incidence of concurrent morbidity does not account for all of the association between severely low WA and mortality. This may be because the study only controlled for the presence/absence of morbidity in the preceding mo, which does not fully account for the possible weight-depressing effects of morbidity.

Although the authors did not conceptualize it as such, another form of control over the confounding effects of morbidity was exercised in the studies by Briend and Bari (1989b) and Yambi (1988). These analyses were designed to compare indicators of weight change over the course of $\geq$1 mo to indicators of attained weight in the same mortality-prediction model, for the purpose of identifying the best indicator for screening purposes. However, this can also be conceptualized as an effort to distinguish the immediate weight-reducing effects of a current illness (as reflected in the weight change variable) from functional impairments arising from low WA (e.g., possible de-

[14] A parallel situation exists in studies of adult mortality, in which elevated mortality among lean subjects is due to chronic weight loss from cancer or other illnesses rather from an effect of body size or composition per se.

pressed immune function that affects the severity of disease).

In both studies the multivariate logistic regression results show that severely low weight-for-age retains a highly significant relationship with mortality even after weight change in the preceding 1–2 months is accounted for in the model. Although the effectiveness of this approach for controlling for acute weight loss is limited by the compounded measurement error in the weight change variable, the size of the weight-for-age effect [i.e., an odds ratio of 9.7 with a standard error of 2.25 in Briend and Bari (1989b)] is such that a relationship is likely to persist even if measurement error were greatly reduced (de Klerk et al. 1989).

Thus, within the limits of their respective methodologies these four studies do not support the hypothesis that the association between WA and mortality is an artefact of morbidity-induced weight loss immediately before death. Rather, the accumulated evidence lends support to the existence of a direct link between anthropometric indicators and mortality, possibly mediated through the severity and duration of illness (Martorell and Ho 1984). It is important to note, however, that the available studies did not examine whether similar results are found in mild-to-moderate, as opposed to severe, malnutrition.

Breastfeeding. Two reports in the literature, from different areas of rural Bangladesh, have documented that breastfeeding is associated with reduced risk of mortality in the month after measurement. In both cases this effect was observed only among severely malnourished children, as defined by WA < 60% (Briend and Bari 1989b) or arm circumference < 110 mm (Briend et al. 1988). In the former study this association was shown to be independent of child's age in a multivariate logistic regression. The latter study did not control for age; however, the direction of the difference in mean age between weaned (29 months) and breastfed (22 months) children suggests that the estimated effect of breastfeeding would probably be greater if age were controlled.

Although both of the above reports emphasized the positive effects of breastfeeding among severely malnourished children, they also provide evidence that breastfeeding acts as an effect modifier of the relation between anthropometric indicators and mortality. As shown in **Table 14** the mortality rate is 8–10/1,000 child-months among breastfed children in the most extreme category of malnutrition, as compared with 34–41/100 for weaned children. These differences are even greater when the mortality rate in the extreme category is examined in relation to that of the highest nutritional category (i.e., relative risks as shown in Table 14). Thus, although mortality is associated with anthropometric indicators in all cases, the effect is significantly attenuated in the presence of breastfeeding. This is reminiscent of the findings of Butz et al. (1984) that the effects of environmental factors (e.g., type of sanitation and water supply) on infant mortality are significantly affected by the presence or absence of breastfeeding.

In both of the above studies the positive effect of breastfeeding on survival, which is seen when all nutritional categories are combined, is not accounted for by better nutritional status of breastfed children. This observation, together with the evidence in one study (Briend et al. 1988) that the effect is not accounted for by cessation of breastfeeding due to intercurrent illness, is the reason for juxtaposing breastfeeding with immune status in Figure 7, rather than with nutrient intake. One interpretation is that, among these older children at least (>12 months), the primary benefit of breastfeeding may lie in its immunologic rather than nutritional properties. An alternative hypothesis is that the breastfeeding mothers in this sample also differ in their use of health services, and it is the latter which accounts for the lower mortality.

TABLE 14

***Mortality in relation to anthropometric indicators and breastfeeding, Bangladesh (studies 6a and 3b)*[1,2]**

	Breastfed			Weaned		
Indicator level	Deaths	Child-months	Rate/1000	Deaths	Child-months	Rate/1000
Chandpur-Comilla Highway, Bangladesh						
WA < 60%	12	1,230	9.76	35	851	41.1
WA ≥ 60%	14	7,940	1.76	8	4,898	1.6
Relative risk	—	—	5.55	—	—	25.7
Matlab, Bangladesh						
AC ≤ 110 mm	8	984	8.13	20	592	33.8
AC 111–125 mm	13	6,511	2.00	1	2,098	0.4
AC > 125 mm	6	12,537	0.48	1	4,953	0.2
Relative risk	—	—	16.94	—	—	169.0

[1] Adapted from Briend and Bari (1989a) and Briend et al. (1988).

[2] WA = weight-for-age; AC = arm circumference; relative risk = rate for severe category divided by rate for normal catetory.

Seasonality. One of the most pervasive aspects of life in developing countries is the existence of seasonal variation in access to food at the household level, daily activity schedules (with implications for quality of child care), child nutritional status, morbidity and mortality. These patterns are such that the seasons with highest mortality are also those with highest morbidity, least access to health care and highest levels of malnutrition. This suggests the strong possibility that, depending upon the timing of the survey, the association between child mortality and anthropometric indicators may be at least partially confounded by season of measurement, with morbidity and child/health care acting as proximate mechanisms for the confounding (see Fig. 7).

