Chapter 2

Childhood and maternal underweight

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Summary

Undernutrition, as measured by underweight status, has been associated with substantially increased risk of childhood mortality worldwide, but the magnitude of its effect on specific causes of mortality and morbidity has been less well described. We reviewed extant data and conducted statistical meta-analyses in order to determine the relative risk of disease and death attributable to underweight status. We used these estimates in combination with estimates of underweight prevalence among children aged 0–4 years and women of reproductive age (15–44 years) to calculate the global burden of disease attributable to undernutrition.

Underweight was defined for children aged 0–4 years as low weight-for-age relative to the National Center for Health Statistics/World Health Organization (NCHS/WHO) reference median. The theoretical-minimum-risk distribution for this population was equivalent to the reference population distribution, wherein 2.3% of children have a weight-for-age below –2 standard deviations (SDs) (weight-for-age <–2 SDs, or weight-for-age *z*-score [WAZ] <–2) and are classified as underweight. The relationship between low weight-for-age among children and mortality was analysed as a multiple categorical variable having four levels of exposure: WAZ <–3, WAZ –3 to –2, WAZ –2 to –1 and WAZ >–1 (reference category). Morbidity was analysed as a dichotomous variable, comparing WAZ <–2 to WAZ >–2 (reference category). Underweight was defined for women of reproductive age as pre-pregnant body mass index (BMI) below 20 kg/m^2. The outcomes chosen for review were major diseases and disabilities commonly associated with nutritional deficiencies. Sufficient prevalence and risk data were available to analyse childhood underweight status as a risk factor for mortality due to diarrhoeal disease, measles, malaria and pneumonia.

Childhood underweight status was also analysed as a risk factor for increased incidence of these infectious diseases. Maternal underweight was considered as a risk for increased risk of conditions arising during the perinatal period. The relationships were considered causal on the basis of consistent evidence observed across different populations and settings, and persistence of the association after adjustment for recognized confounders and effect modifiers such as age, breastfeeding status and prior morbidity. Current prevalence and risk data were not suitable for deriving burden estimates relating to other outcomes or older populations, although this does not imply the lack of risk in those populations. We therefore reviewed and summarized two major areas of undernutrition research without calculating burden estimates: (i) effects of maternal underweight status during pregnancy on maternal mortality and fetal death; and (ii) the association between undernutrition and cognitive function.

Prevalence of undernutrition among children aged 0–4 years was obtained from the WHO Global Database on Child Growth and Malnutrition, derived from a systematic analysis of raw data sets from 310 nationally representative nutritional surveys that collected data on child anthropometry in 112 countries. Categorical underweight estimates for most subregions[1] were calculated using multilevel modelling that adjusted for variability between regions, countries and surveys. Prevalence of underweight status was highest in SEAR-B (25.8%), SEAR-D (45.9%), AFR-D (32.2%), AFR-E (31.1%) and EMR-D (25.1%). Projections based on national trends suggested declining prevalence in most subregions over the next 30 years but increasing prevalence in the African subregions.

Cause-specific mortality was estimated by extending the methods of Pelletier et al. (1994) to data obtained from the investigators of 10 cohort studies in which both weight-for-age category (<–3 SDs; –3 to –2 SDs; –2 to –1 SDs; and >–1 SD) and cause-of-death information were available. All studies contributed information on weight-for-age and risk of diarrhoea, pneumonia and all-cause mortality. However, only six studies contributed information on deaths due to measles, and only three studies contributed information on deaths due to malaria or, in some cases, fever. By calculating the logarithm of the mortality rates by cause and anthropometric status in each country and utilizing weighted random effects models, we estimated the relation between weight-for-age and risk of death and calculated the relative risk of dying for each cause and all-cause mortality from these models. The relative risks of mortality due to low weight-for-age were elevated for each cause of death. The attributable fractions of mortality associated with weight-for-age below –1 SD were 44.8% for measles, 57.3% for malaria, 52.3% for pneumonia and 60.7% for diarrhoea.

Estimation of cause-specific morbidity was based on statistical meta-analysis of published data identified through systematic literature

searches of Medline and other databases. We selected longitudinal studies that compared incidence data according to past anthropometric status. Underweight status among preschool-age children was significantly associated with subsequent risk of diarrhoea and pneumonia episodes, but the association with malaria was not statistically significant. There was no evidence that underweight status influenced susceptibility to measles infection. The overall attributable fractions of morbidity associated with weight-for-age below –2 SDs were 16.5%, 5.3% and 8.2% for pneumonia, diarrhoea and malaria, respectively.

Mortality due to perinatal conditions was calculated by estimating the attributable fraction of neonatal mortality due to intrauterine growth retardation (IUGR) and then multiplying the value by the estimated attributable fraction of IUGR due to low maternal pre-pregnancy BMI for each subregion. Attributable fractions were based on prevalence and risk estimates from available published and unpublished sources.

Overall, undernutrition in children aged 0–4 years, as reflected in underweight or low weight-for-age, caused 3 599 800 deaths, including 815 900 diarrhoea deaths, 1 042 900 pneumonia deaths, 261 300 measles deaths and 549 200 malaria deaths. There were an additional 148 400 low-birth-weight neonatal deaths associated with low maternal pre-pregnancy BMI. The total number of child deaths associated with maternal or child underweight status, 3 748 200, represented 34.7% of all child deaths in the age group 0–4 years. The loss in disability-adjusted life years (DALYs) associated with these deaths was over 126 million. An additional 879 900 DALYs were lost due to increased morbidity from pneumonia, diarrhoea and malaria. Of the total DALYs associated with undernutrition, 44 million (32.0%) were lost in SEAR-D, 30 million (21.7%) in AFR-D, 33 million (23.9%) in AFR-E, 16 million (11.5%) in EMR-D and 8 million (5.8%) lost in WPR-B. Altogether, these five subregions accounted for 95% of the total DALYs lost due to undernutrition.

The true burden of disease associated with undernutrition extends beyond the narrow focus of this analysis. In addition to the effects of poor pre-pregnancy BMI, inadequate weight gain during pregnancy can lead to IUGR. Fetal growth retardation, in turn, may be associated with impaired immunocompetence, as well as increased risk of chronic disease in later life. Chronic undernutrition, especially in conjunction with poor environmental stimulation, is associated with impaired cognitive development, and severe undernutrition during infancy may contribute to lasting intellectual deficits. Chronic undernutrition among adults can contribute to diminished work capacity. Additional research is necessary into the prevalence and impact of undernutrition throughout the life cycle.

1. Introduction

Child undernutrition—measured as poor anthropometric status—is internationally recognized as an important public health indicator for monitoring nutritional status and health in populations. Young children are most vulnerable to undernutrition and face the greatest risk of its adverse consequences. Those who suffer from growth retardation as a result of poor diets and/or recurrent infections tend to have more frequent episodes of severe diarrhoea and are more susceptible to several infectious diseases, such as meningitis and pneumonia (Man et al. 1998; Tomkins and Watson 1989; Victora et al. 1994). A number of studies have demonstrated the association between increasing severity of anthropometric deficits and mortality, and undernutrition is thought to be a contributing factor in over half of all child deaths in developing countries (Pelletier 1994; Pelletier et al. 1993; Rice et al. 2000; Schroeder and Brown 1994). There is strong evidence that poor growth is associated with delayed mental development (de Onis 2001; Mendez and Adair 1999; Pollitt et al. 1993; WHO 1999a), and several studies have shown a relationship between impaired growth status and poor school performance as well as reduced intellectual achievement (Martorell et al. 1992; PAHO 1998). In addition, growth retardation in early childhood is associated with significant functional impairment in adult life (Martorell et al. 1992) and reduced work capacity (Spurr et al. 1977), which in turn has an impact on economic productivity.

The purpose of this chapter is to describe our review and analysis of existing data in order to calculate relative risks of specific causes of death and morbidity attributable to undernutrition (defined by low weight-for-age or "underweight"), and to use the estimates, in combination with estimates of underweight prevalence from a comprehensive WHO database, to calculate the global burden of disease associated with underweight status.

1.1 Determinants of childhood undernutrition

Undernutrition has several levels of determinants. Poverty is a strong underlying determinant that leads to household food insecurity, poor childcare, maternal undernutrition, unhealthy environments and poor health care. These factors then lead to the immediate determinants of childhood undernutrition, that is, low birth weight, inadequate dietary intake of nutrients and frequent infectious diseases (Baqui and Black 2002). Low birth weight, primarily due to IUGR in developing countries, is a consequence of maternal undernutrition prior to and during pregnancy and subsequently contributes to undernutrition in infancy and childhood (Villar and Belizan 1982). This is especially important in areas such as South Asia, where there is a very high prevalence of low birth weight. The diets of many children in developing countries are inadequate, and children in the first two years of life are at particular risk.

During this period, children have a high rate of growth and demand for calories, protein, essential fats, vitamins and minerals. Breastfeeding provides excellent nutritional support for six months (Kramer and Kakuma 2002), but unfortunately many children in such settings are given fluids and other foods before this age, resulting in reduction of breast milk intake and exposure to infectious agents causing diarrhoea (Lutter 2000). After six months of age, even when breastfeeding is continued, children frequently lack sufficient dietary intake, or consume a diet that is of poor nutritional content (WHO 1998). Again, these dietary deficiencies may involve not only the macronutrients, but also the so-called micronutrients, that is, vitamins and minerals. In young children, the frequently contaminated environments and poor childcare practices cause high rates of infectious disease. These infections result in a reduction in nutrient intake, as well as increased utilization and loss of nutrients, such as vitamin A and zinc.

The relative importance of the three immediate determinants of undernutrition varies by setting. The percentage of growth faltering (compared with an international reference population) due to diarrhoea in developing countries in Latin America, Africa and Asia has been reported to range from 10% to 80% (Black 1991). A study in Bangladesh simultaneously examined the role of diarrhoea, other febrile illnesses and dietary intake on weight gain (Becker et al. 1991). In a model using data from this study, it was estimated that improving dietary intake to recommended levels would have a slightly greater effect than eliminating diarrhoea and febrile illness; however, doing both at the same time would be necessary to achieve growth equivalent to an international reference population. There are a number of factors specific to particular developing country settings that can moderate the effect of illness on growth (Black 1991). For example, the adverse effect of diarrhoea on growth is less in exclusively breast-fed children than in children after weaning. Furthermore, an adequate diet, such as that provided in supplementation programmes, may prevent the adverse effect of diarrhoea on growth in some (Lutter et al. 1989) but not all settings (Bhandari et al. 2001). Thus, undernutrition is due to a variety of determinants that act along a pathway from poverty to dietary deficiency and frequent infectious diseases of childhood.

1.2 Other age groups

Undernutrition is not limited to young children. Over 815 million people of all ages are considered undernourished (FAO 2001), including roughly 243 million adults in developing countries who are considered severely underweight (ACC/SCN 2000a). Poor anthropometric status among adults and adolescents has been associated with maternal complications, diminished work capacity and increased risk of mortality (Martorell et al. 1992; Rotimi et al. 1999; Spurr et al. 1977; WHO 1995b). However, prevalence data for most adults, school-age children, adolescents and the

elderly are scarce, and understanding of the health effects of undernutrition in these populations is incomplete. There is a need for further research on undernutrition throughout the life cycle, especially as populations in developing countries go through demographic and epidemiological transitions.

1.3 Exposure variable

The internationally recommended way to assess undernutrition at the population level is to take body or anthropometric measurements (e.g. weight and height). Based on combinations of these body measurements anthropometric indices are constructed. These indices are essential for the interpretation of measurements, as the value for body weight alone, for example, has no meaning unless it is related to an individual's age or height (WHO 1995b). In children the three most commonly used anthropometric indices are weight-for-height, height-for-age and weight-for-age. These anthropometric indices can be expressed in terms of *z*-scores, percentiles, or percentage of median, which can then be used to compare a child or a group of children with a reference population.

Weight-for-age was chosen as the index of child nutritional status for this analysis because it is the most widely used in developing countries, allowing for the inclusion of the largest number of studies. Although it does not distinguish between wasting and stunting, low weight-for-age (underweight) represents a combination of both aspects and has a high positive predictive value as an indicator for child malnutrition in developing countries (WHO 1995b). Underweight is defined internationally as the proportion of preschool children falling below –2 SDs (weight-for-age <–2 SDs, or WAZ <–2) from the NCHS/WHO international reference median value for weight-for-age (de Onis and Blössner 1997; WHO 1995b). The burden of disease estimates for mortality were based on four weight-for-age categories: <–3 SDs; between –3 and –2 SDs; between –2 and –1 SDs; and the reference category of >–1 SD. The estimates for morbidity were based on two categories: weight-for-age <–2 SDs and the reference category of >–2 SDs.

Although the terms are often used synonymously, "underweight" should be distinguished from the terms "malnourished" and "undernourished". "Undernourished" and "malnourished" refer to the internal, physiological state of nutriture. "Underweight" is an observable anthropometric marker for that state, while the internal process can only be inferred.

BMI was selected as the index of adult nutritional status recommended by the International Dietary Energy Consultative Group of the Administrative Committee on Coordination/Sub-Committee on Nutrition (Shetty and James 1994). BMI is less biased by height differences than other adult indicators, such as absolute weight, and correlates with

health-related outcome variables such as overall mortality risk. For evaluation of nutritional status in relation to pregnancy, only pre-pregnant BMI was considered. Unlike the classification of underweight status in children, there is no comparable international reference for BMI among adults. A BMI <18.5 kg/m^2 represents chronic energy deficiency, and the Institute of Medicine recommends a BMI cut-off of <19.8 kg/m^2 as the definition for underweight (Institute of Medicine 1990; Shetty and James 1994). The cut-off of 20 kg/m^2 is commonly used in studies of maternal risk, and, therefore, underweight status among women of reproductive age was defined as BMI <20 kg/m^2 for this analysis. Further details on BMI can be found in the chapter on overweight and obesity (chapter 8).

1.4 Considerations in choice of child anthropometric indicator

Nutritional status can be assessed using clinical signs of malnutrition, biochemical indicators and anthropometry. Inadequacies in nutritional intake eventually alter functional capacity and result in many adverse health outcomes that are distinct expressions of malnutrition's different levels of severity. Initially, children adapt to inadequate diets through reduced physical activity and slowed rates of growth. At moderate degrees of malnutrition, activity and growth rates are affected to a greater degree, and in addition signs of wasting and some biochemical abnormalities (e.g. reduction in serum albumin), begin to show. At advanced stages of severity, all linear growth ceases, physical activity is severely curtailed, body wasting is marked and clinical signs (e.g. oedema, hair and skin changes) are noticeable. Anthropometry thus has an important advantage over other nutritional indicators: body measurements are sensitive over the full spectrum of malnutrition, whereas biochemical and clinical indicators are useful only at the extremes. In addition, anthropometric measurements are non-invasive, inexpensive and relatively easy to obtain. The main disadvantage of anthropometry is its lack of specificity, as changes in body measurements are also sensitive to several other factors, including infection, altitude, stress and genetic background (de Onis 2000).

A child's body responds to malnutrition in two ways that can be measured by anthropometry: (i) a deceleration or cessation of growth, which over the long-term results in low height-for-age or stunting; and (ii) body wasting, which is a short-term response to inadequate intakes, commonly assessed by weight relative to height. Height-for-age and weight-for-height thus discriminate between different biological processes, unlike weight-for-age, which could be low because of stunting (short stature) and/or wasting (recent weight loss). The current estimates relied exclusively on weight-for-age, which reflects body mass relative to chronological age. It is influenced by both the height of the child (height-for-age) and her weight (weight-for-height). Hence weight-

for-age cannot discriminate between short- and long-term forms of malnutrition given that children classified on its basis are a mixed group in terms of their nutritional status. In the 1990s weight-for-height emerged as a very important indicator (WHO 1995b) and, in fact, several authors have identified low weight-for-height as the indicator of choice for screening malnourished children who are at increased risk of dying (de Onis 2000).

1.5 Theoretical-minimum-exposure distribution

The prevalence of underweight status is defined as the proportion of preschool-age children falling below –2 SDs indicated from the NCHS/WHO international reference median value (de Onis and Blössner 1997; WHO 1995b). The theoretical-minimum-risk distribution is equivalent to this distribution, wherein 13.6% of children aged 0–4 years have weight-for-age between –1 to –2 SDs and 2.3% have a weight-for-age <–2 SDs.

Anthropometric values are compared across individuals or populations in relation to a set of reference values based upon a healthy population. The choice of reference population to assess nutritional status has a significant impact on the proportion of children identified as being malnourished and, in turn, important implications for establishing relationships between nutritional status and functional outcomes (WHO 1995a). Much has been written about growth references, but unanswered questions remain about the many factors that determine human growth and indeed what constitutes "normal" growth. WHO has been recommending since the late 1970s the use of the NCHS/WHO international reference population *z*-scores for the comparison and presentation of child malnutrition data (Waterlow et al. 1977). A detailed account of the different growth references used prior to the current international reference is provided elsewhere (de Onis and Yip 1996). In the mid-1990s the NCHS/WHO international reference was found to have important technical and biological drawbacks (de Onis and Habicht 1996; de Onis and Yip 1996; WHO 1995a). Consequently, an international effort coordinated by WHO is under way to develop a new international growth reference for infants and young children (de Onis et al. 1997). The new international reference—which will be constructed from primary data collected for this purpose—includes a number of features that, taken together, will result in a reference population substantially different from the existing ones (de Onis et al. 2001). An important characteristic of this new reference is that it will be based on a truly international sample. Six countries, representing the major global geographic regions, are participating in this effort. Another notable feature is that it takes the breast-fed infant as the biological "norm", recognizing the health and nutritional benefits of breastfeeding. The extent to which the new curves differ from the current ones in shape and the spread of values around the mean will affect the relationship—established using the NCHS/WHO

reference values—between child anthropometry and functional outcomes such as morbidity and mortality.

