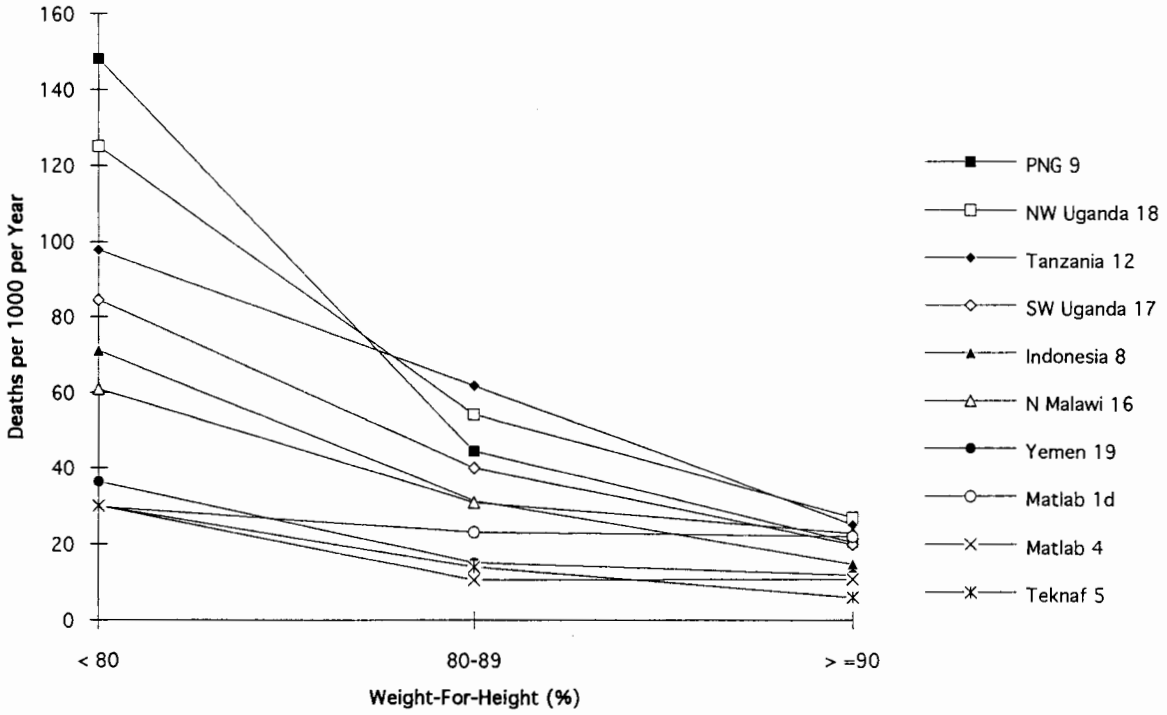


FIGURE 3 Relationship between child mortality and weight-for-height as a percentage of international median. Adapted from Heywood (1982), Vella et al. (1992), Yambi (1988), Vella et al. (1994), Katz et al. (1989), Pelletier et al. (1993), Bagenholm and Nasher (1989), Coghill (1982), Bairagi et al. (1985)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

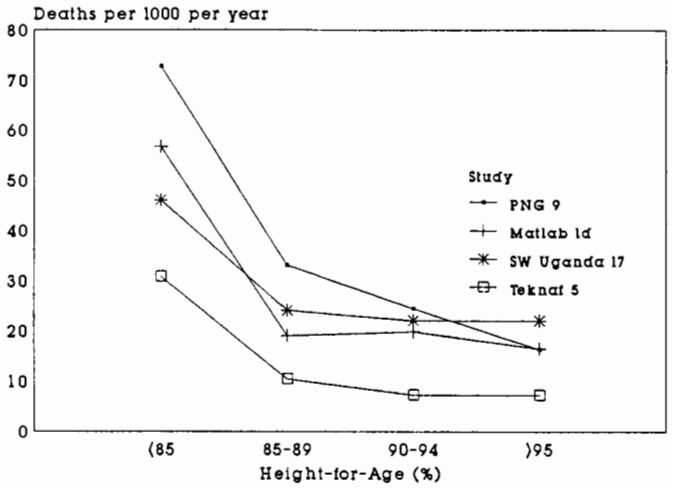
tributable to mild-to-moderate AC deficits (as defined here, 110–129 mm) is 42–63%. These percentages, of course, are highly sensitive to the choice of cutoff points delimiting these categories and largely reflect the cutoff points employed in the original reports.