Given the pervasiveness of this situation, it is striking that the effects of seasonality have not been systematically accounted for in the literature. Of the 28 reports reviewed here, only the one from Narangwal describes (in the text) the effect of seasonality on the relationship between anthropometry and mortality (Kielmann and McCord 1978). The study reports that "malnourished children (<70% of Harvard standard) during the first 6 months of the year (January–June) ran the highest risk of dying within 6 months. The risk of death was considerably lower if they were malnourished during the second half of the year (July–December). The risk of death within 6 months appears to be independent of the season of the survey for those at or above 80% of the Harvard weight median" (p. 1249). Thus, the verbal description clearly indicates that the effect of weight-for-age on mortality depends on the season. However, the lack of quantitative data in the report precludes drawing inferences concerning the extent to which the anthropometry-mortality relationship is itself a statistical artefact of seasonality. Thus, on the basis of one study, seasonality appears to be an effect modifier in the anthropometry-mortality relationship, but there is no direct evidence examining the possibility of seasonality as a confounder of this relationship.

Socioeconomic factors. In a manner similar to seasonality, socioeconomic status (SES) may represent a major confounding factor between child anthropometry and mortality. Referring to Figure 7, it can be seen that SES may influence mortality through some pathways that also have a strong impact on anthropometry (e.g., intake and morbidity). However, SES may also influence mortality through some pathways that save children's lives (e.g., use of health care and better quality illness management at home) but have relatively smaller impacts on anthropometry. In the latter case, confounding by SES could account for much, if not all, of the association between anthropometry and mortality.

Several studies in the literature have directly examined this possibility. The early study by Chen et al. (1980) included consideration of SES in the analysis, and two reports from the same data set have since analyzed these relationships in further detail (Chowdhury 1988, Cogill 1982). The other studies are from Iringa Region, Tanzania (Yambi 1988) and Uganda (Vella et al. 1994).

Table 15 shows that among Bangladeshi children the relationship between anthropometric deficits and mortality is remarkably stable, being found in each of the socioeconomic strata examined in two studies of this data set. The relative risk of mortality due to poor nutritional status varies between 2.16 and 4.23 across these strata. Surprisingly, both of the studies shown in this table suggest that the relative risk of mortality due to anthropometric deficits is actually greater in the *higher* economic strata (those with bigger houses and more maternal education), contrary to theoretical expectations.

The interrelationships of anthropometric indicators, mortality and socioeconomic indicators were examined in greater detail by Cogill (1982) using multivariate discriminant analysis. In a model which potentially includes age of the mother and child, parity, religion, education of mother and father, number of cows, floor area, mother's height and weight and all of the anthropometric indicators, the results show that each of the child anthropometric indicators is statis-

TABLE 15

Relationship between child anthropometry and mortality in Bangladesh, stratified by socioeconomic indicators, Bangladesh (studies 1a and 1c)[1]

SES group	Mortality rates (per 1000/year)		
	Poor nutritional status	Better nutritional status	Relative risk
Matlab, Bangladesh[2] (Study 1a)			
Floor space in living quarters			
<242 sq. ft.	123.6	53.9	2.29
≥242 sq. ft.	99.5	23.5	4.23
Maternal height			
<147.5 cms.	130.9	37.9	3.45
≥147.5 cms.	82.4	38.2	2.16
Matlab, Bangladesh[3] (Study 1c)			
Maternal age and education			
<25 and no education	114.0	43.0	2.65
<25 and some education	125.0	30.0	4.17
≥25 and no education	119.0	48.0	2.48
≥25 and some education	94.0	23.0	4.09

[1] Adapted from Chen et al. (1980) and Chowdhury (1988).

[2] Nutritional status defined as above or below 85% of median height-for-age. The text of the report states that the same relationships were found with the other anthropometric indicators; however, results were not presented.

[3] Nutritional status defined as above or below 60% of median weight-for-age.

TABLE 16

***Mortality rate (per 1000 per year) and relative risks by anthropometric deficits and socioeconomic status, SW Uganda (study 17)*[1,2]**

		Anthropometric deficit					
		Severe		Moderate		Normal	
Indicators	SES group	Mortality	(RR)	Mortality	(RR)	Mortality	(RR)
WA	High	105	(6.18)	45	(2.65)	17	(1.00)
	Medium	85	(4.47)	48	(2.53)	19	(1.00)
	Low	83	(2.59)	54	(1.69)	32	(1.00)
	All groups	91	(4.42)	48	(2.36)	20	(1.00)
HA	High	46	(2.42)	26	(1.37)	19	(1.00)
	Medium	30	(1.20)	25	(1.00)	25	(1.00)
	Low	80	(2.90)	16	(.52)	31	(1.00)
	All groups	49	(2.05)	23	(.98)	23	(1.00)
WH	High	65	(3.09)			21	(1.00)
	Medium	140	(6.36)			22	(1.00)
	Low	42	(1.08)			39	(1.00)
	All groups	94	(2.72)			25	(1.00)
AC	High	107	(7.13)	56	(3.73)	15	(1.00)
	Medium	95	(5.28)	49	(2.72)	18	(1.00)
	Low	164	(6.07)	25	(.93)	27	(1.00)
	All groups	117	(6.44)	46	(2.54)	18	(1.00)

	Definitions		
	Severe	Moderate	Normal
WA (Z)	<−2.5	−2.5 to −1.51	>1.5
HA (Z)	<−3.0	−3.0 to −2.01	>2.0
WH (Z)	<−1.50	−1.5 to −1.01	>−1.0
AC (cm)	<12.5	12.5 to 13.4	>13.5

[1] Calculated from Vella et al. (1994).
[2] WA = weight-for-age; HA = height-for-age; WH = weight-for-height; AC = arm circumference.

tically significant except for weight-for-age.[15] The magnitude of the standardized coefficients suggest that the discriminatory power of the anthropometric indicators is 2.3 times (in the case of HA) to 23 times (in the case of AC) greater than any of the SES indicators.