Once a reference population has been selected, it is necessary to determine the limits of "normality". Current practice is to use a defined cut-off point using one of the three available classification systems for comparing a child, or a group of children, to the reference population: *z*-scores (SD scores), percentiles and percentage of median. The use of a statistically defined cut-off point (e.g. –2 SD *z*-score) is not unique to anthropometry; indeed, it is widely applied in many fields of biology and medicine. Nevertheless, it is important to bear in mind that using a cut-off-based criterion to define what is "abnormal" is arbitrary. In reality, there are not two distinct populations—one well nourished and the other malnourished—but rather a continuous gradation of nutritional status (de Onis 2000). The risk of undesirable health outcomes such as mortality does not change dramatically by simply crossing the cut-off line; risks are continuous within the "normal" range. For many purposes, the best descriptor of a population's nutritional status is the mean *z*-score, which in less developed environments is usually shifted to the left. This concept is based on data from many different countries which showed a high consistency in the SD of weight-for-height among young children (de Onis and Blössner 1997). Even under extreme conditions, such as during famines, where the mean *z*-score is two or three units below the reference, the value of the SD of *z*-scores is very close to unity. This shows that the entire distribution is shifted so that all individuals, not only those below a given cut-off point, are affected (Rose 1985). Taking such a population-approach resolves the problem of focusing solely on the severely malnourished subpopulation falling below a certain cut-off. In most instances, the mild and moderately malnourished subpopulations will be of greater importance from a public health perspective because there are many more children in these categories than in the severely malnourished one. For this reason, estimates are presented using low weight-for-age as both a dichotomous and multi-categorical variable when possible.

2. Prevalence of underweight among children

Over the years the WHO Department of Nutrition has been monitoring trends in child malnutrition using anthropometric data. A major difficulty has been the lack of comparability of survey results. Although many nutritional surveys have been conducted since the 1970s, many of them had used distinct definitions of malnutrition (i.e. different anthropometric indicators, reporting systems, cut-off points and reference values) making comparison among studies difficult. This lack of comparable data prompted the beginning of WHO's systematic collection and standardization of data on the nutritional status of the world's population of children <5 years. Initial results of this effort, published in 1993 (de

Onis et al. 1993), were updated in 1997 and presented together with estimates of trends in child growth retardation in developing countries (de Onis and Blössner 1997). A more recent analysis using multilevel modelling updated these earlier estimates, describing trends in child malnutrition from 1980–2005 (de Onis et al. 2000).

2.1 Data sources and quality control

Cross-sectional data on the prevalence of underweight were obtained from nationally representative nutritional surveys that collected data on child anthropometry and are included in the WHO Global Database on Child Growth and Malnutrition. This database was initiated in 1986 to compile, standardize and disseminate the results of nutritional surveys performed in both developing and developed countries (de Onis and Blössner 1997). A distinct feature of this database is the systematic analysis of raw data sets in a standard format to produce comparable results. Only nationally representative data have been used for the present analysis. Three hundred and ten national nutrition surveys from 112 countries carried out from 1965 onwards were analysed to estimate the prevalence of underweight in children. The majority of surveys were conducted by either the relevant national ministry of the country concerned, by the Demographic and Health Surveys (DHS) programme of Macro International, or by the Multiple Indicators Cluster Surveys (MICS) carried out by the United Nations Children's Fund (UNICEF). Appendix A lists all national surveys included in the present analysis by country in alphabetical order.

Survey data for inclusion in the WHO Global Database on Child Growth and Malnutrition are identified by various ways and mechanisms.

- An automated Medline search provides results according to the established search history following the weekly online Medline updates. Selected abstracts are reviewed and relevant surveys with data searched for in the library. Data are extracted and frequently the principal investigators and/or data holders are contacted to complement the data provided in the article and/or give clarification on methodological issues.
- A wide network of collaborators, including international organizations (e.g. UNICEF, Food and Agriculture Organization of the United Nations [FAO]), nongovernmental organizations (NGOs) (e.g. Helen Keller International), Ministries of Health and National Institutes of Nutrition, as well as independent institutions (e.g. Macro International) and individual researchers provide their data directly to the WHO Global Database. In case of queries on the data the same procedure as above takes place to clarify any unclear issues.
- Principal investigators contact the WHO Global Database to enquire about possible inclusion of their data into the Global Database.

- Other WHO database managers within WHO headquarters share data sets and survey documents with the Global Database.

The sampling methods for each of the survey data used in this analysis were carefully reviewed to ensure national representativeness. Multistage random sampling methods were applied for sample selection in the majority of countries. Only a few countries, such as Argentina, Chile, Croatia, Uruguay and Venezuela based their estimates on well-established national nutritional surveillance systems with high population coverage (country-specific details on sampling procedures are available from the authors on request). Surveys generally followed standard procedures of measuring length up to 24 months of age and height from 24 months onwards. The anthropometric measurement techniques used in each survey are described in the comprehensive survey reports, which are available on request. Some surveys included information on reliability of the measurements while others did not.

Survey results were checked for inconsistencies between the estimates based on height-for-age, weight-for-age and weight-for-height. The observed SDs of the *z*-score distribution were used to assess the quality of the survey results. With accurate age estimates and anthropometric measurements, the SDs of the observed height-for-age, weight-for-age and weight-for-height *z*-score distributions should be relatively constant and close to the expected value of 1.0 for the reference distribution (ranging within approximately 0.2 units). This nearly constant SD in height-based and weight-based *z*-score distributions provides an opportunity to assess data quality (WHO 1995b). Surveys with a SD outside the expected ranges were subjected to closer examination because of possible problems related to age assessment and anthropometric measurements. Surveys with inaccurate data resulting from measurement error or incorrect age reporting were excluded from this analysis.

2.2 Statistical analysis

The analysis followed three separate steps, which are described below. The number of countries and percentage of population aged <5 years covered by data for each subregion are listed in Appendix B. The population aged <5 years derived from the 1998 revision of the UN population estimates (UN 1999) for the particular survey year were added as population weights. As there were no significant differences observed between male and female child underweight prevalence, the model exercise included data only for sexes combined, and estimates were assumed to be the same for males and females.

Primary analysis of raw data sets

As an essential first step in producing subregional estimates, primary analysis of the 310 national raw data sets was conducted to produce

standardized estimates of prevalence of underweight as defined in section 1.1. This was necessary because many nutritional surveys used distinct definitions of malnutrition (i.e. different anthropometric indicators, reporting systems, cut-off points and reference values) making impossible the pooling of reported prevalences. There were three steps involved in this analysis: (i) identifying the data holder for each of the individual surveys; (ii) requesting re-analysis of the original data sets following the standardized definition, or otherwise, requesting a copy of the raw data set for standard analysis by the WHO Department of Nutrition; and (iii) initiating quality control procedures for each individual survey. Country-specific prevalences of child underweight by age, sex and subregion derived for each of the national surveys listed in Appendix A can be found on the web site of the WHO Global Database on Child Growth and Malnutrition at http://www.who.int/nutgrowthdb.

Subregional estimates: underweight as a dichotomous variable

The methodology applied to derive the underweight estimates depended on the availability of data points for the different subregions. In detail these were as follows: for AFR-D, AFR-E, AMR-B, AMR-D, EMR-B, EMR-D, SEAR-B, SEAR-D and WPR-B the underweight estimates were calculated using a multilevel model. Multilevel modelling allows for more than one component of variation, which in this case includes between subregions, between countries within subregions and between surveys over time within countries. This multilevel model is an extended form of regression, which separates estimates for the three levels of variation, and the total variation is obtained by combining over the three levels. To adjust for skewness in the distribution of prevalence, the model used the logit transform of the underweight prevalence, with the additional advantage of stabilizing the variance. Because estimates were calculated on the logistic scale, estimated prevalence and their CIs were derived by back-transformation. The nature of the logistic function ensured that all estimated prevalence and their CIs would lie above zero. CIs for prevalences close to zero were asymmetric, and were narrower than for values close to 50%—these are intrinsic properties of the logistic function (Armitage and Berry 1994).

Multilevel modelling was implemented in the Statistical Analysis System (SAS) Proc Mixed program, a procedure that accommodates both categorical and continuous covariates and incomplete series of time measurements, and allows for the added variability introduced by the multiple levels of analysis. Analyses were conducted separately for each subregion, given that the trend of underweight between subregions could differ substantially.

Separate analysis for each subregion required that two rather than three levels be modelled, thus reducing complexity of the model. An assumption of the analyses was that the extent of available data for countries was not related to the prevalence of underweight. In other

words, it was assumed that the trend of the prevalence over time for a subregion could be estimated in an unbiased manner from the data available. Under this assumption the model accommodated the variable patterns of available surveys for the countries. Countries with only one survey contributed information to the estimation of the overall intercept, whereas countries with more than one survey also contributed to the estimation of the regression coefficient(s) relating the trend to the survey year. The models specified a linear trend relationship between prevalence of underweight and survey year. Thus, these models assumed that the rate of change in the prevalence of underweight was constant.

Possible non-linear trends were examined by including quadratic and cubic polynomial terms. There was some evidence for non-linear relationship in only one of the subregions (SEAR-D), showing slightly larger differences between countries and indicating a better fit using the quadratic and cubic functions. However, due to unrealistic drops in prevalence the model outcome was discarded. Further explanation on the multilevel modelling approach to derive estimates has been described elsewhere (ACC/SCN 2000b; de Onis et al. 2000).

No modelling was possible in AMR-A, EUR-A, EUR-B, EUR-C and WPR-A due to lack of sufficient national survey data. To derive estimates for these subregions we proceeded as follows:

- In AMR-A and EUR-A, which are composed of developed countries, we assumed that children have a similar nutritional status as the reference population. Therefore, these two subregions were assigned the prevalence values derived from the international reference population, i.e. 2.3% (theoretical minimum). For these subregions no CIs were available and uncertainty was assumed as zero.
- In EUR-B, EUR-C and WPR-A, we calculated the weighted mean prevalence of underweight following the method of de Onis and Blössner (2000) for estimating prevalence and trends of overweight among preschool-age children in developing countries. Based on the available survey data the CIs fell within a 95% CI of: 0.1–15.5 in EUR-B; 0.1–5.1 in EUR-C; and 0.7–6.9 in WPR-A.

Subregional estimates: underweight as a multi-categorical variable

Growth in populations of children is normally distributed. In a standard normal distribution the SD is 1 and the mean is located at 0. As there is a relationship between the prevalence falling below the cut-off (<–2 SDs) and the mean *z*-score, to convert the mean *z*-score to a prevalence based on <–2 SDs, the probit function, which converts *z*-score values to cumulative percentiles, was used as recommended by an Expert Committee (WHO 1995b). To derive probability that the *z*-score of a child is between two cut-off points (<–3 SDs; –3 SDs to –2 SDs; –2 SDs to –1 SD; and >–1 SD), we calculated the difference between the two cumulative

probabilities obtained from the probit function. Prevalence data below –3 SDs were derived by subtracting the calculated values for the interval –3 to –2 SDs from the total of the prevalence below <–2 SDs provided by the categorical analysis (see Table 2.1 and section 4.2), which included all ranges below this cut-off.

2.3 Exposure estimates

The prevalence of low weight-for-age among children aged 0–4 years by subregion is listed as a dichotomous variable in Table 2.1 (a) and as a multi-categorical variable in Table 2.1 (b), below.

3. Prevalence of underweight among women of reproductive age

Subregional BMI estimates listed in Table 2.2 are from chapter 8. Underweight status is widespread among women of reproductive age in developing countries, especially in sub-Saharan Africa and South Asia. Of 26 sub-Saharan African countries with recent (<10 years old) survey estimates, 23 have greater than 30% prevalence of BMI <20 kg/m^2—five of which (Chad, Eritrea, Ethiopia, Madagascar and the Niger) have prevalence >50%. Among South Asian women, 70–76% in Bangladesh and India have a BMI <20 kg/m^2, along with 54–61% in Cambodia and Nepal. Low BMI is less common in the Americas, where prevalence of

Table 2.1(a) Underweight prevalence by subregion[a]

Subregion	*Prevalence of underweight (% below –2 SDs)*	*95% CI*
AFR-D	32.2	(26.7–38.2)
AFR-E	31.0	(24.7–38.0)
AMR-A	2.3	(2.0–2.6)
AMR-B	5.0	(3.8–6.6)
AMR-D	12.4	(7.7–19.6)
EMR-B	8.1	(5.4–12.1)
EMR-D	25.1	(14.5–39.7)
EUR-A	2.3	(2.0–2.6)
EUR-B	7.6	(0.1–15.5)
EUR-C	2.6	(0.1–5.1)
SEAR-B	25.8	(16.3–38.4)
SEAR-D	45.9	(40.7–51.2)
WPR-A	3.8	(0.7–6.9)
WPR-B	16.0	(12.7–19.9)

[a] Dichotomous estimate.

Table 2.1(b) Mean z-scores and underweight prevalence by weight-for-age category and subregion[a]

Subregion	Mean z-score	Percentage of children in weight-for-age category			
		<–3 SDs	>–3 to <–2 SDs	>–2 to <–1 SD	>–1 SD to <0
AFR-D	–1.54	7.1 (4.0–10.4)	25.1 (19.7–30.3)	38.3 (32.3–44.2)	23.3 (18.1–28.5)
AFR-E	–1.5	6.8 (3.1–10.3)	24.2 (18.1–30.4)	38.3 (31.3–45.3)	24.2 (17.9–30.2)
AMR-A	0	0.1 (0.1–0.2)	2.1 (1.9–2.4)	13.6 (13.0–14.3)	34.1 (33.3–35.1)
AMR-B	–0.35	0.5 (0.0–0.8)	4.5 (3.3–6.0)	20.8 (18.4–23.7)	37.9 (34.8–41.1)
AMR-D	–0.84	1.6 (0.0–3.8)	10.8 (5.3–16.5)	31.3 (23.1–39.8)	36.3 (27.6–44.9)
EMR-B	–0.6	0.8 (0.0–1.9)	7.3 (4.1–10.5)	26.3 (21.1–31.9)	38.1 (32.2–44.1)
EMR-D	–1.33	4.7 (0.0–10.9)	20.4 (8.7–32.1)	37.8 (23.7–51.9)	27.9 (14.9–41.0)
EUR-A	0	0.1 (0.1–0.2)	2.1 (1.9–2.4)	13.6 (13.0–14.3)	34.1 (33.3–35.1)
EUR-B	–0.57	0.7 (0.0–3.3)	6.9 (0.0–14.2)	25.7 (13.0–38.4)	38.2 (24.1–52.3)
EUR-C	–0.05	0.2 (0.0–0.8)	2.4 (0.0–4.9)	14.5 (9.1–20.2)	34.9 (27.5–42.5)
SEAR-B	–1.35	5.0 (0.0–10.4)	20.8 (10.6–31.1)	37.9 (25.7–50.2)	27.5 (16.2–38.7)
SEAR-D	–1.9	13.4 (9.9–17.1)	32.5 (27.5–37.3)	35.8 (30.6–40.7)	15.5 (11.8–19.4)
WPR-A	–0.22	0.3 (0.0–1.1)	3.5 (0.5–6.5)	18.0 (11.9–24.4)	36.9 (29.2–44.8)
WPR-B	–1	2.3 (0.8–3.8)	13.6 (10.3–17.1)	34.1 (29.6–38.9)	34.1 (29.4–38.7)

[a] Multi-categorical estimate.

Table 2.2 BMI distribution of women aged 15–44 years, by subregion

Subregion	Mean BMI (kg/m^2) (SD)		Percentage of women with BMI ≤20 kg/m^2	
	15–29 years	30–44 years	15–29 years	30–44 years
AFR-D	20.6 (3.5)	22.1 (3.9)	43.3	29.5
AFR-E	21.2 (3.9)	22.9 (4.8)	37.8	27.4
AMR-A	23.8 (5.7)	26.1 (7.1)	25.1	19.5
AMR-B	23.5 (4.7)	26.2 (5.1)	22.7	11.1
AMR-D	23.7 (3.1)	25.8 (4.4)	11.7	9.3
EMR-B	22.5 (4.4)	25.8 (5.1)	28.4	12.7
EMR-D	21.6 (6.3)	23.8 (8.5)	40.1	32.6
EUR-A	23.3 (4.1)	25.1 (4.7)	20.9	13.8
EUR-B	22.7 (3.9)	25.6 (5.2)	24.5	14.0
EUR-C	22.7 (3.9)	26.5 (5.0)	24.5	9.7
SEAR-B	20.5 (3.5)	22.7 (2.3)	44.4	12.1
SEAR-D	19.5 (3.0)	20.8 (4.0)	56.7	42.1
WPR-A	20.8 (3.5)	22.5 (4.1)	40.9	27.1
WPR-B	21.9 (4.2)	22.8 (4.1)	32.6	24.8

BMI <20 kg/m^2 among women of reproductive age is generally below 20% (ACC/SCN 2000b; DHS web site, http://www.measuredhs.com).

4. Risk factor–disease relationships

A synergistic relationship between malnutrition and infection has been recognized for decades (Scrimshaw et al. 1968), providing a foundation for the choice of health outcomes in this analysis, but the bi-directional nature of the relationship complicates attributing causality. The changes in body size used as a marker for undernutrition in this analysis are commonly the result, in part, of previous infections, raising the possibility that co-existing infection is responsible for some of the observed risk relationship. Age, sex, socioeconomic status, season, breastfeeding status and behavioural factors will also influence both anthropometric status and risk of infection (Pelletier 1994).