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.



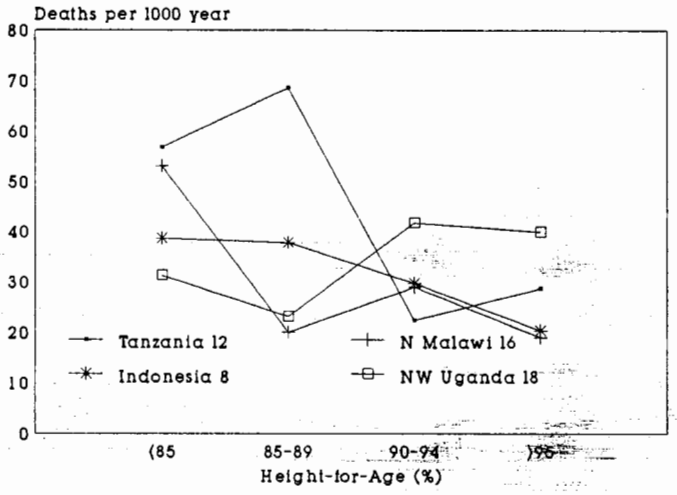


FIGURE 4 Relationship between child mortality and height-for-age as a percentage of international median. Adapted from Heywood (1982), Coghill (1982), Vella et al. (1994), Alam et al. (1989), Yambi (1988), Katz et al. (1989), Pelletier et al. (1993) and Vella et al. (1992). Study descriptions are provided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

	Height-for-age Z-score			
Child's age	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.

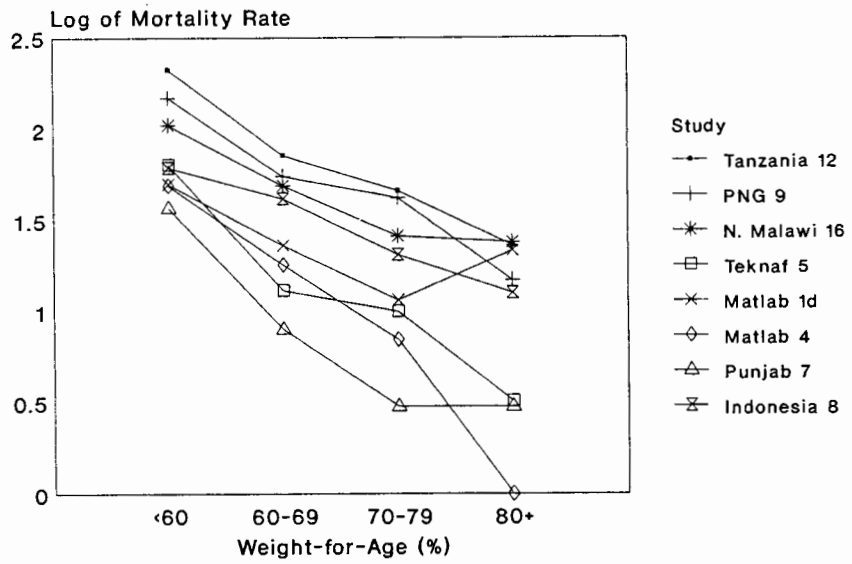


FIGURE 5 Relationship between log of mortality and weight-for-age. Adapted from Yambi (1988), Heywood (1982), Pelletier et al. (1994a), Alam et al. (1989), Cogill (1982), Bairagi et al. (1985) and Katz et al. (1989). Study descriptions are provided in Table 1. PNG = Papua New Guinea.