In the Tanzanian study (Yambi 1988), SES variables were controlled in multivariate logistic regression models that included weight-for-age and child's age. The results show that WA retains its statistical significance in relation to mortality even after controlling for maternal age (model 1), maternal education (model 2), household size and number of rooms (model 3) and household agricultural variables (model 4).

Table 16, from Uganda (Vella et al. 1994), shows the mortality rates and relative risks for four anthropometric indicators stratified by a combined index of socioeconomic status formed from 19 individual variables. As in the other studies, the association between severe deficits and mortality is apparent even within SES groups and is not significantly diminished in magnitude. The significance of this study is the opportunity it affords for examining whether a relationship persists between *moderate* deficits and mortality, after stratifying by SES, in contrast to the other studies that restricted their attention to the case of severe deficits. The results show, in general, that the relative risk for individual SES groups varies fairly widely compared with that observed for all SES groups combined. In the case of HA, WH and (especially) AC, the relative risks are higher in the high or medium SES groups than in the lowest SES group, reminiscent of the findings from Bangladesh in Table 15. The statistical significance of this variation in relative risks is uncertain, although the original paper (Vella et al. 1994) reports that there are no significant interactions between SES and anthropometric variables when tested by logistic regres-

[15] The indicators tested include WA, HA weight/height (W/H), weight/height^2, AC, arm circumference/height (AC/HT) and arm circumference/age (AC/A). The lack of significance for WA is probably due to multicollinearity with the other anthropometric indicators.

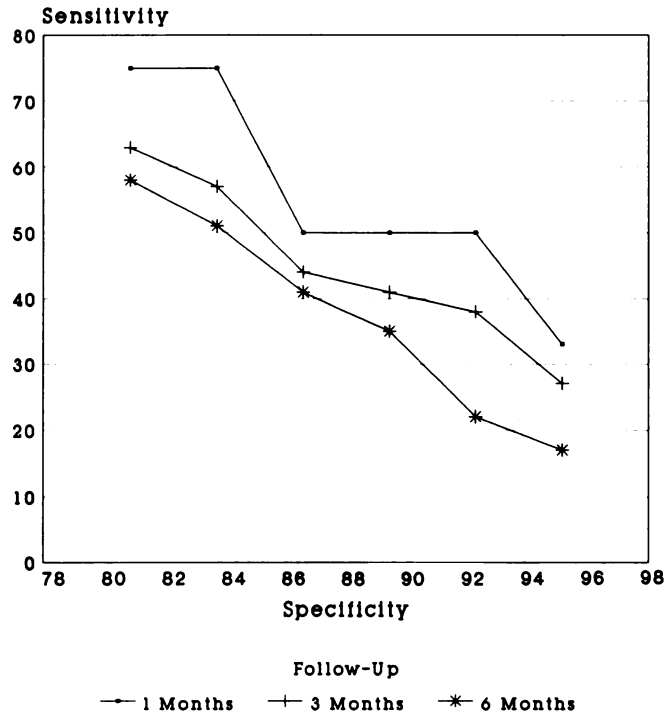


FIGURE 12 Matlab, Bangladesh (study 2c): sensitivity-specificity curves predicting mortality from arm circumference. Adapted with permission from Briend and Zimicki (1986).

sion and when considering the entire distribution of anthropometric deficits.

Thus, it appears that SES may not be a confounder of the anthropometry-mortality relationships as much as it is an effect modifier. Moreover, the accumulated evidence suggests that malnutrition, as reflected in anthropometry, may be more strongly related to mortality among the higher SES groups than among the lower SES groups. This result is contrary to theoretical expectations and requires further investigation. Additional analysis of these relationships is provided in Pelletier et al. (1994a) in this supplement.

Length of follow-up

A question with important programmatic implications is the extent to which the predictive ability of anthropometric indicators is attenuated as the length of follow-up is extended or, put another way, the extent to which nutrition-related deaths are concentrated in the period immediately after measurement. Although most growth monitoring programs strive for monthly measurements, usually this can only be attained in the face of, or at the expense of, constraints on mothers' time and travel costs, staff time in health facilities, and perhaps sustained motivation levels in community-based programs. Depending on the strength of the length of follow-up effect and a variety of local considerations, it may therefore be appropriate to consider trade-offs between screening efficiency, frequency of measurement and choice of indicators.

In order to examine this question systematically it would be necessary to compare the sensitivity-specificity distributions of various indicators, across many cutoff points and over varying follow-up periods unconfounded by maturation and seasonal effects. Although several studies have provided data related to length of follow-up in general (Table 1, studies 1a, 2a, 2c, 5, 6b, 7 and 12), none of them provides the type of systematic analysis described above. Nonetheless, most of these studies agree in showing that mortality prediction is inversely related to length of follow-up. The notable exception is the study by Chen et al. (1980), which found that the relative risk of mortality for severely malnourished children was greater during the second year of follow-up than in the year immediately after measurement. This enigma is even more striking in that it was true for indicators of acute malnutrition (WA and WH) but not for chronic malnutrition (HA). Similar findings are evident in a study from Northern Malawi (Pelletier et al. 1994a).

Figure 12 (taken from Briend and Zimicki 1986) illustrates the effect of follow-up length on the sensitivity and specificity of simple arm circumference over a range of cutoff points. Prediction is clearly greatest in the mo immediately after measurement and continues to decline beyond 3 months of follow-up. In the original report of this study (study 2a, which used AC/HT as the indicator) it was observed that the relative risk declines rapidly from 19.8 in the first month to 12.2 in months 1–3 and 4.5 by months 4–6, leveling off at about 3.0 from that point through 18 months follow-up. The interpretation of both reports is seriously hampered, however, by the fact that all children in this study were measured in 1 month [De-

TABLE 17

***Mortality and relative risks of mortality for severely malnourished children as defined by anthropometric indicators over two follow-up periods, Bangladesh (study 5)*[1,2]**

Indicator/ cutoff point	Follow-up months 1–3		Follow-up months 4–6	
	Mortality rate	Relative risk	Mortality rate	Relative risk
WA < 60%	19.7	24.6	11.8	14.8
HA < 85%	9.4	7.8	6.0	2.5
WH < 80%	13.6	5.4	1.4	0.5
AC < 121	23.4	16.7	13.4	7.9
ACA < 75%	17.9	10.5	11.0	6.5
ACHT < 80%	15.2	10.1	8.4	5.6

[1] Data from Alam et al. (1989).