Undernutrition is most prevalent where poverty persists, and it is often difficult to isolate the effects of undernutrition from the complex web of environmental and socioeconomic factors that contribute to mortality and morbidity in such surroundings. In reviewing the various risk relationships, the likelihood of causality was considered according to Hill's standards (Hill 1965). In each case, there was judged to be sufficient biological plausibility and experimental evidence to support the relationship. We chose risk relationships that have been reported by multiple researchers in different populations under different circumstances. The associations generally, albeit not always, persisted after adjustment for prior morbidity and known confounders. Further, we selected for analysis only those studies in which anthropometric assessment clearly preceded the observed health outcome. In previous analyses (Pelletier 1994) and in our analysis, the consistency of the risk relationship between weight-for-age and mortality in different settings with quite different mortality rates and environmental conditions strongly suggests a causal rather than confounding relationship.

The relationship between undernutrition and disease may be mediated through many biological mechanisms. The lack of adequate protein and energy is associated with numerous immunological effects, including impairment of antibody and complement formation; atrophy of the thymus and other lymphoid tissues; reduction in T-lymphocytes; depressed lymphocyte activation; decreased secretory immunoglobin A (IgA) concentrations; and delayed cutaneous hypersensitivity reaction (Rivera and Martorell 1988; Scrimshaw and SanGiovanni 1997). Undernutrition is associated with impaired turnover and maturation of intestinal epithelial cells and compromised epithelial tissue integrity (Patwari 1999). Other anatomic barriers and secretory forms of protection such as lysozymes and mucus are also altered by undernutrition (Scrimshaw and SanGiovanni 1997).

In the course of the review, several health outcomes were dropped from further consideration toward the final burden of disease estimate. In many instances, there was insufficient published evidence within a developing country setting to support a causal relationship between low weight-for-age or BMI and the outcome, although the relationship might be more widely supported given a different choice of anthropometric indicator. Ultimately, risk estimates were derived for eight outcomes for childhood underweight:

- malaria mortality;
- malaria incidence;
- pneumonia (acute lower respiratory infection [ALRI]) mortality;
- pneumonia/ALRI incidence;
- diarrhoea mortality;
- diarrhoea incidence;
- measles mortality; and
- all-cause child mortality.

All of the estimates apply to children aged 0–4 years. In addition, mortality due to perinatal causes among neonates was attributed to maternal underweight status. There was insufficient evidence to evaluate relationships among older age groups. Although some individual studies might suggest sex differences, the summary risk estimates here were assumed to be the same for males and females. Further, although risk relationships might vary by setting, the risk estimates were regarded in the analysis as constant across all subregions of the world.

Other outcomes such as cognitive impairment and additional pregnancy effects were reviewed below, but were not included toward the burden of disease estimate. The lack of current epidemiological evidence suitable to calculate a burden estimate does not necessarily imply the lack of an effect. For example, there is a large body of research into the influence of undernutrition on cognitive function. However, it was felt that there was too much heterogeneity in the types of studies and interventions, exposure variables, and outcome measurements to adequately quantify the undernutrition relationship through meta-analysis. There is also ample evidence describing the role of insufficient weight gain during pregnancy on the risk of IUGR, but prevalence data on pregnancy weight gain in developing countries are scarce.

4.1 Risk of mortality due to childhood infections

It has been demonstrated that a child's risk of dying due to undernutrition is not limited to only those children with the most severe undernutrition (Pelletier et al. 1994). Rather, there exists a spectrum of risk

associated with all degrees of undernutrition—mild, moderate and severe. Although the risk of dying is highest among those most undernourished, when one considers the small but significantly elevated risk of mortality associated with mild and moderate undernutrition and the high prevalence of mild undernutrition worldwide, much of the burden of childhood deaths due to undernutrition may be attributable to mild and moderate, rather than to severe undernutrition.

Pelletier et al. (1994) estimated the relations between weight-for-age and risk of mortality from all causes, and used this information to calculate the burden of child deaths attributable to undernutrition. These relations however are likely to vary depending on specific causes of death. For example, the relative risk of dying from malaria associated with varying degrees of undernutrition may not be similar to the risk for diarrhoea. Despite problems in defining cause of death, particularly in developing countries, in which one may often need to rely on verbal autopsy methods, it is worthwhile to consolidate the evidence to-date and consider the implications of analyses relating variation in anthropometric status and risk of dying across the principal causes of death for children in developing countries: diarrhoea, pneumonia, measles and malaria.

Methods for estimating risk of mortality

Initially, we surveyed the published literature to gather data to estimate the relationship between anthropometric status and cause-specific mortality (Rice et al. 2000). Because insufficient data were available, we contacted investigators with relevant data (published or unpublished) and asked them to contribute specific study results for this analysis. We sought community-based prospective cohort studies in which anthropometric status was assessed to determine weight-for-age, vital status monitored and cause of death determined by documented methods. Data sets ultimately contributing to the mortality analysis are listed in Table 2.3.

Each investigator was asked to provide the following information: (i) a description of their study; (ii) the number of children in the study or child-years of study with WAZ <–3, –2 to –3, –1 to –2, 0 to –1 and >0; and (iii) deaths in each category attributable to diarrhoea, pneumonia, measles, malaria, or other cause and total (all-cause). During analysis, we collapsed the last two categories of anthropometric status, so that the reference group would be composed of those children with WAZ >–1.

Analytic methods used to combine mortality data

We followed the procedures of Pelletier et al. (1993, 1994, 1995) and used the SAS Proc Mixed program for all procedures. Because this has been described elsewhere, we describe the steps only briefly here. First, we calculated the mortality rate (per 1000) by anthropometric status and each cause of death for each study. These rates are listed in Table 2.4

Table 2.3 Data sets with information contributing to the childhood mortality analysis

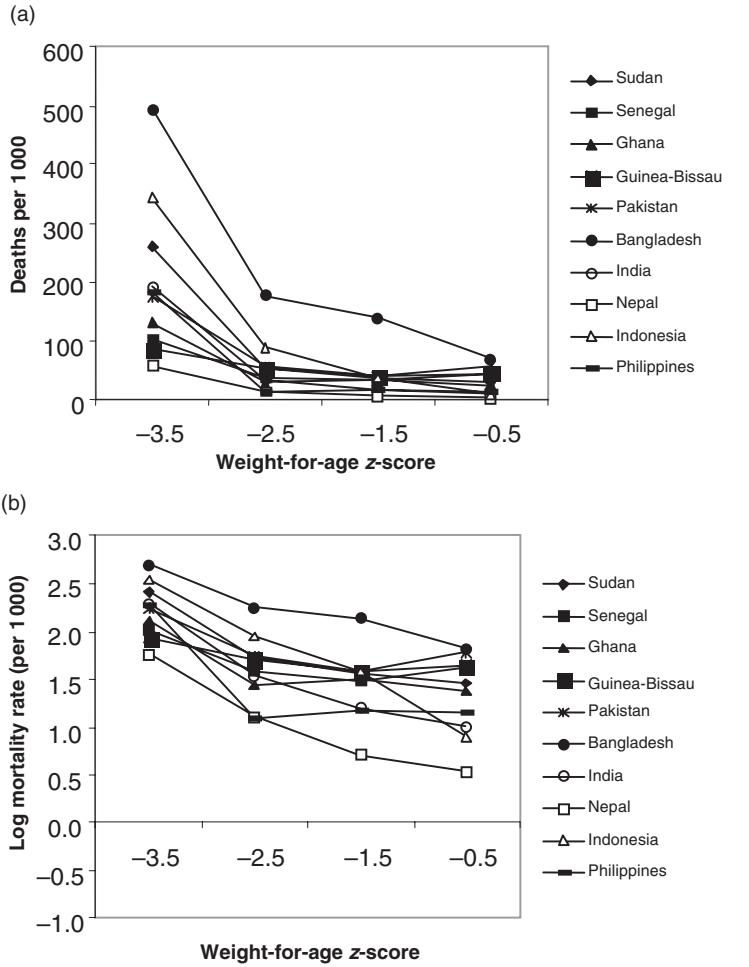
				Number of children			
Country	*Study*	*Age range (months)*	*Follow-up (months)*	*<–3 SDs*	*>–3 to <–2 SDs*	*>–2 to <–1 SD*	*>–1 SD*
Sudan	Fawzi et al. (1997)	6–72	6	365	985	1 123	592
Senegal	Garenne et al. (1987)	0–59	6	1 663	2 186	2 035	1 249
Guinea-Bissau	Andersen (1997)	0–59	6–12	1 155	4 366	7 341	2 731
Ghana	WHO/CHD (1998)	0–12	12	46	183	506	1 410
Nepal	West et al. (1991, 1997)	0–72	12	1 030	2 082	2 002	892
Bangladesh	Arifeen et al. (2001)	0–11	3	79	250	475	478
Pakistan	Khan et al. (1993); Jalil et al. (1993)	0–24	6	309	778	1 182	1 165
India	WHO/CHD (1998)	0–12	12	172	639	1 259	1 295
Indonesia	Sutrisna et al. (1993)	0–60	18	207	240	797	4 856
Philippines	Ricci and Becker (1996)	0–59	3	800	3 144	4 834	5 115

Table 2.4 Mortality rate (000s) by anthropometric status and cause of death

WAZ	Sudan	Senegal	Ghana	Guinea-Bissau	Nepal	Pakistan	Bangladesh	Indonesia	India	Philippines
All-cause mortality										
<–3	260.3	102.2	130.4	86.6	57.3	146.2	493.7	343.0	191.9	182.5
–3 to –2	52.8	37.5	27.3	51.5	12.5	48.0	176.0	87.5	34.4	12.1
–2 to –1	35.6	30.5	31.6	38.7	5.0	34.6	136.8	37.6	15.9	14.9
>–1	28.7	41.6	23.5	44.1	3.4	52.4	66.9	7.8	10.0	14.1
Diarrhoea										
<–3	147.9	42.7	21.7	13.0	21.4	87.0	126.6	29.0	64.0	81.3
–3 to –2	14.2	14.6	5.5	6.0	8.2	16.0	36.0	4.2	23.5	6.4
–2 to –1	11.6	8.8	11.9	2.6	2.0	6.4	18.9	2.5	4.8	6.8
>–1	6.8	15.2	4.2	3.7	1.1	6.1	16.7	1.0	0.8	7.4
Pneumonia										
<–3	32.9	6.6	65.2	2.6	19.4	23.7	151.9	159.4	34.9	87.5
–3 to –2	7.1	4.1	10.9	2.3	4.8	2.9	52.0	37.5	4.7	4.5
–2 to –1	2.7	6.4	4.0	1.6	1.5	7.3	31.6	18.8	1.6	6.6
>–1	1.7	8.8	2.8	1.9	1.1	12.2	18.8	3.1	3.1	5.1
Malaria										
<–3	—	3.6	21.7	33.8	—	—	—	—	—	—
–3 to –2	—	5.0	0.0	19.7	—	—	—	—	—	—
–2 to –1	—	2.5	2.0	15.5	—	—	—	—	—	—
>–1	—	3.2	4.2	16.2	—	—	—	—	—	—
Measles										
<–3	—	12.6	0.0	2.6	3.9	—	—	14.5	—	13.8
–3 to –2	—	2.7	0.0	2.5	1.0	—	—	4.2	—	1.3
–2 to –1	—	3.4	4.0	1.0	1.0	—	—	1.3	—	1.4
>–1	—	2.4	0.0	1.6	0.0	—	—	1.4	—	1.6

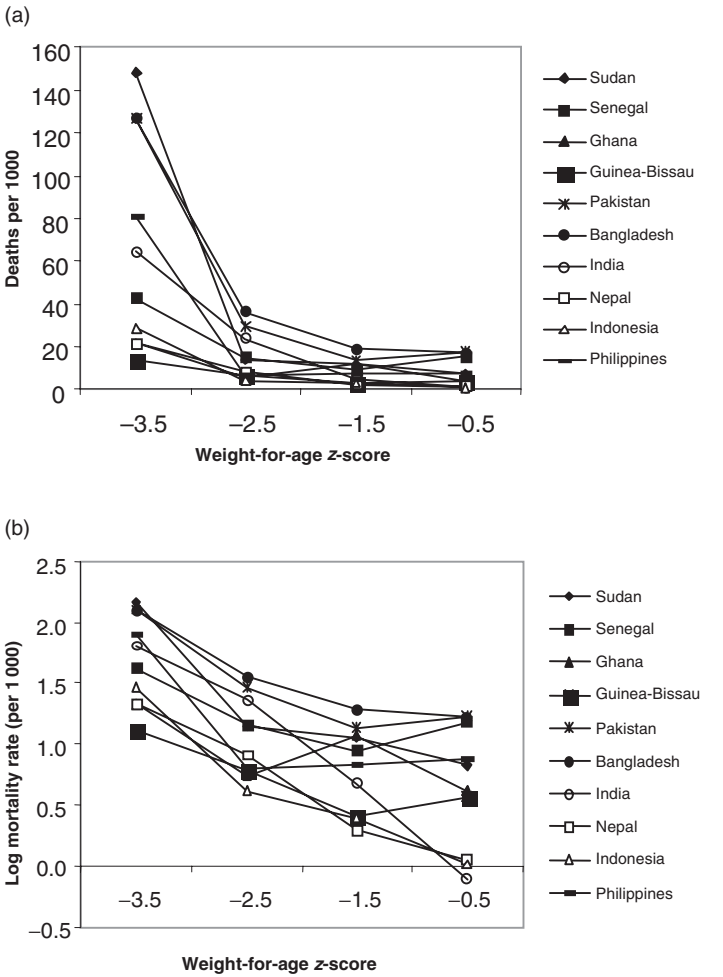
— No data.

Figure 2.1 Underweight and all-cause mortality: (a) deaths per 1000; (b) logarithm of deaths per 1000



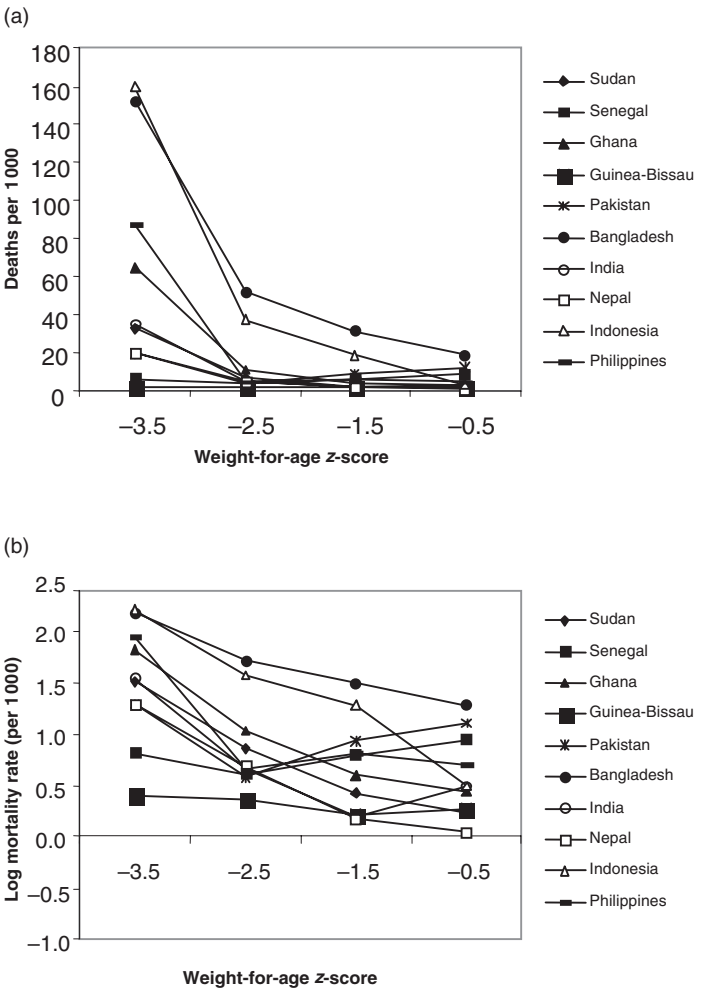
and presented graphically in Figures 2.1(a)–2.5(a). We then calculated the logarithm of the mortality rate by anthropometric status and each cause of death for each study, and re-graphed the data (Figures 2.1[b]–2.5[b]). Second, we regressed the logarithm of mortality on WAZ and compared these results with regressions of the simple mortality rates by WAZ. Third, we compared the goodness-of-fit characteristics for the two models (R^2 and MSE). The models with log mortality rate as the outcome had better goodness-of-fit. For these analyses, we utilized a weighted regression with the weighting scheme of Pelletier and col-

Figure 2.2 Underweight and diarrhoea mortality: (a) deaths per 1000; (b) logarithm of deaths per 1000



leagues: (1/deaths) + (1/children). Fourth, we utilized weighted random effects models (Proc Mixed in SAS—see section 2.2 under subregional estimates), to provide combined estimates of the relation between weight-for-age and risk of mortality. The midpoint of the anthropometric category was used for estimation, and –0.5 SD was considered the value of the reference category. The results of the regression analyses are provided in Table 2.5. From these coefficients, estimated relative risk and 95% CIs were calculated. These steps were performed for all-cause mortality as well as by cause of death.