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-

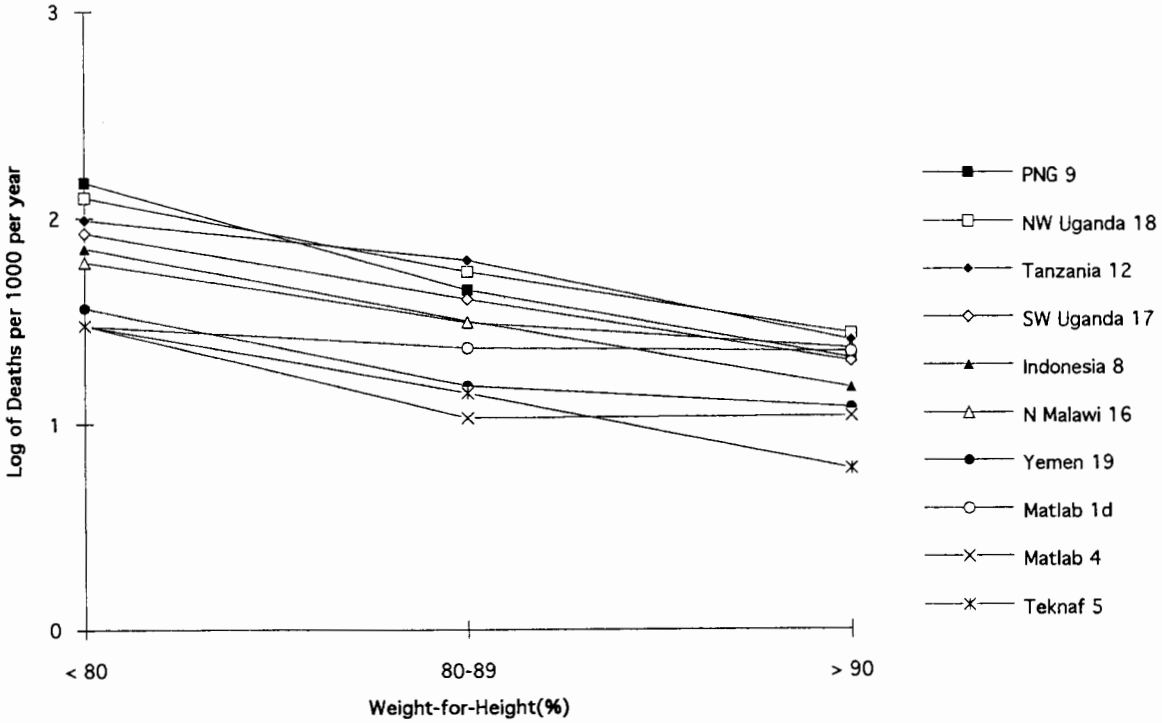


FIGURE 6 Relationship between log of mortality and weight-for-height as a percentage of international median. Adapted from Heywood (1982), Vella et al. (1992), Yambi (1988), Vella et al. (1994), Katz et al. (1989), Pelletier et al. (1994a), Bagenholm and Nasher (1989), Cogill (1982), Bairagi et al. (1985) 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]**

	Study					
			SW Uganda (17)[4] 6–59 mo			
Cause of death	Matlab (1a)[2] 12–23 mo WA	Matlab (20)[3] 6–36 mo Wasting	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