[2] Mortality rate = deaths per 1000 children per 3-mo period; Relative risk = defined in relation to the group above the cutoff point specified for each indicator.

cember 1970, as noted in Sommer and Lowenstein (1975)], which was the month after severe flooding. Thus, secular changes in mortality and nutritional status may have occurred due to the usual seasonal effects, the disaster and maturation of this cohort of children (1–4-year-olds).

Table 17 [from Alam et al. (1989)] shows that the mortality rate of children severely malnourished at baseline is higher in the 3 months after measurement than in the subsequent 3 months. Similarly, the relative risk of mortality for these children is higher in the first interval. This result is seen with each of the six indicators. However, it is not possible with the data provided to estimate the loss in predictive power, viz. sensitivity and specificity (at various cutoff points), which is incurred due to the longer follow-up period. Nor is it possible to single out the loss in power involved with a 3-month follow-up for all children as opposed to a 1-month period typically sought in most growth-monitoring programs. Similar problems exist in the other studies mentioned above, and thus it is not possible at this time to provide concrete guidance concerning the implications of changing the measurement frequency in mortality prevention programs that use growth monitoring to screen high-risk children. Although the conservative strategy would be to continue to strive for monthly measurements, this may be done at the expense of mothers' time and clinic logistics, as mentioned above, which vary in importance from one setting to another.

Comparing the predictive ability of anthropometric indicators

Much of the literature on anthropometry-mortality relationships has been directed towards comparing indicators in order to identify the single indicator which is the best predictor of mortality. Of the 28 studies reviewed here, 15 of them provided data which implicitly or explicitly compared indicators in this respect. However, of these only eight employed appropriate criteria and methods for comparison, and only these studies are reviewed below.[16] Because of the large number of possible contrasts among indicators the results are considered in three stages. First, the results bearing on HA, WA and WH are described, followed by comparisons among the arm circumference-based indicators, and finally, comparisons among HA, WA and arm circumference indicators.

Table 18 shows the final results of the eight studies that used appropriate methods of comparison. It should be noted that in all cases the follow-up period varies between 6 and 24 months, thereby ensuring that large distortions are not introduced by studies with very short follow-up intervals (e.g., 1–3 months). However, the age range covered does vary substantially across studies and, in light of the potential for age-specific effects of indicators described earlier, is a factor that must be considered in comparing across studies.

In comparing across studies and across the various comparative criteria, the most consistent observation

[16]Brownie et al. (1986) have provided a detailed critique of the methods for comparing indicators up to that point in time and have provided empirical support for the use of the alternative comparative criteria adopted in this section. Their results suggest that the single best comparative criterion is the normalized distance statistic (d_n), followed by the maximum sum of sensitivity and specificity (MSS). The use of "receiver operating characteristic" (ROC) curves is also highly recommended. The least satisfactory basis for comparison,

TABLE 18

***Comparison of height-for-age, weight-for-age, and arm circumference-based indicators in relation to mortality*[1,2]**

Study	Age range in months	Follow-up period	ROC curves: High Sp	ROC curves: Low Sp	MSS	d_n
1b	12–23	24 mo	AC > WA > HA	WA > HA > AC	—	—
	12–23	6 mo	AC > WA > HA	HA > AC > WA	—	—
5	12–59	6 mo	AC > HA > WA	AC = WA > HA	—	—
14	6–59	6 mo	AC > WA > HA	—	AC > WA > HA	AC > WA > HA
17	6–59	12 mo	AC > WA > HA	WA > AC > HA	—	—
1d	12–23	24 mo Males	ACHT > AC > ACA > WA > HA		ACA = WA > AC > HA	ACA > AC > WA > HA
	12–23	24 mo Females	HA > WA > ACA > AC		HA > WA > AC = ACA	HA > WA > ACA > AC
	12–23	24 mo Combined	HA > AC > WA > ACA		HA > WA > AC ≥ ACA	HA > AC > WA > ACA

[1] Adapted from Bairagi (1981), Alam et al. (1989), Briend et al. (1989), Vella et al. (1994) and Cogill (1982). Study descriptions are provided in Table 1. *Studies 1b and 1d are based on the same basic data set; however, the results differ because of different methods of analysis. The indicator ranks shown here for study 1b are estimated from Figure 1 of the published report; however, the ROC curves for AC, WA, and HA intersect throughout the range of specificity analyzed. Study 1d reported quantitative estimates of the total area under each indicator's ROC curve, which forms a better basis for comparison in this case. Note: WH not shown because of its poor performance across all studies [Pelletier (1991a)].

[2] ROC = receiver operating characteristic; High SP = High specificity; Low Sp = Low specificity; MSS = maximum sum of sensitivity and specificity; d_n = normalized distance statistics; AC = arm circumference; WA = weight-for height; HA = height-for-age; ACHT = arm circumference-for-height; ACA = arm circumference-for age.

is that WH is the least effective predictor of mortality (and is therefore omitted from Table 18). Although one might hypothesize that this is because WH reflects acute malnutrition and may therefore predict only short-term mortality (i.e., <6 months of follow-up), the favorable performance of AC-based indicator (which also reflects wasting) in relation to HA and WA suggests this is not the case. It may be that WH has more measurement error than AC in these studies due to compounding the independent measurement errors in weight and height and the greater susceptibility of WH to variation in levels of hydration, stomach contents, posture during measurements, etc. Another lesson from this finding is that misleading conclusions can be derived if indicators are compared on the basis of simple analysis like that portrayed in Figures 1–4. These figures suggests that WH is among the most consistent indicator across studies, but the ROC curves reveal that WH has much lower sensitivity for a given specificity than other indicators.