Figure 2.3 Underweight and pneumonia mortality: (a) deaths per 1000; (b) logarithm of deaths per 1000

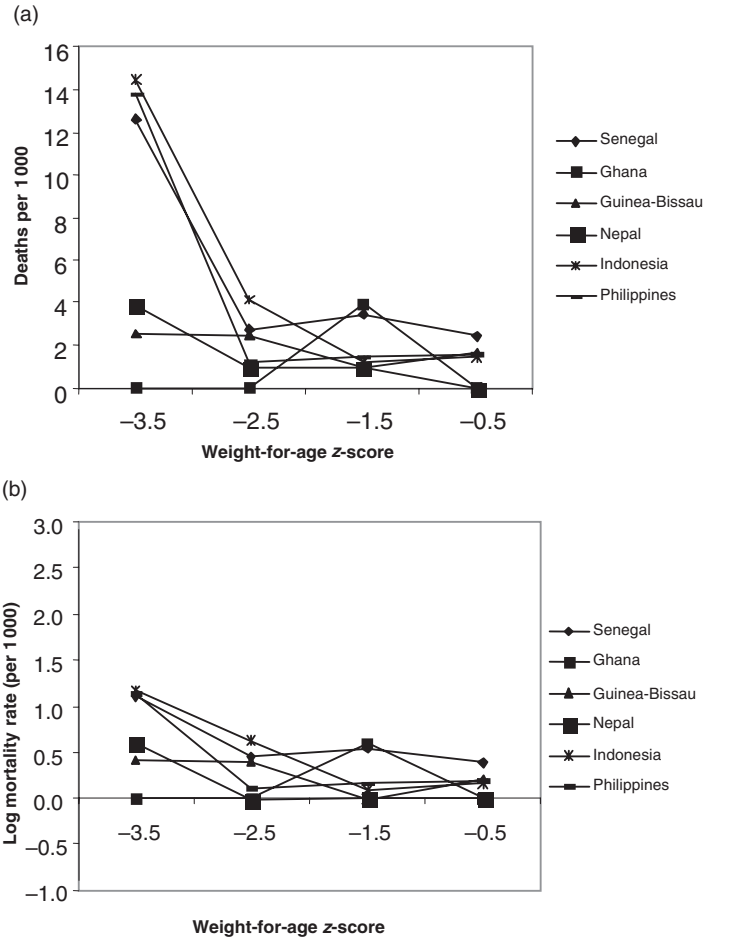


Results

In all, we identified 12 potential studies, and have utilized data from 10 studies in the current analysis. We excluded two studies from the analysis: data from Peru were excluded because the study was too small and provided insufficient deaths for the analysis; data from Brazil were excluded because they resulted from a case–control study and were therefore not appropriate for our analysis.

The 10 studies were conducted in sub-Saharan Africa and Asia (Table 2.3). Each study contributed data on deaths due to diarrhoea and pneu-

Figure 2.4 Underweight and measles mortality: (a) deaths per 1000; (b) logarithm of deaths per 1000



monia (see Figures 2.2 and 2.3); however, exposure to infectious diseases such as malaria and measles depends on the ecology of the study setting and health care utilization (i.e. measles vaccination rates), and thus, not all studies contributed data on these causes of death. In all, six studies from Ghana, Guinea-Bissau, Indonesia, Nepal, the Philippines and Senegal contributed data on deaths due to measles (Figure 2.4). Three studies, from Ghana, Guinea-Bissau and Senegal contributed data on malaria-related deaths (Figure 2.5).

From the regression analyses we calculated the relative risk (95% CI) of death by cause of death for each category of weight-for-age as

Figure 2.5 Underweight and malaria mortality: (a) deaths per 1000; (b) logarithm of deaths per 1000

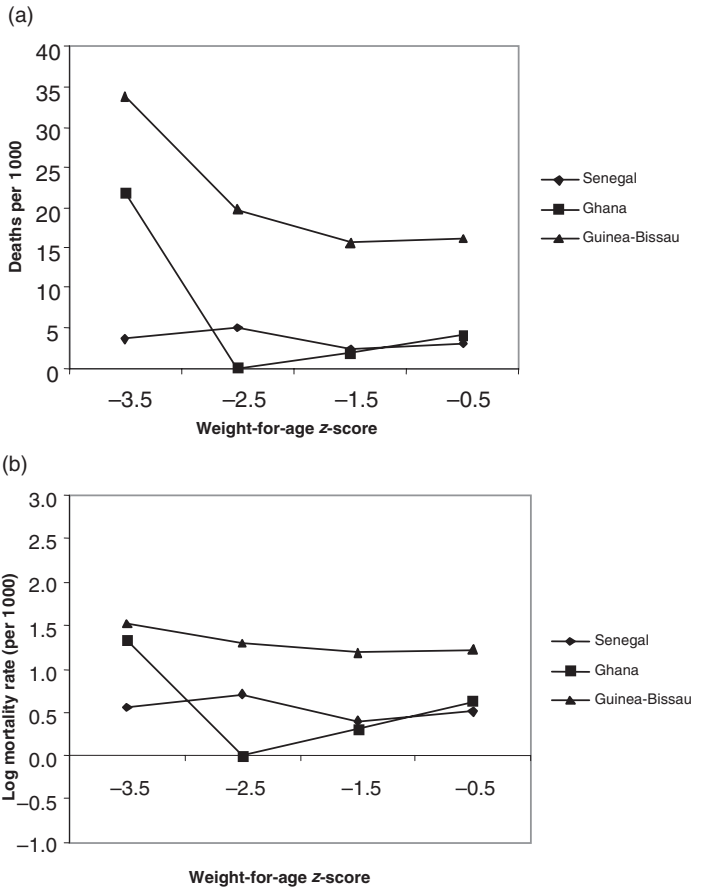


Table 2.5 Regression coefficients for models relating low weight-for-age with mortality, by cause

Outcome	*Intercept (SE)*	*Weight-for-age (SE)*
All-cause mortality	+2.26[a] (0.30)	–0.722[a] (0.077)
Diarrhoea mortality	+0.490 (0.338)	–0.842[a] (0.094)
Pneumonia mortality	+0.459 (0.361)	–0.697[a] (0.105)
Malaria mortality	+0.058 (0.454)	–0.750[a] (0.182)
Measles mortality	–0.263 (0.370)	–0.551[a] (0.140)

[a] $P < 0.05$.

Table 2.6 RR of mortality associated with low weight-for-age estimated from regression analysis, by cause of death[a]

Cause of death	*<–3 SDs (95% CI)*	*–2 to –3 SDs (95% CI)*	*–1 to –2 SDs (95% CI)*	*>–1 SD*
Diarrhoea	12.50 (7.19–21.73)	5.39 (3.73–7.79)	2.32 (1.93–2.79)	1.0
Pneumonia	8.09 (4.36–15.01)	4.03 (2.67–6.08)	2.01 (1.63–2.47)	1.0
Malaria	9.49 (3.25–27.66)	4.48 (2.20–9.15)	2.12 (1.48–3.02)	1.0
Measles	5.22 (2.29–11.88)	3.01 (1.74–5.21)	1.73 (1.32–2.28)	1.0
All-cause	8.72 (5.55–13.72)	4.24 (3.13–5.73)	2.06 (1.77–2.39)	1.0

[a] Calculated at –3.5, –2.5, –1.5 vs 0.5 SD weight-for-age.

compared to the reference category of weight-for-age >–1 SD (Table 2.6). As shown, there are significant risks of death associated with low weight-for-age for overall mortality as well as for each cause of death examined.

Several results on the relationships between anthropometric status and specific causes of mortality should be interpreted with caution. First, studies on the influence of low weight-for-age on measles mortality have yielded equivocal results. For example, a community-based longitudinal study by Chen et al. (1980) in Bangladesh found that underweight children were at over twice the risk for measles mortality (risk ratio 2.37, 95% CI 0.96–5.86), while Aaby et al. (1988) observed little effect attributable to pre-existing nutritional status in Guinea-Bissau (estimated risk ratio 0.83, 95% CI 0.25–2.78). Other factors, such as intra-household crowding and the different levels of exposure experienced by primary and secondary cases, appeared to have greater influence on clinical outcome. Second, in the study by Andersen (1997) in Guinea-Bissau, deaths due to malaria were reported as due to fever, and thus deaths due to febrile illnesses other than malaria were necessarily included. However, as shown in Figure 2.5 (b), the results are quite consistent with those from Senegal. The results from Ghana suggest a different pattern of relationship with mortality risk, but it should be noted that the number of deaths due to malaria is quite small ($n=8$). Further research is needed to refine these estimates. Finally, as described earlier, poor nutritional status is associated with poverty and poor environment. Some of the effects of such factors on child health are mediated through modifying anthropometry and some through other mechanisms. Therefore, it is likely that the hazard estimates presented here are affected by confounding due to a number of such factors.

4.2 Risk of morbidity from childhood infections

The incidence of infection and the risk of other morbidities associated with underweight status were estimated through a literature review and meta-analysis of published data.

Search strategy for identifying studies

Articles relating to undernutrition were identified for review based upon database searches of literature published between 1966 and 2001 in English, or having an English abstract. Searches of Medline, Popline and PsychInfo databases were conducted through PubMed (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi) and Internet Grateful Med (http://igm.nlm.nih.gov, now discontinued) using numerous keywords, author names and "related articles" links. After reviewing the abstracts, English-language articles that examined the relationship between anthropometric status and health outcomes among human populations were selected.

Once copies of articles were obtained, additional publications were identified from the reference lists of those articles. Several nutrition researchers were consulted as to their awareness of any ongoing studies or additional published or unpublished data. However, beyond this and the search strategy described above, there was no systematic attempt to identify unpublished studies or dissertations.

Inclusion/exclusion criteria

The studies considered for review were restricted to: (i) original research reports of community-based or facility-based controlled trials, cohort studies, case–control studies, cross-sectional studies and retrospective analyses of records; (ii) critical review articles that contained original research results not available elsewhere; and (iii) meta-analyses of original research results. Studies that were excluded from the review and analysis were: (i) animal studies; (ii) case reports; (iii) ecological analyses; (iv) studies of special populations (e.g. dialysis patients, acquired immunodeficiency syndrome [AIDS] patients); (v) studies without a control or comparison group; (vi) studies where nutritional status was defined only by birth weight; (vii) studies where the term "malnutrition" was used without further descriptive information or reference to a source providing such information; and (viii) studies where clear risk estimates were not presented or could not be calculated from the reported data.

The analysis was limited to studies conducted among populations in developing countries, among whom poor anthropometric status was considered more likely to reflect undernutrition. Studies of protein/energy supplementation or fortification were excluded from the analysis because the exposure variable (i.e. supplemented vs not supplemented) was not compatible with the anthropometric definition of undernutrition used here. The analysis of studies was limited to those reporting weight-for-age according to either *z*-score or percentage of median relative to the Harvard (Jelliffe 1966) or NCHS/WHO (Hamill 1977; WHO 1978) references.

Analytic methods used to combine individual study risk estimates

Meta-analyses were performed using unadjusted data from studies that satisfied all of the inclusion/exclusion criteria and provided sufficient statistical information (e.g. sample sizes per group, means and SDs or significance test values). Original source data were not obtained for these meta-analyses. Each meta-analysis was based on data exactly as reported in the publication, whenever possible. In some cases, the data had to be adapted from reported rates and statistical information in order to convert them into comparable units of measurement or to conform to comparable nutritional categories. For example, a multiplier would be used to convert diarrhoea episodes per month into diarrhoea episodes per year, or categorical data corresponding to two or more degrees of malnutrition would be collapsed into a single category. Estimates were made of data plotted in figure form if the figure provided sufficient clarity and precision.

All modelling and testing was conducted using Intercooled STATA 6.0. Studies reviewing malaria risk associated with underweight status were combined using Mantel-Haenszel fixed-effect models and tests of heterogeneity. Relative risk of diarrhoea associated with low weight-for-age was calculated from summary data and estimates of lower and upper confidence limits were calculated using the incidence-density binomial approximation to the normal distribution. Individual study estimates were then combined using Mantel-Haenszel combination techniques and tests of heterogeneity. Random effects models were used to combine individual study estimates for risk of pneumonia associated with low weight-for-age. For each outcome, sensitivity analyses were conducted to determine if any studies were significantly driving the estimate.

Although *z*-score is now preferable, most studies reported anthropometric status in terms of percentage of median, and cut-off points to classify malnutrition varied among different studies. As a result, the definition of underweight was not absolutely consistent among the studies combined in each summary estimate. The differences in definition in such cases were distinguished in this review through the use of multiple analysis "levels", wherein studies were separated by their nutritional categorization scheme during the analysis process in order to compare and contrast the results. Analysis levels are described further in the sections on pneumonia and diarrhoea results.

Weight-for-age was analysed according to multiple categories of severity, when possible, in order to describe any potential dose–response effect and better characterize possible health risks among the large proportion of mild-to-moderate malnutrition cases. The ability to examine multiple weight-for-age levels, however, was highly limited by the available published data; the majority of studies analysed anthropometric status as a dichotomous variable only. Therefore, for all health outcomes, underweight was analysed as a dichotomous variable, comparing individuals

falling below the designated weight-for-age cut-off point to those above the cut-off point (the reference group). A drawback to the dichotomous approach is that if a dose–response trend exists, combining intermediate-risk and low-risk groups into a single category for a dichotomous analysis could have the effect of underestimating the true risk. An additional analysis was conducted when data on multiple weight-for-age categories were available, in which case data were conflated into three anthropometric categories: moderately to severely underweight (e.g. weight-for-age below –2 SDs), mildly underweight (e.g. weight-for-age between –1 and –2 SDs) and normal (e.g. weight-for-age greater than –1 SD). Risk ratios were then estimated for both categories in relation to the reference category of weight-for-age >–1 SD. When weight-for-age was defined by percentage of median, the overall analysis was subdivided into separate analysis levels according to the cut-off points used.

Measles results

There were an inadequate number of eligible studies for a summary risk analysis of weight-for-age and measles incidence. Evidence from an observational study in the Gambia (Lamb 1988) and a controlled supplementation trial in India (Gopalan et al. 1973) suggest, however, that pre-morbid underweight status has no significant effect on the incidence of measles infection. In India, Sinha et al. (1977) observed a reduced incidence of measles rash among children in the lowest weight percentiles, but this may represent a lowered immune response rather than a reduced incidence of infection. Beau et al. (1987) in Senegal noted a higher prevalence of measles among hospitalized children who were wasted at admission, but reverse causality would explain this observation.

Malaria results

Research and clinical observation over the past several decades have generated debate as to how undernutrition may influence susceptibility to malaria. Conflicting results occur, in part, because susceptibility to malaria attack and/or infection is highly dependent on the individual's immune status, which, in turn, is influenced by factors such as age, pregnancy status and the level of endemicity within a population (Shankar 2000). The relationship between malaria and undernutrition has been examined according to several outcome variables, including presence or density of malaria parasites in the blood, episodes of malarial illness, incidence of complicated malaria (cerebral malaria or severe malarial anaemia) among malaria cases and case-fatality. This comparative risk analysis focused only on the incidence of malaria attacks, defined clinically as episodes of fever with slide-confirmation of parasites in the blood, or, in some studies, less specifically as fever with chills. Most of the data related to infection by *Plasmodium falciparum*, although some studies included data on *Plasmodium vivax*, as well.

Table 2.7 Risk of malaria attack associated with low weight-for-age from five studies

Location	*Study*	*Study type*	*Cut-off point*	*Sample size*	*Risk estimate (95% CI)*
Congo	Tonglet et al. (1999)	Community-based cohort	WA <25th percentile	842	RR = 0.92 (0.72–1.17)
Gambia	Snow et al. (1991)	Community-based cohort	WA <70%	138	RR = 1.52 (0.59–3.93)
Nigeria	Adelekan et al. (1997)	Facility-based case–control	WAZ <–2	65	OR = 1.50 (0.51–4.41)
Sudan	el Samani et al. (1987)	Community-based cross-sectional	WA <75%	445	RR = 1.63 (1.18–2.27)
Vanuatu	Williams et al. (1997)	Community-based cohort	WAZ <–2	911	RR = 1.28 (0.87–1.89)

WA Weight-for-age.

It is difficult to adequately estimate any effect of low weight-for-age on risk of malaria morbidity given the scarcity of studies reporting both weight-for-age and malaria data. Five studies were identified that fulfilled that initial requirement (Table 2.7). These studies examined nutritional status as a dichotomous variable, comparing underweight children (defined as WAZ <–2, weight-for-age <70%, or weight-for-age <75% of the NCHS reference median) to adequate-weight children; there were insufficient data from eligible prospective studies to examine any potential risk associated with milder degrees of malnutrition (i.e. WAZ between –1 and –2). Of the five studies, cohort studies from the Gambia (Snow et al. 1991), the Congo (Tonglet et al. 1999) and Vanuatu (Williams et al. 1997) among children aged 1–4 years, <2 years and <10 years, respectively, reported statistically non-significant differences in malaria morbidity between underweight and normal children. In a facility-based case–control study in Nigeria (Adelekan et al. 1997) malaria cases in children aged <8 years had slightly, albeit not significantly, lower weight-for-age than non-infected controls, but this study was dropped from the final analysis because nutritional status was assessed after the onset of illness, leaving causality ambiguous. A cross-sectional study in the Sudan (el Samani et al. 1987) observed significantly greater history of malaria attacks (defined as fever with rigors and joint pain) among underweight children aged <5 years, but the causality was unclear in this case, as well.

Of the remaining three prospective studies, the Congo study (Tonglet et al. 1999), which reported a relative risk of 0.92 (95% CI 0.72–1.17) for a malaria episode, was dropped from the summary analysis because nutritional categories were defined according to local reference data. Following meta-analysis of the two remaining cohort studies, the most

Table 2.8 Combined estimate of risk for malaria attack associated with low weight-for-age from two cohort studies

Location	*Study*	*Risk estimate (95% CI)*
Gambia	Snow et al. (1991)	1.52 (0.59–3.93)
Vanuatu	Williams et al. (1997)	1.28 (0.87–1.89)
Combined estimate		1.31 (0.92–1.88)

appropriate for this assessment, the combined relative risk for malaria among children having weight-for-age <–2 SDs was statistically non-significant (RR 1.31, 95% CI 0.92–1.88) (Table 2.8). This overall estimate was more heavily influenced by the Vanuatu cohort study, which reported a slightly but not significantly higher incidence of *P. falciparum* malaria (defined as fever with parasitaemia >1000/µl) among children who were classified as underweight prior to the period of morbidity follow-up.