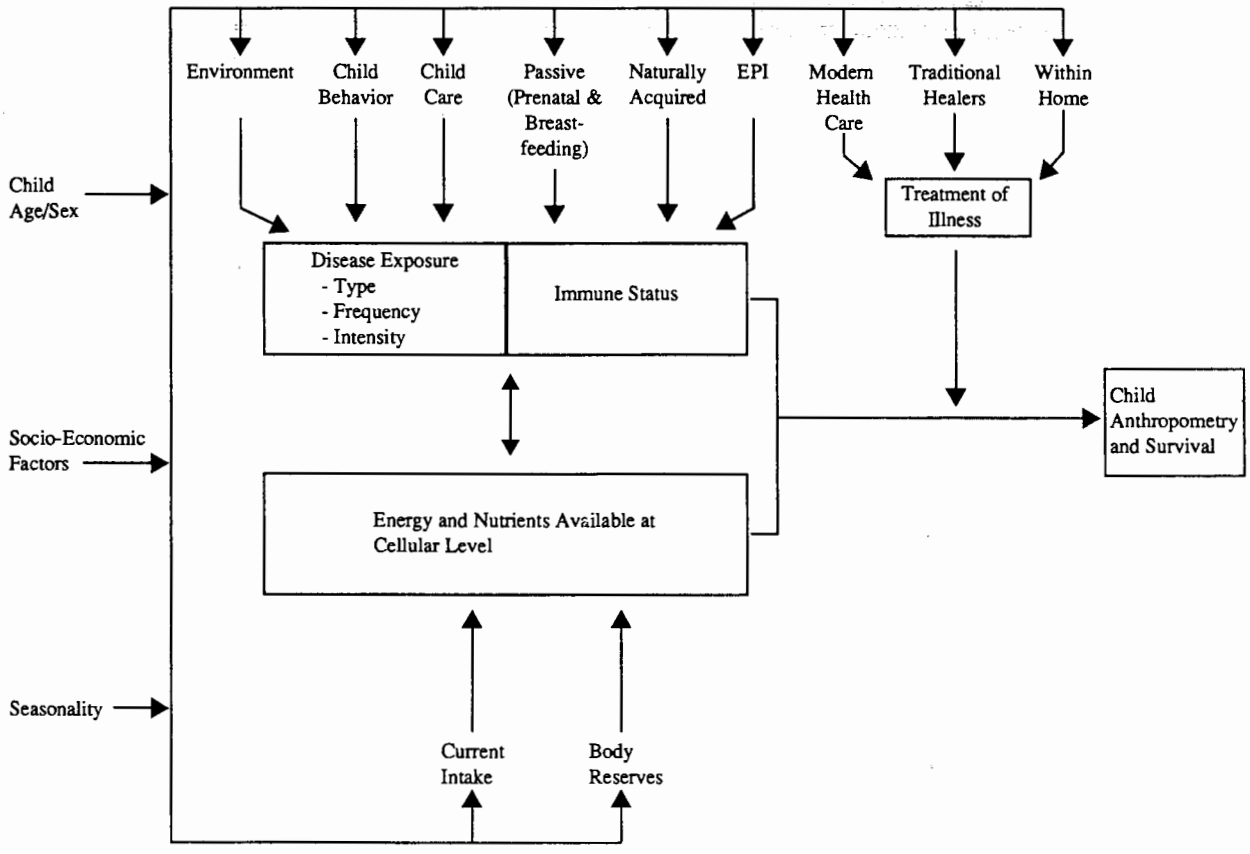


FIGURE 7 Biobehavioral determinants of child anthropometry and survival.

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.