Apart from the relatively poor performance of WH, when the variation in the strengths of each study is taken into account, Table 18 suggests that WA is superior to HA according to all performance criteria. The two notable exceptions are the study of Bairagi et al. (1985) (which is the most difficult to interpret due to small sample sizes and confounding by seasonality) and females in the Bangladesh data from Chen et al. (1980). The latter finding does not appear to be an artefact of study design and suggests that under certain circumstances the performance of indicators may be affected by child's sex as well as age. The results for sexes combined in that study resemble those for females because of the disproportionate number of female deaths in this sample.

When the three AC-based indicators are compared according to these same performance criteria (cf. Pelletier 1991) it appears that standardizing AC with HT does not add to the predictive ability of AC. Thus, AC/HT is consistently ranked poorly in such analyses, with the notable exception of males from the study by Chen et al. (1980) as reanalyzed by Cogill (1982). In addition, the results suggest that simple arm circumference (AC) is superior to AC corrected for age except among females in the data from Chen et al. (1980). The latter result may simply reflect age confounding, however, because AC increases sharply with age through 12 mo and more gradually thereafter (Frisancho 1990, Jelliffe 1966), exactly opposite to the trend in mortality with age.

Finally, Table 18 compares the AC-based indicators with HA and WA. For the criterion that is common to all studies (ROC curves), it appears that *at high specificities* simple AC is superior to HA and WA. This is true in all except the females and sexes combined in the study by Chen et al. (1980) as reanalyzed by Cogill (1982). This is not true in the range of low specificity, however, for those studies providing data in those ranges. Similar conclusions are reached based on the MSS and d_a criteria, although not all studies used these performance criteria.

The possibility that the superior performance of simple AC may be due to age confounding has been directly examined by Yip and Pelletier (1994) using data from Northern Malawi. Their analysis, based on ROC analysis, confirms that simple AC outperforms WA, whereas use of age-adjusted AC produces results comparable to WA.

Thus, on the basis of the few studies for which a valid basis for comparison exists, it appears that, at extremely low cutoff points (i.e., high specificity), simple arm circumference may be superior to HA or WA in predicting mortality within the subsequent 6–24 months. It is likely that this reflects age confounding, however. The indicator results are mixed at lower specificity values (less extreme cutoff points). More important than this conclusion, however, is the fact that the ranking of indicators with respect to these technical criteria often obscures the overall similarity that exists in their performance. Few studies employed statistical tests to determine whether the observed differences in prediction are statistically significant (Brownie et al. 1986). In addition, there are suggestions that the indicator rankings may be subject to modification by such factors as the child's sex and socioeconomic status (Cogill 1982), child's age (Katz et al. 1989; Pelletier et al. 1994a) and probably seasonality and other factors. Thus, the ultimate choice of indicators should take into consideration operational characteristics of the local programs, such as the expense and logistics of performing various measurements, the validity with which these measurements can be taken in the local context and the resources available for interventions (Haas and Habicht 1990). It should also be noted that mortality prediction (screening) is only one of the objectives for taking anthropometric measurements in health programs, and that AC is not necessarily the indicator best suited to other applications such as nutrition education (Ruel 1994).

Attained weight versus weight change in relation to mortality

In recent years it has become increasingly common for primary health care programs to stress indicators of weight change to complement or supplant measures of attained weight. Weight change has the theoretical advantage that it may provide early detection of growth faltering even among children whose attained weight is above conventional cutoff points for defining risk. In addition, by monitoring the change in weight rather than absolute position of the child's weight on the growth chart, it is explicitly acknowledged that

and the one most commonly employed up to that time, is relative risk.

some children may never approximate a normal position on the chart (often due to stunting in earlier years) and that attention should instead be directed towards maintaining a growth trajectory that is parallel to the normal trajectory.

Despite these theoretical advantages, the use of weight change indicators raises a number of operational and conceptual questions. First, because weight change indicators are subject to measurement errors twice (i.e., at a given visit and at a subsequent visit), the indicator has a larger proportion of false positives and negatives than a static indicator. Given that false positives imply targeting scarce intervention resources to the less needy and that false negatives imply a failure to intervene upon a child at high risk of dying, this is obviously a matter of some concern. Second, the use of weight change indicators may also be conceptually flawed depending on 1) the way in which weight change indicators are used (e.g., whether and how they are combined with static indicators), 2) the types of interventions actually available and 3) the explicit decision-making algorithm for linking indicators to interventions. For instance, it is not at all clear from the available studies that a child who is above 80% WA and shows growth faltering has a similar risk of death to a child at 60% WA who is growing parallel to the normal trajectory. It may well be reasonable to direct health education to the former, and education plus curative attention to the latter. However, it is not clear what should be done with the variety of possibilities between these two extremes. Nor is it clear whether health workers with rudimentary training can effectively distinguish between these different types of risk.

Although some of these issues must be resolved through operations research, there are two fundamental questions in relation to mortality prediction: 1) Do weight change indicators bear any relation at all to mortality risk; and 2) Do they provide significant improvement over indicators based on attained size. There are four reports in the literature that address these questions to some extent.