Pneumonia/ALRI results

Undernutrition may increase the risk of pneumonia through a number of potential mechanisms. In general, cell-mediated immunity is depressed in undernourished children (Rivera and Martorell 1988; Scrimshaw and SanGiovanni 1997). Respiratory muscles weaken, impairing the cough reflex and reducing a child's ability to adequately clear secretions from the respiratory tract or respond to hypoxia through tachypnea (Wilson 1985). Salivary IgA is decreased, compromising the integrity of the respiratory tract mucosa and leaving the mucosa susceptible to invasion by microorganisms (Lehmann et al. 1988).

Over 60 studies evaluating the relationship between acute respiratory infections and malnutrition were initially identified. These included a collaborative data group from the United States National Academy of Sciences Research Program of the Board on Science and Technology for International Development (BOSTID). We immediately excluded studies that examined upper respiratory tract infections only; did not distinguish between upper and lower respiratory tract infections in their data collection and/or analysis; limited the report to single etiological agents (e.g. only pneumococcus); or did not report anthropometric status in terms of weight-for-age. The remaining studies are briefly described below, although not all fulfilled the criteria for inclusion in a summary analysis, usually for reasons of insufficient data or statistical information. Studies that provided estimates of risk are listed in Tables 2.9 and 2.10.

Studies varied in their case definitions of ALRI and pneumonia, classifying them on the basis of mothers' recall of the physician's diagnosis (Victora et al. 1990); radiologically confirmed pneumonia (Alam et al. 1984; Fonseca et al. 1996; Victora et al. 1994); or cough and fever with

Table 2.9 Risk of pneumonia/ALRI incidence associated with low weight-for-age from studies included in summary estimate

Analysis level	*Study*	*Location*	*Cut-off point*	*Sample size*	*Crude RR (95% CI)*	*Adjusted RR*[a] *(95% CI)*
1	Victora et al. (1990)	Brazil	WAZ <–2	4 486	1.32 (0.70–2.47)	1.75[b]
1	Zaman et al. (1996)	Bangladesh	WAZ <–2	696	1.25 (0.76–2.05)	—
3	James (1972)	Costa Rica	WA <75%	137	12.37 (3.11–49.25)	—
4	Ballard and Neumann (1995)	Kenya	WA <80%	109	0.90 (0.37–2.17)	1.8[c] (0.52–6.4)
4	Deb (1998)	India	Unclear	800	2.53 (1.72–3.73)	—

— No data.

[a] All crude relative risks reported in Table 2.9 represent risk associated with weight-for-age below the cut-off point compared to risk associated with weight-for-age above the cut-off point. Adjusted relative risks, however, varied in their referent group and in the variables adjusted as indicated in the table footnotes.

[b] Adjusted for income. Adjusted risk ratio was based on WAZ <–2 relative to WAZ ≥0. Crude risk estimate of 1.32 was based on WAZ <–2 relative to WAZ >–2. (Crude risk estimate based on WAZ <–2 relative to WAZ ≥0 is 2.38 [95% CI 1.18–4.78].)

[c] Adjusted for season, socioeconomic status and geographic region. Adjusted RR was based on weight-for-age <80% relative to weight-for-age >90%; crude RR was based on weight-for-age <80% relative to weight-for-age >80%. (Crude RR based on weight-for-age <80% relative to weight-for-age >90% is 2.03 [95% CI 0.44–9.25].)

rapid respiratory rate (Cunha 2000; Deb 1998; Smith et al. 1991) and/or chest indrawing (Ballard and Neumann 1995; Spooner et al. 1989; Zaman et al. 1996). The case definition for pneumonia recommended by WHO is cough or difficult breathing with rapid respiration (>50 breaths/min for infants aged 2 months to under 1 year; >40 breaths/min for children aged 1 to 5 years) or chest indrawing, stridor or general danger signs such as vomiting, convulsions, lethargy, unconsciousness or inability to drink/breastfeed (WHO 1999b). The coordinated BOSTID data group defined ALRI as the presence of at least one of the following: rales or crepitations, wheezing, stridor, cyanosis, rapid respiratory rate (>50 breaths/min), or chest indrawing (Selwyn 1990). Studies were considered for our summary analysis if the case definition for ALRI was consistent with the recommended WHO definition. With the exception of a Bangladesh study among diarrhoea patients aged <12 years (Alam et al. 1984), all of the studies examined children aged ≤5 years. Incidence was reported predominantly as the number of ALRI episodes over a given follow-up period (i.e. multiple episodes per individual child counted as separate events), although a few studies reported the proportion of children with one or more ALRI episodes (i.e. multiple episodes per individual child counted as a single event).

Table 2.10 Risk of pneumonia/ALRI incidence associated with low weight-for-age from excluded studies

Location	*Study*	*Study type*	*Cut-off point*	*Crude risk estimate (95% CI)*	*Adjusted risk estimate[a] (95% CI)*	*Reason for exclusion*
Brazil	Cunha et al. (2000)	Community-based cohort	WAZ –3 to –2	—	OR = 1.59[b] (1.09–2.33)	Insufficient incidence data
Brazil	Fonseca et al. (1996)	Community-based cohort	WAZ <–2	—	OR = 4.57[c] (2.93–7.13)	Reverse causality
Brazil	Victora et al. (1994)	Facility-based case–control	WAZ <–2	OR = 5.87 (3.30–10.44)	OR = 4.77[d] (2.46–9.06)	Reverse causality
China	Liu et al. (1991)	Community-based case–control	Not reported	OR = 5.79 (3.70–9.14)	—	Reverse causality; no PEM definition
India	Shah et al. (1994)	Facility-based case–control	WA <75%	OR = 2.36 (1.53–3.65)	—	Reverse causality
Papua New Guinea	Binns (1976)	Community-based cohort	WA <80%	RR = 1.59	—	No categorical sample size data
Papua New Guinea	Smith et al. (1991)	Community-based cohort	WAZ <–2	—	RR = 2.1[e] (1.3–3.4)	Data reported in figure form
South Africa	Wesley and Loening (1996)	Community-based case–control	WA <10th percentile	OR = 1.47 (0.38–5.88)	—	Reverse causality; cut-off point

— No data.

PEM Protein–energy malnutrition.

[a] All crude relative risks reported in Table 2.10 represent risk associated with weight-for-age below the cut-off point compared to risk associated with weight-for-age above the cut-off point. Adjusted relative risks, however, varied in their referent group and in the variables adjusted as indicated in the table footnotes.

[b] Adjusted for age, sex, household air pollution and crowding. Adjusted OR is based on WAZ between –2 and –3 relative to WAZ >–1.

[c] Adjusted for income, parents' education and previous episode of pneumonia. Adjusted OR is based on WAZ <–2 relative to WAZ ≥0.

[d] Adjusted for age, sex, father's education, household crowding and other factors. Crude and adjusted ORs are based on WAZ <–2 relative to WAZ >–1.

[e] Adjusted for age. Adjusted risk ratio is based on WAZ <–2 relative to WAZ ≥0.

Excluded studies

The collaborative BOSTID study reported community-based longitudinal data from Guatemala, Papua New Guinea, the Philippines and Uruguay (Selwyn 1990). In each population, researchers observed higher incidence of ALRI among underweight children aged 18–59 months, with relative risks ranging between 1.2 (Guatemala) and 2.7 (Uruguay). An increased risk was observed among the younger group of 0–17-month-old children only in the Philippines (RR = 1.3). While the methodology used in these four countries was standardized for better internal comparability among study sites, the results were not included in our summary analysis because nutritional status was evaluated using an anthropometric cut-off point (10th percentile) higher than WAZ of –2 (equivalent to approximately the third percentile—Pelletier 1994) and the case definitions used in Guatemala and Uruguay could have classified asthma as ALRI.

Ten ALRI investigations were case–control studies or record reviews and were excluded because of the possibility of reverse-causality. In Colombia (Berman et al. 1983) and India (Shah et al. 1994), children with pneumonia were significantly more likely to be moderately or severely malnourished than were non-ALRI paediatric patients or outpatients. In South Africa, in contrast, odds of being malnourished were not significantly different between pneumonia patients and a control group of upper respiratory infection patients (Wesley and Loening 1996). In a case history review in Bangladesh, malnourished diarrhoea patients aged 1–5 years had a higher incidence of pneumonia during hospitalization than diarrhoea patients considered well-nourished, although the relationship was not observed in infants (Alam et al. 1984).

Case–control studies in Brazil, China and Papua New Guinea (Fonseca et al. 1996; Victora et al. 1994) compared cases to healthy controls from the surrounding community. Children having lower respiratory infections in China (Liu et al. 1991) and Papua New Guinea (Spooner et al. 1989) were at significantly greater odds of being underweight compared to healthy controls, and underweight children in Papua New Guinea were four times as likely to be admitted to the hospital with pneumonia as non-malnourished children (Barker et al. as cited in Lehmann et al. 1988). Similarly, in an urban area of southern Brazil, children aged <2 years hospitalized with pneumonia had a reported OR of 5.87 (95% CI 3.30–10.44) for weight-for-age <–2 SDs compared to healthy, age-matched neighbourhood controls; adjusted for age, sex, crowding and socioeconomic factors, the odds ratio decreased slightly to 4.77 (95% CI 2.46–9.06) (Victora et al. 1994). Fonseca et al. (1996) conducted a similarly designed study in northern Brazil and found undernutrition to be the most important risk factor for pneumonia among children aged <2 years attending an outpatient clinic; the odds ratio for weight-for-age <–2 SDs among cases was 4.57 (95% CI 2.93–7.13) adjusted for income, parents' education and previous pneumonia.

In a community-based survey in Brazil, Cunha et al. (2000) compared anthropometric status of children aged <5 years according to mothers' report of respiratory illness in the previous week (Cunha et al. 2000). Adjusted for age, sex and household crowding, children with weight-for-age below –2 SDs had significantly greater odds for an ALRI episode compared to children with weight-for-age >–1 SD.

Prospective cohort studies that assessed anthropometric status prior to illness were more appropriate for evaluating malnutrition as a causal factor in ALRI incidence. In rural Papua New Guinea, Smith et al. (1991) assessed nutritional status and subsequent incidence of ALRI through twice-weekly home visits, reporting an age-adjusted incidence rate ratio of approximately 2.1 (95% CI 1.25–3.4) associated with WAZ <–2 relative to weight-for-age >0 SD. There was no evidence of confounding by socioeconomic status (defined by education, income and household crowding) or previous episodes of illness. The data, however, were reported only in figure form and lacked sufficient statistical information for inclusion in the meta-analysis. An earlier cohort study in Papua New Guinea by Binns (1976) observed a relative risk of 1.59 for pneumonia among children having weight-for-age <80%, but, likewise, did not report sufficient statistical data for inclusion (such as sample sizes per weight-for-age category).

Studies included in the risk estimate

James (1972) in urban Costa Rica reported the highest risk estimate among cohort studies. Based on weekly physician visits and mothers' recall, underweight children aged <5 years had a similar number of overall respiratory tract infections as normal-weight children, but experienced a relative risk of 12.4 (95% CI 3.11–49.25) for bronchopneumonia, with no apparent differences between groups in breastfeeding duration, age distribution, levels of household crowding, incomes or sanitation. In urban Brazil, Victora et al. (1990) followed a cohort of infants for subsequent hospital admissions, reporting a significant relative risk (adjusted for family income) of 1.75 for pneumonia among children with pre-morbid WAZ <–2 compared to ≥0. The risk estimate remained statistically significant after adjusting for previous pneumonia hospitalizations (data not reported). Incidence referred to hospital admissions, which represented only the more severe cases of ALRI, and there is potential bias if underweight ALRI cases are more likely to be admitted than the normal weight cases. In a community-based cohort study in Bangladesh, Zaman et al. (1996) followed up morbidity every four days and observed an increased, but not statistically significant, risk of 1.25 between low weight-for-age and subsequent incidence of ALRI among children aged ≤5 years; adjusted for age, sex and socio-demographic variables, the relative risk became statistically significant, with a one-unit decrease in WAZ being associated with a 55% increase in the incidence of ALRI. A smaller cohort study in Kenya reported a slightly higher,

but statistically non-significant, relative risk for ALRI of 1.8 (95% CI 0.52–6.4) among children aged 18–25 months with weight-for-age <80% relative to weight-for-age >90%, adjusted for season, socioeconomic status and geographic region (Ballard and Neumann 1995). In an urban and rural community-based cohort study in India (Deb 1998), underweight children aged <5 years experienced a significantly higher rate of pneumonia over the course of 18 months relative to normal-weight children (RR=2.53), although it was not reported how comparable malnourished and normal groups were in relation to potentially confounding variables.

Relative risk estimate

There were an insufficient number of studies reporting multiple categories of weight to compare the risk of ALRI associated with mild-to-moderate degrees of underweight, and, therefore, only a dichotomous analysis was performed. Two cohort studies reported anthropometric status in terms of *z*-score and were treated as "analysis level one" (Table 2.11). Analysis levels three and four used cut-off points in terms of percentage of median. (Analysis level two was reserved for studies using a cut-off point of 70%, but no eligible studies using that cut-off point were included here.)

Relative risks according to cut-off point are represented graphically in Figure 2.6 and summary risk estimates are listed in Table 2.12 according to analysis level. Overall, community-based cohort studies that

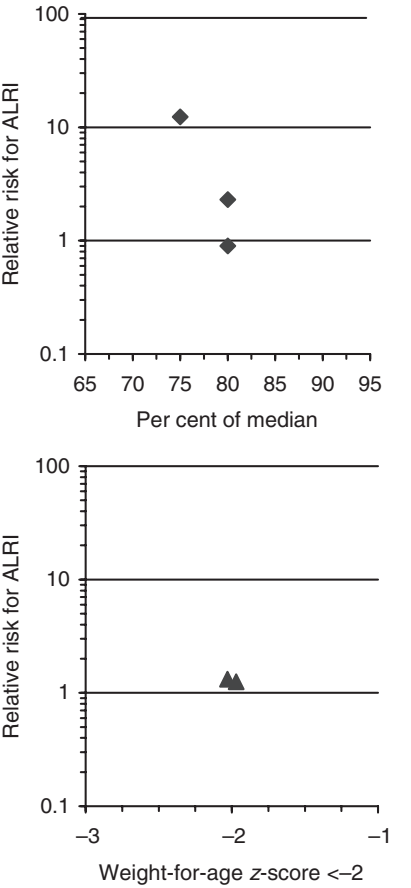
Table 2.11 Analysis levels according to anthropometric classification used

Analysis level	*Comparison*
1	WAZ <–2 vs WAZ >–2
2	WA <70% vs WA >70%
3	WA <75% vs WA >75%
4	WA <80% vs WA >80%

Table 2.12 Combined estimate of risk for pneumonia/ALRI incidence associated with low weight-for-age

Study type	*Analysis level*	*Number of studies*	*Combined risk estimate (95% CI)*
Cohort	1	2	1.28 (0.86–1.89)
Cohort	3	1	12.37 (2.98–51.31)
Cohort	4	2	2.22 (1.55–3.18)
Cohort	1+4	4	1.72 (1.32–2.25)
Combined estimate	1+3+4	5	1.86 (1.06–3.28)

Figure 2.6 Relative risk of pneumonia/ALRI according to weight-for-age cut-off point used in study



provided sufficient results and statistical data yielded a combined risk estimate for ALRI of 1.86 associated with low weight-for-age (Table 2.12). Even excluding the high estimate observed in the study by James (1972), the risk (RR=1.72) remained statistically significant.

Diarrhoea results

There is a large body of literature examining the relationship between malnutrition and diarrhoea, and cross-sectional studies among children have consistently shown a trend of increasing diarrhoea prevalence associated with decreases in anthropometric status. Malnutrition and concomitant growth faltering and weight decline are not uncommon following severe bouts of diarrhoea among children in developing coun-

tries, but the reverse relationship—that pre-morbid undernutrition increases susceptibility to subsequent diarrhoea attack—is more uncertain. We selected prospective studies that specifically examined pre-morbid anthropometric status as a risk factor for subsequent diarrhoea, rather than the reverse relationship. Studies that simultaneously assessed weight-for-age and diarrhoea were not considered because the direction of the relationship could not be determined—i.e. there was potential reverse causality with regard to the risk relationship. All of the studies related to children aged ≤5 years; little if any data were available for older age groups. In some studies, morbidity was compared to the initial assessment of nutritional status only, whereas other studies reclassified children, as necessary, according to periodic follow-up measurements. An episode of diarrhoea was typically defined as three or more loose, liquid or watery stools, or at least one bloody stool in a 24-hour period. An episode was generally considered terminated when normal stool patterns returned for at least three days. Some of the outcome variables relating to diarrhoea included incidence of general diarrhoea, incidence of dysentery and incidence of persistent diarrhoea, defined as diarrhoea lasting 14 days or longer. We restricted our analysis to the incidence of general diarrhoea, and excluded studies that focused solely on a single pathogenic agent (e.g. *Cryptosporidium, Vibrio cholerae*), watery diarrhoea alone or bloody diarrhoea alone.