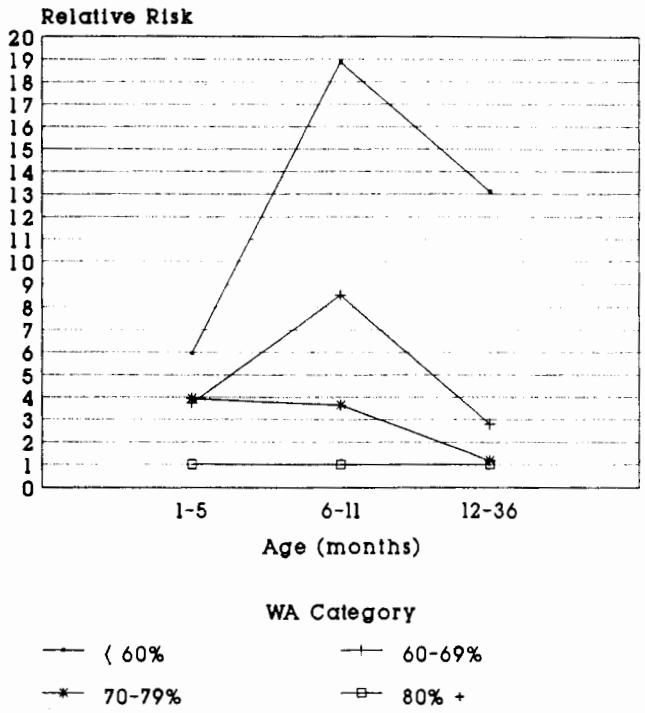


FIGURE 8 Punjab, India (study 7): relative risk of mortality according to child's age and weight-for-age. Data from Kielmann and McCord (1978). WA = weight-for-age.

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.