Of the four studies, three were able to demonstrate that the weight gain of survivors is significantly greater than that of children dying during the succeeding interval (Bairagi et al. 1985). The one negative study employed three different intervals for estimating weight change (2, 4 and 6 months) and examined this in relation to mortality in the following 12 months. However, in no case was a significant association found. Part of the problem may be that this study is limited by small sample sizes (15–23 deaths in various analyses) and is confounded by seasonality. The three studies producing positive results are reviewed below.

The Zaire study (Kasongo Project Team 1986) attempted to relate 2–4-months weight changes to mortality in the succeeding 100 days and found a weak association among 6–24-month-olds and a significant association among 25–59-month-olds. This association was only evident when large deviations in weight were considered (>0.5 WA Z-scores for older children and >1 WA Z-score for younger children). In no case was the association significant in the case of the cutoff point usually employed in actual programs (i.e., presence or absence of any weight loss). The authors conclude that, because the earlier report from this study found no association between mortality and attained weight (Kasongo Project Team 1983), then the weight change indicator is superior. However, the two analyses were performed on different subsamples (105 deaths for attained weight and 52 deaths for weight changes), thereby precluding direct comparison. Moreover the coverage of deaths in the study as a whole appears to be very low (see Table 1).

Both of the remaining studies (Briend and Bari 1989b, Yambi 1988) reported a significant association between mortality and weight change in the preceding interval, and both found that this effect is independent of attained weight in multivariate logistic regression models. The latter study also included initial weight in the model in order to control for the correlation between weight change and initial weight (a portion of which is induced by measurement error). However, that study did not include a comparison of the performance of the weight change indicator in relation to attained weight (using ROC curves, MSS and d_a for instance) and, thus, it is not possible to evaluate its predictive ability against these standard criteria.

The Briend and Bari (1989b) study from Bangladesh is the only one to provide a complete analysis of the performance of weight change versus attained weight. According to all three criteria of performance (d_a, MSS and ROC curves) attained weight-for-age is found to be far superior to weight change as a predictor of mortality. Weight change over a 3-month period (which decreases the influence of measurement error as a proportion of the true change in weight) has a performance intermediate between weight-for-age and 1-month changes according to all three criteria. Thus, in terms of choosing a single best indicator among these three possibilities, it is clear that attained weight-for-age is the indicator of choice. Because of the compounding of measurement errors in calculating weight change, this result is even more likely to hold under actual program conditions in which measurement error is greater than in this study.

In recognition of the fact that programs may be designed to *combine* weight-for-age and weight changes for screening purposes, this study also examined the implications of this strategy. In multivariate logistic regression models, low weight-for-age (<60%) and 1-month weight changes (presence or absence of weight loss) are both found to be significant, although the odds ratio of the former is 9.7

compared with 3.7 for the latter. Both variables retain their significance even when morbidity symptoms are included in the models (measles and respiratory infections).[17] Even more telling, however, is the fact that, at any level of specificity, simple weight-for-age has higher sensitivity than 1-month weight loss alone or weight loss in combination with weight-for-age. Thus, the improved prediction of mortality (sensitivity) that is seen in logistic regression analysis is achieved at the expense of specificity. In an operational sense this implies that more deaths could be prevented by applying both indicators simultaneously; however, this would be accomplished at a higher cost to the program due to the greater number of false positives. Alternatively, if program resources are fixed, then the use of two indicators rather than simply weight-for-age may prevent *fewer* deaths, due to leakage of limited intervention resources to the less needy. In any case, this study suggests that better results could be achieved by simply raising the cutoff point of WA rather than adding weight loss to the screening protocol.

IMPLICATIONS FOR POLICY, PROGRAMS AND RESEARCH

An important observation from this review is that the published literature reflects a much stronger concern for programmatic issues arising from the anthropometry-mortality relationship (chiefly screening) than for policy-related issues. One indication of this is the fact that the PAR estimates presented in this paper, which are of central policy importance, had to be calculated from data culled from the original reports, and this was possible with only a fraction of the studies. It is also reflected in the general lack of attention to possible confounding and effect modification, which are likewise of central importance for some policy purposes and, to some extent, for screening purposes as well. Finally, it was noted that even the analyses directed at screening issues (e.g., those comparing the performance of various anthropometric indicators) often did not employ valid or consistent performance criteria. Although these criteria were only recently developed (Brownie et al. 1986, Cogill 1982), this nonetheless represents a weakness in many studies. Despite these overall weaknesses in the literature, it is possible to identify a number of conclusions of immediate policy and programmatic relevance. These are noted below, followed by suggestions for future research.

Implications for policy

The first important observation is that the fundamental relationship between anthropometric indicators and mortality is found in all studies except one (Kasongo). Second, the threshold effect reported by Chen et al. (1980) has not been found in most studies conducted since that time, including studies from the same geographic area (Matlab) and other areas of Bangladesh, Asia and Africa. An exception is the recent report from Zaire (Van Den Broeck et al. 1993) that may reflect the highly intervened communities in that study. Thus, although mortality risk does increase in an exponential fashion with decreasing levels of anthropometric status, there is nonetheless some elevated risk of mortality even in the mild-to-moderate region of most anthropometric indicators (WA, WH, and AC). This finding is counter to the notion that growth deficits in the mild-to-moderate range may represent physiological adaptations to a poor environment and do not carry any functional consequences of policy importance (Lipton 1983, Seckler 1982).

The policy importance of this latter finding is revealed by the estimates of population attributable risk, which indicate that a substantial proportion (e.g., 46–93% according to weight-for-age) of the "nutrition-related" deaths are found in the mild-to-moderate range of anthropometric indicators. Although the range of estimates for these proportions is rather wide, it is sufficient to indicate that policies designed to address the problem of severe malnutrition can aspire to prevent only a proportion of the nutrition-related deaths or total child deaths in the population. This appears to be especially important in countries outside of South Asia which have a much higher ratio of mild-to-moderate to severe malnutrition.