Literature supports an increased risk of diarrhoea mortality associated with low weight-for-age (Bhan et al. 1986; Chen et al. 1980; Fawzi et al. 1997; Rice et al. 2000; Yoon et al. 1997), but the effect on diarrhoea incidence has been less clear. Cohort studies in Bangladesh (Baqui et al. 1993; Chowdhury et al. 1990), Brazil (Schorling et al. 1990), Costa Rica (James 1972), the Congo (Tonglet et al. 1999), Ethiopia (Lindtjorn et al. 1993), the Gambia (Tomkins et al. 1989), Ghana (Biritwum et al. 1986), Guatemala (Gordon et al. 1964), India (Ghai and Jaiswal 1970; Walia et al. 1989), Mexico (Sepulveda et al. 1988), Papua New Guinea (Binns 1976) and the Sudan (el Samani et al. 1988; Kossman et al. 2000), reported higher diarrhoea incidence rates among underweight children (rate ratios of 1.1 to 2.4), while other studies observed no statistically significant effect (Anand et al. 1994; Bairagi et al. 1987; Bhan et al. 1986; Black et al. 1984; Chen et al. 1981; Henry et al. 1987; Khan and Yunus 1990; Mathur et al. 1985; Molbak et al. 1997b; Thongkrajai et al. 1990; Tomkins 1981; Victora et al. 1990). Factors such as age, socioeconomic status, season, breastfeeding and recent history of diarrhoea, among others, can be important confounders in the relationship between anthropometric status and diarrhoeal illness. When statistical adjustment had been made for such variables, several studies (e.g. el Samani et al. 1988; Kossman et al. 2000; Schorling et al. 1990; Sepulveda et al. 1988; Tonglet et al. 1999; Wierzba et al. 2001) continued to show a significant relationship, while others (Chowdhury et al. 1990; Lindtjorn et al. 1993) did not.

Table 2.13 Diarrhoea cohort studies included in dichotomous analysis

Analysis level	*Study*	*Location*	*Cut-off point*	*Sample size*	*Crude RR (95% CI)*	*Adjusted RR*[a] *(95% CI)*
1	Wierzba et al. (2001)	Egypt	WAZ <–2	143	1.83 (1.36–2.47)	1.7 (1.2–2.3)[b]
1	Baqui et al. (1993)	Bangladesh	WAZ <–2	512	1.11 (1.00–1.23)	1.22 (1.09–1.35)[c]
1	Schorling et al. (1990)	Brazil	WAZ <–2	61	1.18 (1.06–1.34)	OR = 3.4 (1.0–11.9)[d]
1	Victora et al. (1990)	Brazil	WAZ <–2	4 486	0.78 (–0.26–1.95)	0.58[e]
2	Bhan et al. (1986)	India	WA <70%	1 467	0.90 (0.80–1.01)	—
2	Walia et al. (1989)	India	WA <70%	838	1.33 (1.07–1.62)	—
2	Anand et al. (1994)	India	WA <70%	250	1.08 (0.92–1.26)	—
2	Sepulveda et al. (1988)	Mexico	WA <70%	284	1.86 (1.54–2.21)	1.7[f]
3	Mathur et al. (1985)	India	WA <75%	687	1.40 (1.24–1.58)	—
3	Chen et al. (1981)	Bangladesh	WA <75%	207	1.04 (0.91–1.20)	—
3	Black et al. (1984)	Bangladesh	WA <75%	125	1.10 (0.88–1.33)	—
3	Bairagi et al. (1987)	Bangladesh	WA <75%	1 454	1.10 (0.96–1.26)	—
3	Henry et al. (1987)	Bangladesh	WA <75%	300	0.93 (0.82–1.06)	—
3	James (1972)	Costa Rica	WA <75%	137	1.08 (0.93–1.26)	—
3	Gordon et al. (1964)	Guatemala	WA <75%	179	1.73 (1.45–2.07)	—
3	Tomkins et al. (1981)	Nigeria	WA <75%	343	1.22 (1.00–1.45)	—
3	el Samani et al. (1988)	Sudan	WA <75%	403	1.22 (1.00–1.42)	OR = 1.3 (1.0–1.7)[g]
4	Khan and Yunus (1990)	India	WA <80%	183	1.39 (0.75–2.58)	—
4	Biritwum et al. (1986)	Ghana	WA <80%	250	1.53 (1.1– 2.01)	—
5	Chowdhury et al. (1990)	Bangladesh	WA <85%	753	1.54 (0.87–2.72)	—

— No data.

[a] All crude risk estimates in Table 2.13 represent incidence below cut-off relative to incidence above cut-off. Adjusted risk estimates, however, vary in their referent category and adjusted variables as shown in the following table footnotes.

[b] Estimate adjusted for age, sex, socioeconomic status, breastfeeding and previous morbidity. WAZ <–2 vs WAZ >–2.

[c] Estimate adjusted for age. WAZ <–2 vs WAZ >–2. When stratified by previous diarrhoea experience, age-adjusted rate ratios were 1.00 (0.75–1.33) and 1.26 (1.07–1.49) for underweight children without recent morbidity and with recent morbidity, respectively.

[d] Estimate adjusted for previous diarrhoea morbidity, age, sex and household crowding. Estimate is an OR comparing WAZ <–3 to WAZ >–3.

[e] Estimate adjusted for income. WAZ <–2 vs WAZ ≥0.

[f] Estimate adjusted for age. Weight-for-age <75% vs weight-for-age ≥90%.

[g] Estimate adjusted for age, sex, season, socioeconomic status and previous morbidity. Weight-for-age <90% vs weight-for-age ≥90%.

Table 2.14 Analysis levels for dichotomous analysis according to anthropometric definition used in study

Analysis level	*Comparison*
1	WAZ <–2 vs WAZ >–2
2	WA <70% vs WA >70%
3	WA <75% vs WA >75%
4	WA <80% vs WA >80%
5	WA <85% vs WA >85%

Table 2.13 summarizes prospective studies used to estimate diarrhoea incidence according to weight-for-age. The majority of diarrhoea studies reported anthropometric status in terms of percentage of median rather than *z*-score and the cut-off points varied by study. In India, for example, most studies conformed to the Indian Academy of Pediatrics classification in which weight-for-age >80% represents normal status and weight-for-age <70% represents second-degree, third-degree or fourth-degree malnutrition (Indian Academy of Pediatrics 1972). Many others used the Gomez classification, defining second-degree malnutrition and worse as weight-for-age <75% (Gomez et al. 1956). For comparison, studies were grouped into different analysis levels according to the cut-off points used to classify malnutrition (Table 2.14).

Dichotomous estimate

In developing a summary risk estimate, nutritional status was first examined as a dichotomous variable comparing all children below the cut-off point ("malnourished") to children above that point (reference group). The dichotomous approach allowed for the inclusion of the greatest number of studies, since many studies did not further subdivide the malnourished group into discrete anthropometric categories.

Excluded studies

Ten studies that were identified were excluded from the risk analysis (Table 2.15). Seven prospective studies were excluded due to insufficient statistical data (e.g. sample sizes per anthropometric category), data presented as a figure only, or the use of a different growth reference. Six of the seven reported an increased incidence of diarrhoea episodes associated with low weight-for-age, including studies in the Sudan (Kossman et al. 2000) and the Congo (Tonglet et al. 1999) that adjusted for previous diarrhoea morbidity. An additional three cross-sectional studies in Ecuador (Brussow et al. 1993), India (Luwang and Datta 1982) and El Salvador (Stetler et al. 1981) reported risk estimates on the order of 1.45 to 1.57, but reverse causality disqualified the studies.

Studies included in the risk estimate

Figure 2.7 illustrates the distribution of individual study risk estimates according to the cut-off point used. Twenty cohort studies provided sufficient results and statistical data for inclusion in a summary estimate (Table 2.13). The evidence regarding risk of diarrhoea was equivocal

Figure 2.7 Relative risk for diarrhoea according to weight-for-age cut-off point used in study

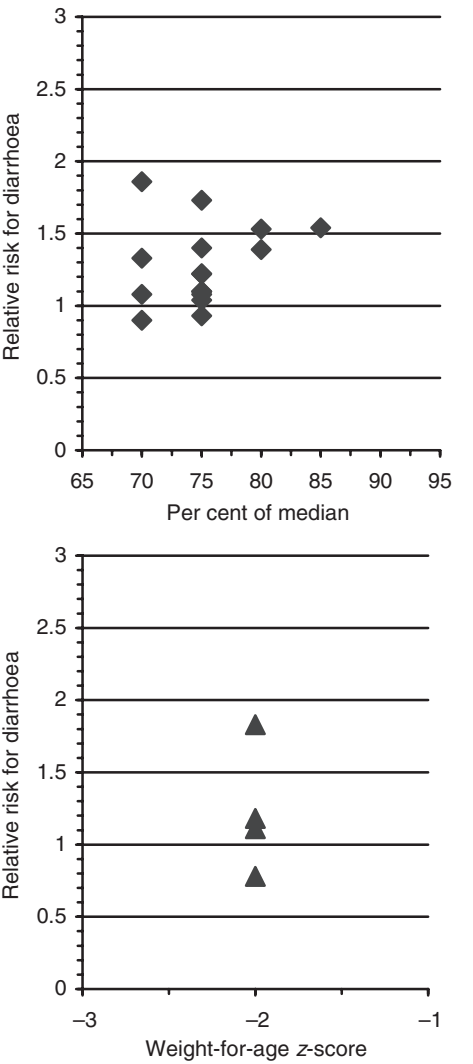


Table 2.15 Diarrhoea studies excluded from dichotomous analysis

Location	*Study*	*Cut-off point*	*Sample size*	*Crude RR (95% CI)*[a]	*Adjusted RR (95% CI)*	*Cause for exclusion*
Congo	Tonglet et al. (1999)	WA <25th percentile	842	1.22[b] (1.02–1.46)	OR= 1.49 (0.94–2.05); 1.50 (1.10–1.89)	Local growth reference
Ecuador	Brussow et al. (1993)	WAZ <–2	321	1.45 (0.99– 2.12)	—	Cross-sectional; reverse causality
El Salvador	Stetler et al. (1981)	WA <75%	3 705	1.46 (1.24–1.71)	—	Cross-sectional; reverse causality
Ethiopia	Lindtjorn et al. (1993)	WAZ <–2	425	2.2[c]	—	Data reported in figure form; no categorical sample size data
Gambia	Tomkins et al. (1989)	WAZ <–2	211	1.18; 1.33[d]	—	Data reported in figure form; no categorical sample size data
India	Ghai and Jaiswal (1970)	WA <85%	925	2.35 (1.97–2.81)	—	Uncertain growth reference
India	Luwang and Datta (1982)	WA <70%	508	1.57 (1.26–1.94)	—	Cross-sectional; reverse causality
Papua New Guinea	Binns (1976)	WA <80%	630	1.75	—	No categorical sample size data
Sudan	Kossman et al. (2000)	WAZ <–2	28 753	1.32; 1.84[e]	OR= 1.13 (1.21–1.45); 1.75 (1.56–1.96)	No categorical sample size data
Thailand	Thongkrajai et al. (1990)	WA <75%	1 339	0.18 (0.06–0.43)	—	Local growth reference

— No data.

[a] Crude risk estimates represent incidence below cut-off point relative to incidence above cut-off point except where indicated as follows.

[b] Weight-for-age <25th percentile vs weight-for-age >75th percentile. Crude RR is a Mantel-Haenszel weighted risk ratio. Adjusted ORs are among children aged <9 months and ≥9 months, respectively. Adjusted for age, sex and previous morbidity.

[c] WAZ <–2 vs WAZ –1 to 0. Estimate of data reported in figure form.

[d] WAZ<–2 vs WAZ –1 to 0. Relative risks of diarrhoea on day of monthly visit during the dry and rainy seasons, respectively. Estimates of data reported in figure form.

[e] WAZ –3 to –2 vs WAZ ≥–1, and WAZ –4 to –3 vs WAZ ≥–1, respectively. OR was adjusted for age, sex, socioeconomic status, season, breastfeeding, vitamin A intake and previous morbidity.

Table 2.16 Combined estimate for diarrhoea incidence from dichotomous analysis

Study type	*Analysis level*	*Number of studies*	*Combined risk estimate (95% CI)*
Cohort	1	4	1.25 (1.04–1.50)
Cohort	2	4	1.24 (0.90–1.70)
Cohort	3	9	1.18 (1.05–1.33)
Cohort	4+5	3	1.51 (1.20–1.90)
Combined estimate	1+2+3+4+5	20	1.23 (1.12–1.35)

among these studies, with half reporting no significant effect. Individual studies reported crude risk estimates ranging between 0.78 in Brazil (Victora et al. 1990) and 1.86 in Mexico (Sepulveda et al. 1988). Three of four studies that adjusted for previous diarrhoea morbidity found a significantly increased risk of diarrhoea incidence associated with low weight-for-age (el Samani et al. 1988; Schorling et al. 1990; Wierzba et al. 2001). The fourth, in Bangladesh, observed an increased risk of diarrhoea among underweight children (RR=1.26) who had already suffered diarrhoea within the preceding three months, but the risk was not significant for those children without recent diarrhoea experience (RR=1.00) (Baqui et al. 1993). In the most recently published investigation, Wierzba et al. (2001) in Egypt observed that WAZ <–2 predisposed toward increased incidence of diarrhoea among children aged <3 years, yielding a relative risk of 1.7 (95% CI 1.2–2.3) compared to *z*-score >–2, after adjusting for age, sex, socioeconomic status, breastfeeding and previous episodes of diarrhoea.

Dichotomous relative risk estimate

Based on all cohort studies, a combined estimate suggested underweight children were at a significantly increased relative risk of 1.23 (95% CI 1.12–1.35) for developing an episode of diarrhoea (Table 2.16). The estimate based on a cut-off point of *z*-score <–2 (RR=1.25) was similar to the estimate using percentage of median <70% (RR=1.24) and percentage of median <75% (RR=1.18). Higher cut-off points of 80% or 85% yielded a higher relative risk of 1.51, possibly due to the small number of studies or the comparatively better nutritional status of the referent group.

Multi-categorical estimate

We also attempted to examine the risk of diarrhoea incidence according to multiple anthropometric categories, comparing incidence among moderately to severely underweight (e.g. WAZ <–2), mildly underweight (e.g. WAZ between –2 and –1) and "normal"(e.g. WAZ >–1) children. The

Table 2.17 Analysis levels for multiple categorical analysis according to anthropometric classification used in study

Analysis level	*Underweight categories*	*Cut-off point*
1	Moderate–severe	WAZ <–2
	Mild	WAZ –2 to –1
	Normal (referent)	WAZ >–1
2	Moderate–severe	WA <70%
	Mild	WA 70–80%
	Normal (referent)	WA >80%
3	Moderate–severe	WA <75%
	Mild	WA 75–90%
	Normal (referent)	WA >90%
4	Moderate–severe	WA <60
	Mild	WA 60–85%
	Normal (referent)	WA >85%

studies used different cut-off points based on percentage of median and *z*-score and were subdivided into analysis levels according to the anthropometric classification scheme used (Table 2.17). A subset of 10 cohort studies reporting multiple anthropometric categories provided sufficient statistical data for inclusion in a summary analysis (Table 2.18).

The different classification schemes complicated combining studies to a greater extent than in the dichotomous analysis. Eight of 10 studies, however, fell into the second or third analysis levels, which produced relative risks of 1.23 and 1.28, respectively, associated with moderate to severely low weight-for-age (Table 2.19). The corresponding risk estimates associated with mildly low weight-for-age were 1.17 and 0.95 for these analysis levels (Table 2.20). The overall risk estimates for diarrhoea based on all analysis levels were 1.25 (statistically significant) for moderate and severe underweight and 1.12 (not statistically significant) for mild underweight. Since the risk relationship was not significant for mild underweight, only the risk for moderate–severe underweight was considered toward the burden of disease estimate. This value (1.25) was similar to the value calculated in the dichotomous analysis (1.23). As there was no significant difference between the two estimates and the dichotomous value was based on a larger number of studies, the dichotomous value of 1.23 was selected for calculating burden of disease.

Summary of morbidity risk estimates

Three morbidity outcomes contributed to the overall calculation of disease burden: diarrhoea incidence, pneumonia/ALRI incidence and malaria incidence. Table 2.21 summarizes the risk of disease incidence associated with low weight-for-age for each outcome. There was no evidence that undernutrition contributed to incidence of measles and other disease relationships cannot be adequately quantified at this time.

Table 2.18 Cohort studies of diarrhoea incidence included in categorical analysis

Analysis level	*Study*	*Location*	*Sample size*	*Crude risk estimate (95% CI)*	
1	Victora et al. (1990)	Brazil	4 486	Severe–moderate Mild	0.94 (0.22–3.94) 1.93 (1.05–3.56)
2	Walia et al. (1989)	India	838	Severe–moderate Mild	1.37 (1.01–1.85) 1.05 (0.84–1.32)
2	Anand et al. (1994)	India	250	Severe–moderate Mild	1.07 (0.74–1.54) 0.98 (0.69–1.40)
2	Bhan et al. (1986)	India	1 399	Severe–moderate Mild	0.996 (0.85–1.17) 1.27 (1.10–1.48)
2	Sepulveda et al. (1988)	Mexico	239	Severe–moderate Mild	1.96 (1.20–3.21) 1.18 (0.85–1.64)
3	Mathur et al. (1985)	India	687	Severe–moderate Mild	1.10 (0.81–1.49) 0.72 (0.53–0.99)
3	Chen et al. (1981)	Bangladesh	207	Severe–moderate Mild	0.79 (0.44–1.42) 0.71 (0.38–1.32)
3	Gordon et al. (1964)	Guatemala	179	Severe–moderate Mild	2.56 (1.53–4.29) 1.64 (0.97–2.78)
3	el Samani et al. (1988)	Sudan	445	Severe–moderate Mild	1.22 (0.80–1.87) 1.03 (0.75–1.39)
4	Chowdhury et al. (1990)	Bangladesh	753	Severe–moderate Mild	1.47 (0.65–3.34) 2.15 (0.99–4.64)

Table 2.19 Combined estimates of diarrhoea incidence associated with severe to moderate underweight status relative to WAZ >–1

Analysis level	*Category*	*Number of studies*	*Combined risk estimate (95% CI)*
1	WAZ <–2	1	0.94 (0.22–3.94)
2	WA <70%	4	1.23 (0.95–1.60)
3	WA <75%	4	1.28 (0.85–1.94)
4	WA <60%	1	1.47 (0.65–3.34)
1+2+3		9	1.25 (1.02–1.53)
1+2+3+4		10	1.25 (1.03–1.52)

4.3 Risk of mortality due to perinatal conditions

"Perinatal conditions" are responsible for approximately 18% of all deaths among children aged <5 years in developing countries (de Onis et al. 1998). These deaths, concentrated in the neonatal period (≤28 days postpartum), result mostly from low birth weight, birth asphyxia and trauma, neonatal infections (e.g. tetanus and syphilis) and congenital

Table 2.20 Combined estimates of diarrhoea incidence associated with mild underweight status relative to WAZ >–1

Analysis level	*Category*	*Number of studies*	*Combined risk estimate (95% CI)*
1	WAZ –2 to –1	1	1.93 (1.05–3.56)
2	WA 70–80%	4	1.17 (1.05–1.31)
3	WA 75–90%	4	0.95 (0.68–1.34)
4	WA 60–85%	1	2.15 (0.99–4.64)
1+2+3		9	1.09 (0.92–1.29)
1+2+3+4		10	1.12 (0.95–1.32)

Table 2.21 Relative risk of morbidity associated with weight-for-age <–2 SDs

Morbidity outcome	*Relative risk (95% CI)*
Diarrhoea incidence	1.23 (1.12–1.35)
Pneumonia incidence	1.86 (1.06–3.28)
Malaria incidence	1.31 (0.92–1.88)

anomalies (Anonymous 1999). In developed regions such as North America and western Europe, 23% of neonatal deaths are due to congenital anomalies and an additional 65% are the result of other perinatal conditions (C. Stein, personal communication, 2001). Of the deaths from perinatal conditions, the largest share (32–65%) is attributed to low birth weight.