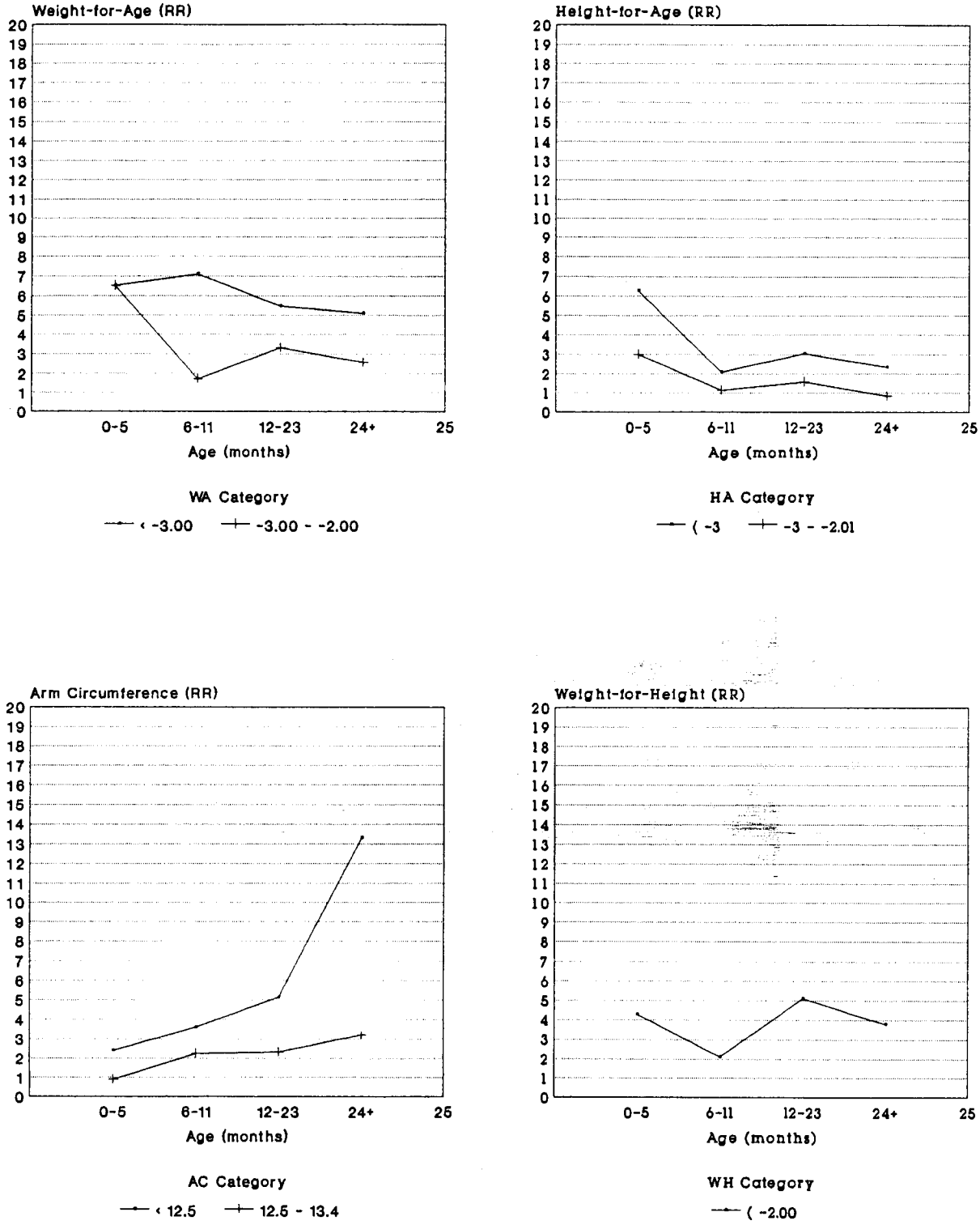


FIGURE 9 SW Uganda (study 17): relative risk of mortality according to child's age and four anthropometric indicators. WA = weight-for-age; HA = height-for-age; RR = relative risk; AC = arm circumference; WH = weight-for-height. Data from Vella et al. 1994.

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

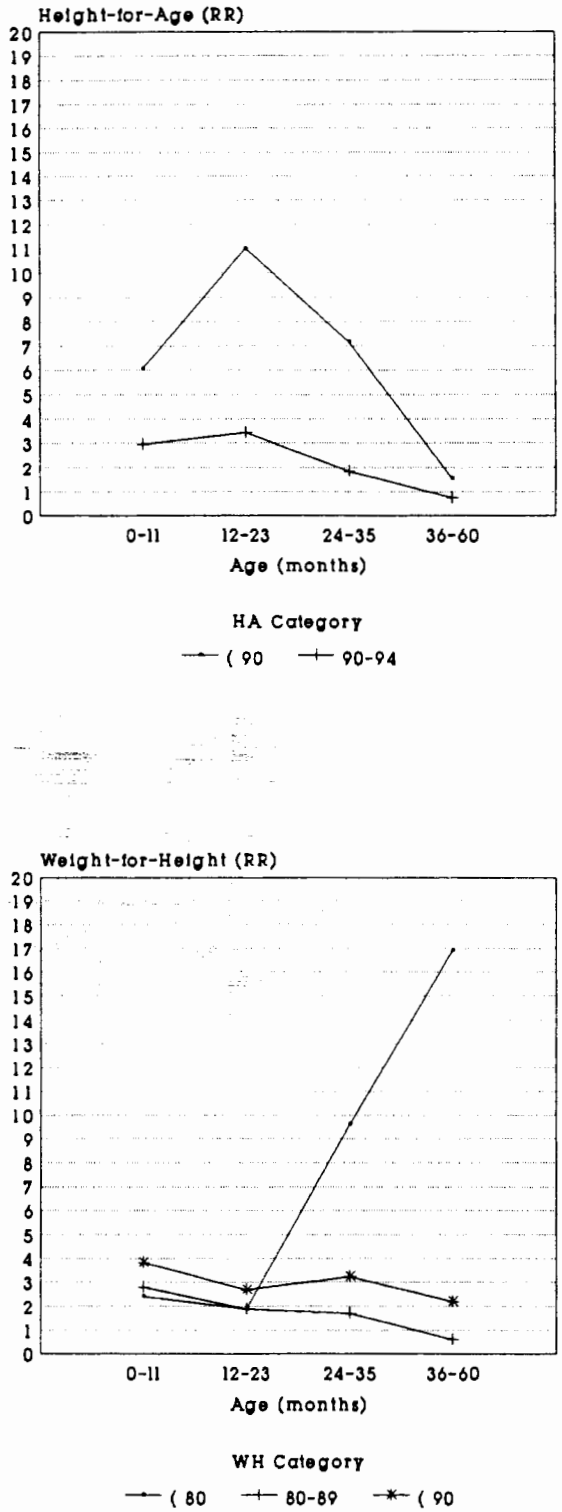


FIGURE 10 Indonesia (study 8): relative risk of mortality according to child's age and two anthropometric indicators. Adapted with permission from Katz et al. (1989). RR = relative risk; HA = height-for-age; WH = weight-for-height.

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-