A related policy implication is that the cost-effectiveness of preventing nutrition-related deaths will be initially much lower and the absolute cost much higher, for policies and programs directed at the mild-to-moderate category. This is because of the much larger size of the population to be covered and the lower risk of death in this segment of the population (i.e., lower sensitivity) as compared with the severely malnourished segment. However, the failure to address this substantial number of nutrition-related deaths in the mild-to-moderate categories may limit the size of the reduction in child mortality rates for the population as a whole. It also leaves a large "reservoir" of mild-to-moderate cases from which severe cases are derived and thereby subjected to an even higher risk of death. It is likely that broader social and economic policy changes may be required to reduce mild-to-moderate malnutrition, in part because individual screening and intervention is so inefficient

[17] Although diarrhea is associated with lower weight changes, it is not associated with higher mortality in this sample and therefore does not affect these results. The authors attribute the lack of association between diarrhea and mortality to the presence of an intensive ORS program and access to one of the study's physicians for intensive care.

when dealing with relatively low risks distributed across a large proportion of the population.

An important caveat to this discussion is, of course, that it is still not clear to what extent the "anthropometry-related" deaths in these studies are truly "nutrition-related." To some extent they may be simply a function of other socioeconomic and behavioral factors being proxied by low anthropometric status. The literature is fairly convincing in suggesting that such confounding does not account for the association between *severe* malnutrition and mortality. The one study examining this issue in mild-to-moderate malnutrition found that the effects persists (and, indeed, is even stronger) within some socioeconomic strata. This latter finding requires confirmation in other populations because of the impact which variation in morbidity patterns and health care utilization may have on this result. The paper by Pelletier et al. (1994a) in this supplement addresses this issue in greater detail based on a study in Northern Malawi.

One of the provocative findings emerging from this synthesis is the observation that malnutrition acts as a potentiator of mortality (i.e., a multiplicative effect), rather than simply as another additive cause of mortality (Figures 5 and 6 and Pelletier et al. 1993). This result is fully consistent with the knowledge of a physiologic synergism between malnutrition and morbidity (Chandra 1991, Scrimshaw et al. 1968), but it has not been previously shown epidemiologically. Earlier intervention studies in Narangwal (Kielmann et al. 1978), Guatemala (Ascoli et al. 1967) and elsewhere (Gwatkin et al. 1980) have either suffered from inadequate sample sizes to demonstrate this effect or were not designed to do so. The implications for policy are as follows: 1) health interventions will have the biggest impact on mortality in the most malnourished populations; 2) nutritional changes (positive and negative) will have their biggest impact in populations with already high mortality levels and 3) the biggest impacts on mortality can be achieved by simultaneously improving health status *and* nutritional status and by targeting populations with the highest mortality rates.

These policy conclusions are not novel, but they are often undervalued, unheeded or otherwise unreflected in the policies of international agencies and governments. The present results provide added empirical support for increasing the level of the resources and attention to nutritional considerations in efforts to lower child mortality. This applies to direct interventions, as typically implemented through the health sector; however, in light of the evidence that the synergism operates over the mild-to-moderate range of malnutrition—and not just the severe range—it also reinforces the importance of explicitly considering the nutritional implications of policies and programmes in other sectors as well. The institutional mechanisms for undertaking effective policy review on an on-going basis remains one of the most difficult, but high-priority issues for attention.

Another result of immediate policy relevance is the observation that the effect of severe malnutrition on mortality is made substantially worse (four times worse) by the absence of breastfeeding (Table 14). In light of the already strong impact of severe malnutrition on mortality, the observation that it is quadrupled in the absence of breastfeeding is quite striking. This result, which does not appear to be confounded by child's age, was found in both of the studies examining this issue. Though it requires more extensive documentation, the apparent magnitude of the effect warrants immediate attention. It suggests not only that current breastfeeding programs should be maintained and strengthened, but also that special efforts should be made to ensure prolonged breastfeeding in children already severely malnourished or at high-risk for becoming so.

Implications for programs

The key programmatic issues arising from this literature relate to the choice of anthropometric indicators for screening, the frequency of measurement required for effective screening, and appropriate age/sex target groups. A positive finding of the review is that, when a uniform set of performance criteria are applied to anthropometric indicators, an emerging consensus is suggested concerning the best predictors of mortality. Specifically, it appears that, with qualifications, simple arm circumference and weight-for-age are superior to other indicators. Moreover, the two studies that explicitly compared the predictive ability of attained weight versus weight change found that attained weight was the better predictor and was not significantly improved by simultaneous consideration of weight change.

The important qualifications to the above conclusions are as follows: 1) Arm circumference outperforms the others (WA and HA) only at high specificities (i.e., extreme cutoff points) and is probably confounded by age; 2) There is suggestive evidence of some sex specificity (and possibly age-specificity) in the ranking of indicators and 3) Although this result arises from a number of studies that vary in age range and length of follow-up, there have been no systematic attempts to examine these relationships over a range of follow-up periods and a range of ages. To study these issues systematically in a single study will require much larger sample sizes than previously available (to accommodate sex- and age-specific analyses), a wide age range and the ability to distinguish several follow-up intervals.

Despite the limitations in the above evidence it is relevant to note that the choice of indicators and frequency of measurement depends in large part on local considerations (logistics and the specific objectives of

the program) and only in part on the technical criteria of screening efficiency (Haas and Habicht 1990). Programs designed to avert deaths in individual children, as in famines or clinic screening for acute cases, obviously require an indicator and a measurement frequency that reflect immediate risk and can be measured with reasonable ease and accuracy. The present results suggest that *for mortality prediction* AC with a low cutoff point is technically superior to WA (and both are superior to WH). This conclusion carries with it two caveats, however. First, simple AC is likely to be confounded with age if the program screens young children (<12 months) as well as old. Second, the studies giving rise to these results all employed a follow-up period of 6–24 months and, therefore, do not truly reflect the risk of death in the immediate future. There is a need to examine this issue with a much shorter follow-up period, using accepted methods for comparing indicators. Moreover, the marginal improvements in prediction using AC need to be weighed against any logistic considerations (e.g., switching from WA to AC in clinics), which vary according to local circumstances.