The Global Burden of Disease (GBD) study categorizes low birth weight, birth asphyxia, birth trauma and other conditions (such as neonatal sepsis, maternal and placental complications, respiratory distress, fetal blood loss, fetal haematological disorders, anaemia, perinatal infections and maternal diabetes) as "perinatal conditions", while deaths due to congenital anomalies, neonatal tetanus and syphilis are addressed separately (WHO 1992a). Among these conditions, low birth weight—specifically that due to IUGR—is the most strongly linked to undernutrition and is the only perinatal condition considered in our analysis.

Low birth weight is defined as birth weight below 2500 grams. It is a product of IUGR, preterm birth, or both in combination (Kramer 1987). In developing countries, the majority of low-birth-weight births are due to IUGR (usually defined as birth weight less than the tenth percentile of weight-for-gestational-age) whereas preterm birth (<37 weeks gestation) is the predominant cause in most developed countries (Ashworth 1998). Preterm low-birth-weight infants tend to have higher neonatal mortality rates than full-term, growth-retarded infants; at

highest risk are low-birth-weight infants who are both growth-retarded and preterm (Barros et al. 1992; Behrman et al. 1971; Cogswell and Yip 1995; Gray et al. 1991; Sappenfield et al. 1987).

The etiology of IUGR is complex and multifactorial. In developing countries maternal undernutrition is the major determinant of IUGR, and evidence across populations has demonstrated a greater incidence of IUGR births among women who are underweight or stunted prior to conceiving, or who fail to gain sufficient weight during pregnancy (Bakketeig et al. 1998; King and Weininger 1989; Kramer 1987; WHO 1997). Poor maternal nutrition during pregnancy is thought to account for 14% of IUGR in developing countries; maternal stunting may account for 18.5% (ACC/SCN 2000b). Malaria, other acute and chronic infections and cigarette smoking are also important etiologic factors for IUGR in developing countries. In developed countries, smoking is the most important determinant of IUGR, followed by factors such as maternal nutrition, pre-eclampsia, genetic factors and alcohol or drug use (Bakketeig et al. 1998). IUGR is also associated with multiple births and primiparity (Cogswell and Yip 1995).

Fetal growth retardation takes different forms, which may have different implications for neonatal and infant health. IUGR can be subdivided into asymmetric (wasted) IUGR, characterized by adequate length and head circumference, but reduced weight and low ponderal index; and symmetric (stunted) IUGR, in which the ponderal index is normal but weight, length and head circumference are all reduced (Bakketeig 1998). Symmetric IUGR generally reflects early onset or chronic undernutrition *in utero*, while asymmetric IUGR is thought to result from undernutrition of later onset (Ashworth 1998). The difference may be clinically important, as asymmetrically growth-retarded infants have demonstrated higher risks of asphyxia, hypoglycaemia and other morbidities, and higher mortality rates in the early neonatal period (Ashworth 1998; Caulfield et al. 1991; Villar et al. 1990). Stunted infants, on the other hand, have greater risks of mortality in later infancy (Ashworth 1998; Balcazar and Haas 1990; Cheung et al. 2001).

The evidence relating low maternal BMI to preterm birth is more ambiguous. A 1995 WHO meta-analysis based on data sets from 20 developed and developing countries calculated a combined OR of 1.3 (95% CI 1.1–1.4) for preterm birth associated with pre-pregnancy BMI $<20\,kg/m^2$ (WHO 1995b). Individual studies, however, have been inconsistent. In Papua New Guinea, BMI was found to be a significant predictor of preterm delivery; a one-unit increase in BMI was associated with a reduced risk of preterm delivery (OR=0.79, 95% CI 0.66–0.94), and remained significant after being adjusted for haemoglobin concentration, smoking, gravidity and anti-malarial use (Allen et al. 1998). A case–control study in India observed an increased risk of preterm birth with underweight status as well (Mavalankar et al. 1994). Other studies in Malawi (Pelletier et al. 1995) and Indonesia (Husaini et al. 1995)

reported little or no association between pre-pregnancy BMI and preterm delivery. In developed settings, studies from Canada (Kramer et al. 1995), Italy (Spinillo et al. 1998), the United Kingdom (Sebire et al. 2001) and the United States (Edwards et al. 1979; Naeye 1990; Siega-Riz et al. 1996) observed higher rates of preterm labour or delivery among underweight women, while other studies in Canada (Kramer et al. 1992), Finland (Rantakallio et al. 1995) and Sweden (Cnattingius et al. 1998) did not. Recently, in a study in the United States of America based on the National Maternal and Infant Health Survey found that underweight women had an increased risk for preterm delivery only when their pregnancy weight gain was inadequate (Schieve et al. 2000). Because of the uncertain risk relationship, low birth weight due to preterm birth was excluded from our analysis.

Although many studies conducted in developing countries examined the influence of maternal body size on infant birth weight and others compared neonatal mortality rates according to birth weight, few studies directly compared maternal weight to neonatal or perinatal mortality. These studies used postpartum weight as a proxy for pre-pregnant weight, and therefore, possible effects of pregnancy weight gain cannot be excluded. Among these, a slightly elevated risk of early neonatal death (adjusted OR=1.4, 95% CI 1.0–1.9) was associated with maternal weight below 50 kg in a nested case–control study in Brazil (Gray et al. 1991). The authors adjusted for low birth weight in one regression model to distinguish between risk factors that might operate in part through low birth weight and factors that might influence neonatal death independently. Low maternal weight remained significantly associated with neonatal death only when low birth weight was excluded from the model, suggesting maternal weight influences neonatal death through low birth weight.

In other studies, maternal weight <40 kg and low weight-to-height ratio were significantly associated with perinatal death in Ahmedabad, India (OR = 2.9, 95% CI 1.8–4.7 and OR=3.0, 95% 1.9–4.4, respectively), adjusted for maternal age, parity, obstetric history and antenatal care (Mavalankar et al. 1994). In the Sudan, maternal weight below 50 kg was significantly associated with perinatal death, after adjustment for birth interval, prior fetal loss and antenatal care, among other factors (adjusted OR=2.3, 95% CI 1.1–4.8) (Taha et al. 1994). In a community-based case–control study in Punjab, perinatal death was significantly associated with weight <40 kg and height <152 cm, but not BMI <20 kg/m^2, after controlling for factors such as birth interval and length of gestation (Sachar and Soni 2000).

In developed countries, there is a consistent association between maternal underweight status and low birth weight, but the evidence relating maternal underweight status to perinatal death has been mixed. In New York City, low BMI prior to pregnancy was associated with slightly, but not significantly, higher perinatal mortality rates in one hospital-

based study (Bracero and Byrne 1998). Another analysis using data from the USA Collaborative Perinatal Study reported reduced perinatal mortality for infants of underweight mothers; among mothers with low pregnancy weight gain (<0.8 kg/month), however, underweight status was associated with higher perinatal mortality (Naeye 1990). In Australia, the odds ratios for stillbirth/neonatal death (adjusted for gestational age, parity, smoking and maternal age) were 1.65 and 2.64 among women with low postpartum BMI (defined for postpartum women as 20–24.4 kg/m^2) and very low postpartum BMI (<20 kg/m^2), respectively (Cattanach et al. 1993). In Finland, perinatal and childhood mortality rates were similar between women having low and normal BMI (Rantakallio et al. 1995), and in Sweden low maternal BMI was associated with reduced risk of late fetal death and a slightly, but not significantly, reduced risk of early neonatal death relative to maternal BMI of 20–25 kg/m^2 (Cnattingius et al. 1998).

Such studies have generated uncertainty about the significance of low birth weight, *per se*, as an intermediate variable in the causal pathway between prenatal factors and perinatal mortality (Rush 2001; Wilcox 2001). As with other anthropometric indicators, birth weight is regarded as a proxy for underlying biological processes that must be inferred. All prenatal factors that influence birth weight may not affect infant health equally. For example, high altitude is associated with lower birth weight, but not necessarily higher mortality (Cogswell and Yip 1995; Wilcox 2001). Therefore, caution must be taken when generalizing across populations.

Estimate of mortality due to perinatal conditions

The relationship between maternal underweight status and neonatal mortality was estimated by considering two stages of the conceptual pathway mediated through IUGR. First, what is the proportion of IUGR attributable to poor maternal pre-pregnancy anthropometric status? And second, what proportion of neonatal mortality can be attributed to IUGR? There are four components to this equation: the proportion of IUGR births among live births in each subregion, an estimate of infant mortality risk associated with IUGR, subregional prevalence of underweight status among women of reproductive age and an estimate of the risk of IUGR associated with pre-pregnant underweight status.

By estimating the risk of IUGR in relation to pre-pregnancy BMI only, we are not considering the fraction of IUGR attributable to inadequate weight gain during pregnancy, and are potentially underestimating the overall influence of maternal weight on infant birth weight. Gains in pre-pregnancy and pregnancy weight have independent, additive effects on birth weight (Krasovec and Anderson 1991). Across diverse populations, low pregnancy weight gain, particularly in the second and third trimesters, has been associated with risk ratios of 1.7 to 2.0 for IUGR; among mothers who are below average height or weight before preg-

nancy, the risk ratios are 3.1 and 5.5, respectively (Strauss and Dietz 1999; WHO 1997). The prevalence data on pregnancy weight gain in developing countries is scarce, but there is evidence that poor weight gain is common, especially among women who have low pre-pregnancy BMI and must gain more to compensate. In rural Indonesia, for example, 79% of women in a cohort study failed to reach their recommended total weight gain, including 82.4% of the women with a pre-pregnancy BMI below 20 kg/m^2 (Winkvist et al. 2002).

Methods

The overall attributable fraction of neonatal mortality due to maternal underweight status was calculated as the product of the attributable fraction of neonatal mortality due to IUGR and the attributable fraction of IUGR due to maternal underweight status. Attributable fractions were calculated according to the formula [(P)(RR–1)]/[1+(P)(RR–1)], where P is the prevalence of the risk factor and RR is the associated risk ratio. Prevalence and risk ratio estimates were derived from available published and unpublished sources.

Incidence of IUGR

Classification of IUGR in developing countries can be problematic because gestational age cannot be accurately determined in most cases, and reference curves that are adjusted for gestational age are not widely used (ACC/SCN 2000b). Therefore, the incidence of low birth weight at term (referred to as "term-LBW" or "IUGR-LBW") is used as the best proxy for IUGR incidence, although this will underestimate the actual incidence of IUGR because it excludes growth-retarded infants born preterm and those born over 2500 grams but below the tenth percentile for their gestational age (de Onis et al. 1998).

Incidence rates of IUGR-LBW for most developing countries were previously estimated by de Onis et al. (1998) using low birth weight estimates from the WHO database on Low Birth Weight, compiled by the Maternal Health and Safe Motherhood Programme (WHO 1992b). The authors applied a linear regression model [Y=–3.2452+0.8528X] derived from numerous studies in developing countries where birth weight and valid gestational age assessments were recorded. The dependent variable in the model was IUGR-LBW and the independent variable was the total incidence rate of low birth weight (β=0.8528; SE =0.0282; P=0.0001; r=0.96) (de Onis et al. 1998; Villar et al. 1994). The linear regression model was found to be unsuitable as a predictor of IUGR-LBW in developed countries, however.

Incidence rates of IUGR-LBW in developed countries were based on Medline (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi), Popline (http://db.jhuccp.org/popinform/index.stm) and Google (http://www.google.com/) searches for country-level data sets and nationally-representative study samples that reported rates of IUGR-

LBW or sufficient statistical information (including numbers of births, mean birth weights and SDs or percentiles according to week of gestation) for calculating the rate of IUGR-LBW among total live births. The studies used in estimating rates were published between 1993 and 2001. The data reported in most studies were collected between 1992 and 2001, although in a few instances the data reflect populations from as early as 1984. In general, the reported data were limited to singleton births and excluded births having major congenital anomalies. Where data were unavailable—as was the case for the majority of European countries—the rate of IUGR-LBW was assumed for these purposes to be equivalent to the rate in AMR-A, or 2.5% of total live births (Kramer et al. 2001; Ventura et al. 1999). Estimates of small-for-gestational-age incidence were not appropriate for this analysis because they can include births weighing over 2500 grams. The estimated incidence rate of IUGR-LBW for each country was multiplied by the estimated total number of live births in that country for the year 2000; the country estimates were then collapsed into the 14 subregions in order to calculate subregional rates (Table 2.22). Estimates of live births per year for each country were taken from the United Nations Population Division (UN 2001).

IUGR and risk of neonatal mortality

Rice et al. (unpublished) calculated the risk of neonatal mortality associated with IUGR-LBW in an initial review and meta-analysis of the rela-

Table 2.22 Incidence of IUGR-LBW, by subregion

Subregion	*Total live births per year (000s)*	*IUGR-LBW births as percentage of total live births*	*Estimated number of IUGR-LBW births per year (000s)*
AFR-D	11 185	10.5	1 176
AFR-E	13 240	9.3	1 229
AMR-A	4 426	2.5	112
AMR-B	9 303	6.7	621
AMR-D	2 011	6.6	133
EMR-B	3 410	4.1	141
EMR-D	12 003	12.1	1 455
EUR-A	4 233	2.5	107
EUR-B	3 743	2.6	96
EUR-C	2 527	2.5	63
SEAR-B	5 933	4.2	250
SEAR-D	42 147	22.7	6 866
WPR-A	358	2.3	8
WPR-B	26 840	2.9	769
World	129 493	10.1	13 027

tionship between malnutrition and cause-specific mortality. The authors conducted a Medline search for English-language research reports published between 1966 and 1999 on child mortality, nutritional status, low birth weight, perinatal causes of death and neonatal mortality, excluding studies conducted in developed countries. They selected community and hospital-based studies that reported sufficient statistical and follow-up data on infants who were low birth weight at term, excluding data on very low birth weight (<1000 g) infants. Follow-up times and cut-off points varied among the studies as summarized in Table 2.23. Unadjusted data from studies were combined according to the random-effects method described by Morris (Everson and Morris 1983; Morris 1983). The combined risk estimate from all studies was 5.53 (3.74–8.17) and the risk estimate from the subset of studies using a 2500 g cut-off was 6.00 (3.63–9.90). Because the overall combined estimate of 5.53 was heavily weighted toward studies with short post-natal follow-up periods and higher birth weight cut-off points, we selected the 6.00 risk estimate for our calculations.

An elevated mortality risk has been observed among IUGR-LBW infants in developed countries, as well. Ashworth (1998) estimated the risk of neonatal mortality according to birth weight, combining 10 data sets from developing and developed countries (predominantly weighted toward births in the United States) and excluding most preterm births. Individual studies showed a consistent dose–response effect of increasing mortality risk with decreasing birth weight. Compared to birth weight of 2500–2999 g, the overall relative risk associated with birth weight between 2000 g and 2499 g was 4.0 and the risk associated with birth weight 1500–1999 g was 18.0. An overall dichotomous risk estimate comparing birth weight <2500 g to >2500 g was not reported, but after pooling published data from four (Behrman et al. 1971; Binkin et al. 1985; Lubchenco et al. 1972; Sappenfield et al. 1987) of six United States studies included in the estimate, the combined risk for neonatal mortality associated with birth weight <2500 g at term was 10.64 (95% CI 9.94–11.38). This crude estimate is based on mostly white, singleton births between 38 and 42 weeks gestation and excludes births below 1000 g.

Prevalence of low maternal BMI

Refer to section 3: prevalence of underweight among women of reproductive age.