Programs with longer term child survival or nutritional improvement objectives (e.g., those delivering health and nutrition education) clearly do not require the same measurement frequency nor indicators of immediate risk. Here an important consideration may be the sensitivity of the indicator to recent health and nutritional conditions (to identify faltering growth early in the process rather than to prevent death per se). In this case change in WA would likely be superior to simple AC or change in AC. Unfortunately much of the literature designed to identify the single best indicator has overlooked these contextual and logistical considerations (Habicht and Pelletier 1990).

Implications for research

The studies included in this review have three interrelated characteristics that make it difficult to draw firm conclusions on several policy and programmatic issues: small sample sizes, variation in analytical methods and the failure to collect and/or use ancillary information in the analysis. In many cases this was compounded by the tendency to focus on questions with programmatic relevance (i.e., identification of the best anthropometric indicator) rather than policy relevance, even though the same data might be applied to both types of questions. This section highlights the key issues requiring further research, some of which might be undertaken with existing data.

The first set of issues relates to the possibility of effect modification, i.e., that the relationship between anthropometric indicators and mortality may differ according to child's age or sex, length of follow-up, season of measurement and a host of other factors. On the basis of a limited number of studies, the review supports the notion that such effect modifiers do exist and that they have significant implications for policies and programs. This is seen in the case of child's age and sex, which influence the relative predictive ability of different anthropometric indicators; in length of follow-up, in which mortality prediction appears to attenuate under longer follow-up and is likely to affect some indicators more than others; in season of measurement, which modifies the risk of death in malnourished children and in breastfeeding, which diminishes the risk of death among malnourished children compared with nonbreastfed children. Unfortunately, the evidence concerning these effect modifiers is quite patchy across the studies, in part due to the three factors noted above.

Greater resolution on these issues could be obtained by reanalyzing appropriate data sets already available. At a minimum it should be possible to examine effect modification by age and sex and possibly by length of follow-up in some studies. In light of the small sample sizes, one option would be to pool the data from several studies (notably the many studies from Bangladesh, but not limited to those) and perform an integrated analysis. An advantage of reanalysis, even if pooling is not performed, would be to adopt a standard set of criteria for comparing the performance of anthropometric indicators as described by Brownie et al. (1986).

Another issue requiring further attention is the strength of the association between mild-to-moderate malnutrition and mortality. In particular, it is important to examine whether the elevated mortality risk in this segment of the population is simply because households with malnourished children have poor environmental and behavioral characteristics, which are the same characteristics giving rise to higher mortality. The analyses to date have suggested that this is not the case with severely malnourished children, but only one study has singled out the mild-to-moderate cases for similar analysis. Moreover, there is a need to consider variation in age, length of follow-up and type of anthropometric indicator in these analyses for the reasons outlined above. Given that most of the children classified as malnourished by anthropometric criteria are of the mild-to-moderate type and that these appear to account for a significant proportion of the nutrition-related deaths in a population, this issue has important policy implications. Related to this is the need to better understand the quantitative impact of malnutrition on mortality due to different types of morbidity and the biological basis for that impact. Such knowledge would help decide the most appropriate mix of nutrition- and health-related interventions at national and subnational levels, by combining information on the morbidity profile and malnutrition prevalences to estimate locally relevant population attributable risks.

The observation that a given deficit in weight-for-age among South Asian children is associated with

lower mortality risk than in children from other regions is worthy of further investigation. It is consistent with earlier cross-national observations that the weight-for-age of South Asian populations is higher than expected, given their child death rates and other indicators of overall quality of life such as access to safe water, national food (calorie) availability, female literacy and infant mortality (Haaga et al. 1985).

A plausible hypothesis for these observations relates to the unusually high rate of low birthweight in South Asian populations and its effects on postnatal growth. According to the World Health Organization (W.H.O.) 31% of births from middle South Asia (including India and Bangladesh) result in low birthweight (<2,500 g), compared with 20% for the rest of Asia and 13–17% for various regions in Africa (World Health Organization 1980). Given the small stature of adult South Asian women (Eveleth and Tanner 1976), a significant proportion of this small size at birth is probably *not* attributable to poor maternal health and nutrition during pregnancy. Similarly, a significant proportion of the small size in early childhood may not reflect protein-energy malnutrition during this period. Rather, to some degree the smallness at birth and in early childhood may reflect relatively benign constitutional constraints to pre- and postnatal growth (Garn and Keating 1980, Gayle et al. 1987, Ounsted et al. 1988), which would not be expected to carry the same functional consequences in infancy and early childhood (Rasmussen et al. 1985). Research to test this hypothesis would require examination of possible interactions between birthweight and maternal characteristics (e.g., stature) as they relate to infant and child mortality.

Finally, it is worth noting that there are limitations to the policy inferences to be drawn from prospective, community-based mortality studies of the type reviewed here. In particular the demonstration that anthropometry predicts future mortality, even if unconfounded, does not mean that child growth and nutrition *must* be improved in order to reduce mortality. It may well be possible to improve health conditions and health care to the point where the incidence of disease is sharply reduced and the remaining morbidity is treated through competent health care. The evidence that malnutrition potentiates already existing mortality in a population certainly suggests that the success of health interventions would be accelerated through nutritional improvement. However, the most realistic and relevant way to estimate the role of nutrition in reducing child mortality, and the most cost-effective mix of strategies, is through intervention programs that improve nutritional and health conditions in real world settings.

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