Maternal pre-pregnancy BMI and risk of IUGR

In 1995, WHO published a meta-analysis based on data sets from 25 studies that related maternal anthropometry to pregnancy outcomes (WHO 1995b). These sets represented over 111 000 births in 20 developing and developed countries throughout the world. Countries were grouped together for analysis according to similarities in the population

Table 2.23 Risk of neonatal (≤28 days) or early neonatal (≤7 days) death among term infants according to birth weight[a]

Location	*Study*	*Study type*	*Neonatal period studied*	*Sample size*	*Birth weight comparison*	*Crude relative risk (95% CI)*
Bolivia	Haas et al. (1987)	Retrospective cohort	≤2 days	12 280	<2900 g vs ≥2900 g	3.63 (1.93–6.82)
Brazil	Barros et al. (1987)	Prospective cohort	≤7 days	5 356	<2500 g vs ≥2500 g	6.28 (2.57–15.33)
Brazil	de Almeida and Jorge (1998)	Retrospective cohort	≤28 days	2 014	<10th percentile vs ≥10th percentile	10.61 (3.34–33.72)
Guatemala	Mata (1978)	Community-based cohort	≤28 days	385	<2500 g vs ≥2500 g	3.41 (0.63–18.40)
India	Arora et al. (1987)	Prospective cohort	≤28 days	200	<10th percentile vs ≥10th percentile	3.03 (0.32–28.64)
India	Bhargava et al. (1985)	Prospective cohort	≤7 days	13 806	<–2 SDs vs ≥–2 SDs[b]	11.08 (7.34–16.73)
India	Ghosh et al. (1979)	Community-based cohort	≤28 days	3 650	≤2500 g vs >2500 g	6.25 (3.14–12.42)
Mexico	Balcazar and Haas (1991)	Retrospective cohort	≤3 days	8 526	<10th percentile vs ≥10th percentile	5.35 (2.69–10.65)
Mexico	Haas et al. (1987)	Retrospective cohort	≤2 days	9 228	<2900 g vs ≥2900 g	2.13 (1.12–4.06)
Combined estimate (all studies)						5.53 (3.74–8.17)
Combined estimate (Barros et al. 1987; Mata et al. 1978; Ghosh et al. 1979)						6.00 (3.63–9.90)

[a] Table adapted from Rice et al. (unpublished).

[b] SD according to North Indian reference.

Table 2.24 Analysis groups used by WHO Collaborative Study on Maternal Anthropometry and Pregnancy Outcomes

Group	*Countries/data sets included in group*	*25th quartile cut-off (BMI)*	*75th quartile cut-off (BMI)*
1	India (Pune), Sri Lanka	17.3	20.1
2	China, Gambia, India (Hyderabad), Indonesia, Myanmar, Nepal (Rural), Viet Nam	18.4	21.0
3	Guatemala, Malawi, Thailand	19.4	22.7
4	Argentina, Cuba, United Kingdom, USA	20.1	25.0
5	Colombia, Ireland, USA	21.0	26.7

Source: WHO 1995a.

Table 2.25 Odds ratio for IUGR according to pre-pregnancy BMI

Group	*BMI cut-off*	*BMI referent*	*OR for IUGR (95% CI)*
1	≤17.3	≥20.1	0.7 (0.1–2.6)
2	≤18.4	≥21.0	1.8 (1.5–2.3)
3	≤19.4	≥22.7	1.8 (1.3–2.5)
4	≤20.1	≥25.0	2.0 (1.8–2.2)
5	≤21.0	≥26.7	1.5 (1.3–1.7)
Combined	≤19.7[a]	≥24.2[a]	1.8 (1.7–2.0)

[a] Combined BMI cut-offs were not reported. Values here are the weighted means of the 25th and 75th quartile values from each data set.

Source: WHO 1995a.

distributions for various anthropometric indicators, including pre-pregnancy weight, height, BMI and arm circumference. Table 2.24 describes the composition of analysis groups and the corresponding BMI cut-offs. Unadjusted odds ratios for pregnancy outcomes were then calculated by comparing the lowest quartile to the highest quartile within each analysis group.

Results are presented in Table 2.25. The meta-analysis reported significantly greater risk for IUGR associated with pre-pregnancy BMI below 19.7 kg/m^2 relative to BMI above 24.2 kg/m^2, with an overall OR of 1.8 (95% CI 1.7–2.0). This estimate was consistent across the analysis groups (with the exception of group one studies) despite the differences in their cut-off and reference points.

Results

Subregional estimates of the attributable fraction of IUGR due to low pre-pregnancy BMI are listed in Table 2.26 for women aged 15–29 and

Table 2.26 Attributable fraction of IUGR due to low BMI among women aged 15–44 years, by subregion

	Prevalence of BMI ≤20 kg/m² (%)		*Fraction of IUGR attributable to BMI ≤20 kg/m² (%)*	
Subregion	*15–29 years*	*30–44 years*	*15–29 years*	*30–44 years*
AFR-D	43.3	29.5	25.7	19.1
AFR-E	37.8	27.4	23.2	18.0
AMR-A	25.1	19.5	16.7	13.5
AMR-B	22.7	11.1	15.4	8.2
AMR-D	11.7	9.3	8.6	6.9
EMR-B	28.4	12.7	18.5	9.2
EMR-D	40.1	32.6	24.3	20.7
EUR-A	20.9	13.8	14.3	9.9
EUR-B	24.5	14.0	16.4	10.1
EUR-C	24.5	9.7	16.4	7.2
SEAR-B	44.4	12.1	26.2	8.8
SEAR-D	56.7	42.1	31.2	25.2
WPR-A	40.9	27.1	24.7	17.8
WPR-B	32.6	24.8	20.7	16.6

30–44 years based on the prevalence of low BMI and the risk ratio of 1.8 reported by the WHO meta-analysis. Table 2.27 lists the attributable fraction of neonatal deaths due to IUGR-LBW for each subregion based on the prevalence of IUGR-LBW and the risk ratio of 6.0 calculated by Rice et al. (unpublished). The results of both tables are summarized in Table 2.28 with the overall attributable fraction of neonatal deaths due to low pre-pregnancy BMI. The fraction of neonatal deaths attributed to maternal BMI ranged between 0.8% (women aged 30–44 years in EUR-C) and 16.6% (women aged 15–29 years in SEAR-D).

4.4 Other health outcomes

Other important health outcomes related to underweight status were reviewed, but ultimately not used for burden of disease estimates. The adverse effects of undernutrition on risk of dysentery and persistent diarrhoea, pregnancy outcome, cognitive function and chronic diseases in later life are described below.

Dysentery and persistent diarrhoea

Dysentery, defined by diarrhoea with blood, was specifically examined in two community-based prospective studies, both among children aged <24 months in Bangladesh. Henry et al. (1987) observed no significant difference in incidence of dysentery between children having weight-for-

Table 2.27 Attributable fraction of neonatal mortality due to IUGR-LBW, by subregion

Subregion	*Incidence of IUGR-LBW (% of total live births per year)*	*Attributable fraction of neonatal deaths due to IUGR-LBW (%)*
AFR-D	10.5	34.4
AFR-E	9.3	31.7
AMR-A	2.5	11.1
AMR-B	6.7	25.1
AMR-D	6.6	24.8
EMR-B	4.1	17.0
EMR-D	12.1	37.7
EUR-A	2.5	11.1
EUR-B	2.6	11.5
EUR-C	2.5	11.1
SEAR-B	4.2	17.4
SEAR-D	22.7	53.2
WPR-A	2.3	10.3
WPR-B	2.9	12.7

Table 2.28 Attributable fraction of neonatal mortality due to low maternal BMI, by subregion

	Attributable fraction of IUGR due to BMI ≤20 kg/m^2 (%)		*Attributable fraction of neonatal deaths due to IUGR (%)*	*Attributable fraction of neonatal deaths due to low BMI (%)*	
Subregion	*15–29 years*	*30–44 years*		*15–29 years*	*30–44 years*
AFR-D	25.7	19.1	34.4	8.9	6.6
AFR-E	23.2	18.0	31.7	7.4	5.7
AMR-A	16.7	13.5	11.1	1.9	1.5
AMR-B	15.4	8.2	25.1	3.9	2.1
AMR-D	8.6	6.9	24.8	2.1	1.7
EMR-B	18.5	9.2	17.0	3.2	1.6
EMR-D	24.3	20.7	37.7	9.2	7.8
EUR-A	14.3	9.9	11.1	1.6	1.1
EUR-B	16.4	10.1	11.5	1.9	1.2
EUR-C	16.4	7.2	11.1	1.8	0.8
SEAR-B	26.2	8.8	17.4	4.6	1.5
SEAR-D	31.2	25.2	53.2	16.6	13.4
WPR-A	24.7	17.8	10.3	2.5	1.8
WPR-B	20.7	16.6	12.7	2.6	2.1

age <75% and those above 75%. Black et al. (1984) found that incidence of *Shigella* diarrhoea, specifically, did not vary significantly according to weight-for-age.

The incidence of persistent diarrhoea, defined as an episode lasting 14 or more days, was greater among underweight children in community-based studies in Brazil (Guerrant et al. 1992; Schorling et al. 1990), India (Bhandari et al. 1989) and Bangladesh (Baqui 1990). Children aged <5 years having weight-for-age at or below 75% in the Brazil cohort study were at a relative risk of 1.59 for persistent diarrhoea. In Bangladesh, the relative risk for persistent diarrhoea among children having *z*-score <–2 was 1.29. Combining unadjusted figures from the two cohort studies yielded an overall relative risk of 1.35 (95% CI 0.97–1.90). In a case–control study in India, pre-morbid weight-for-age below 70% was significantly more common among persistent diarrhoea cases compared to healthy age-matched controls, producing an OR of 3.25 (95% CI 1.46–7.29). A community-based cohort study in Guinea-Bissau (Molbak et al. 1997) assessed risk according to stature and observed a statistically non-significant relative risk of 1.17 (95% CI 0.68–2.01) associated with height-for-age *z*-score <–2.

Other pregnancy outcomes

There is extensive literature supporting the influence of maternal undernutrition on fetal growth and risk of low birth weight as described in previous sections, but the relationship between low pre-pregnancy BMI and other adverse reproductive outcomes such as fetal death and maternal mortality is less clear. Research on maternal undernutrition as a risk factor for congenital anomalies, other than those attributable to micronutrient deficiencies (e.g. folic acid) is lacking.

Obstructed labour and maternal mortality

Few studies from developing countries have been published relating low BMI to maternal mortality, although studies have described an increased risk of obstructed or prolonged labour associated with maternal height of 160 cm and below (Adadevoh et al. 1989; Anonymous 1984; Bhatt et al. 1967; Essex and Everett 1977; Konje and Ladipo 2000; Kwawukume et al. 1993; Mati 1983; Sokal et al. 1991; Tsu 1992), and one study in India reported an increased risk of caesarean section associated with low weight–height product index (weight times height over weight times height of reference population median) (Thilothammal et al. 1992). This risk may result from cephalopelvic disproportion (CPD) between mother and fetus (Rush 2000), increasing the likelihood of intrapartum caesarean delivery (Adadevoh et al. 1989; Merchant et al. 2001; Sokal et al. 1991; Tsu 1992) and perinatal distress (Merchant et al. 2001). In Nigeria, primigravida women below 150 cm were at a relative risk of 10.34 for CPD relative to women 160 cm or taller (Harrison et al. 1985). In Malawi, the odds ratio for CPD associated

Table 2.29 Study of early fetal death (<28 weeks)

Study	*Location*	*Study type*	*Comparison*	*Crude risk estimate (95% CI)*	*Adjusted risk estimate (95% CI)*
Agarwal et al. (1998)	India	Community-based cohort	Weight <42.5 kg vs >42.5 kg	RR = 1.21 (1.05–1.38)	—
Same as above	Same as above	Same as above	Height <147.5 cm vs >147.5 cm	RR = 1.21 (1.06–1.38)	—

— No data.

with height at or below 154 cm was 3.8, adjusted for birth weight and parity (Brabin et al. 2002).

The 1995 WHO meta-analysis examined assisted delivery, defined as non-spontaneous delivery covering a range of complications (but not prolonged labour), and estimated an odds ratio between 1.0 and 2.1 (1.6, overall) associated with maternal height below the 25th percentile (WHO 1995b). The odds ratio for assisted delivery according to pre-pregnancy BMI was 0.7, explained as the result of constrained fetal growth reducing the risk of CPD; however, numerous studies have found that an association between maternal anthropometric status and assisted delivery remains after adjusting for multiple parameters of fetal size (Witter et al. 1995).

Fetal death

Fetal death may be more common among underweight mothers (Tables 2.29 and 2.30). A community-based cohort study in India reported significant risks of spontaneous abortion (defined in the study as death <28 weeks' gestation) and late fetal death (>28 weeks) associated with both short stature and low absolute weight (Agarwal et al. 1998). A case–control study in the Sudan reported an adjusted odds ratio for stillbirth of 2.3 associated with absolute weight below 50 kg (Taha et al. 1994). On the other hand, Conde-Agudelo et al. (2000) analysed over 800 000 births recorded in the Perinatal Information System database, a system used by over 700 hospitals throughout Latin American and the Caribbean, and found no significant risk of fetal death associated with BMI <19.8 kg/m^2 relative to BMI between 19.8 and 26.0 kg/m^2.

Post-neonatal consequences of IUGR

Growth-retarded neonates may experience partial catch-up growth in the first two years of life relative to non-IUGR controls, but they remain shorter and lighter than controls and are more likely to be classified as underweight (Martorell et al. 1998). They are at increased risk of infection in infancy as a result of multiple immunological abnormalities, including reduced T- and B-lymphocyte numbers and activity, lower

Table 2.30 Studies of stillbirth

Study	*Location*	*Study type*	*Comparison*	*Crude risk estimate (95% CI)*	*Adjusted risk estimate (95% CI)*
Agarwal et al. (1998)	India	Community-based cohort	Weight <45 kg vs >45 kg	RR = 5.07 (2.94–8.73)	—
Same as above	Same as above	Same as above	Height <147.5 cm vs >147.5 cm	RR = 4.18 (2.88–6.05)	—
Taha et al. (1994)	Sudan	Facility-based case–control	Weight <50 kg vs ≥70 kg	OR = 2.0 (1.0–4.1)	OR = 2.3 (1.1–4.8)[a]
Conde-Agudelo et al. (2000)	Latin America, Caribbean	Facility-based cohort	BMI <19.8 kg/m^2 vs 19.8–26.0 kg/m^2	—	RR = 0.98 (0.88–1.08)[b]

— No data.

[a] OR adjusted for prior fetal loss, number of antenatal care visits and malaria in early pregnancy. Several socioeconomic status indicators were assessed, but none was significantly associated with stillbirth and therefore were not included in the final regression model.

[b] Relative risk adjusted for antenatal care, age, literacy, cigarette smoking and prior fetal loss. Study addressed fetal death, defined as death >20 weeks' gestation.

levels of IgG and impaired bactericidal polymorphonuclear neutrophil function (Ferro-Luzzi et al. 1998; Xanthou 1985). Depending on the severity of the growth retardation, this immune deficiency may persist into later childhood (Chandra 1977). In addition to higher neonatal mortality rates, IUGR infants are at increased risk of mortality in the post-neonatal period, including an increased risk of sudden infant death syndrome (Ferro-Luzzi et al. 1998).

IUGR may increase the risk of neurological dysfunction and mild cognitive impairment. IUGR infants show higher rates of hyperactivity, attention deficit and impaired motor coordination, depending on the degree of growth retardation, but the association of IUGR with low socioeconomic status and hypoxia makes it difficult to interpret the impact of IUGR (Goldenberg et al. 1998). Similarly, IUGR children have manifested small, but statistically significant IQ and developmental deficits, aggravated by impoverished environmental surroundings and poor psychosocial stimulation, but few studies have been conducted in developing countries, and socioeconomic factors may be confounding the relationship (Grantham-McGregor et al. 1998).

IUGR has been associated with increased susceptibility to chronic diseases in later life. The "fetal origins of disease" hypothesis posits that fetal undernutrition causes permanent structural and metabolic changes that potentiate subsequent risk of cardiovascular and endocrine disease (Barker 1995). IUGR infants have demonstrated insulin resistance and

higher blood pressure in childhood, and increased rates of ischaemic heart disease and non-insulin-dependent diabetes mellitus have been observed among adults who were born growth-retarded (Barker 1995; Chatelain et al. 1998; Law et al. 2001; Stein et al. 1996). Studies are inconsistent and causality is uncertain, but the possible risk relationship has important health implications for developing countries as they undergo epidemiological transition.

Cognitive function

Cognitive function has been studied in terms of global measures of development and intelligence such as IQ, along with school performance and more narrowly defined intellectual, psychomotor, and behavioural skills such as attention, memory, verbal reasoning, motivation, visual–spatial abilities and social interaction. An array of psychometric tests and scales have been used to evaluate these functions, including modified versions of Stanford-Binet, Weschler Intelligence Scale for Children (WISC), Bayley Scales of Infant Development, Griffiths Mental Development Scale, Goodenough Drawing Test, Raven's Progressive Matrices, Piagetian tests of conservation and the Bender Visual Motor Gestalt Test. A major portion of research into malnutrition and cognition has addressed whether early childhood presents a critical period of vulnerability during which severe undernutrition causes lasting cognitive deficits in later life, and if potential deficits are amenable to subsequent nutritional and psychosocial interventions.

The immediate response to acute malnutrition is irritability, lethargy and apathy (Grantham-McGregor 1984). During this stage, children demonstrate lower activity levels and reduced exploratory behaviour and developmental quotients tend to be extremely low (Grantham-McGregor 1984). Electroencephalograms performed on children with acute malnutrition show nonspecific abnormalities (e.g. diffuse slowing of background rhythm) that improve with recovery (Chopra and Sharma 1992). During recovery and rehabilitation, behaviour improves to normal or near-normal levels and developmental quotients generally improve, but the long-term developmental implications remain uncertain (Grantham-McGregor 1995).

Numerous short-term and long-term follow-up studies have reported lasting developmental deficits associated with an early history of marasmus and/or kwashiorkor. Persistent deficits have been described in short-term memory (Nwuga 1977), visual-spatial perception (Champakam et al. 1968; Cravioto et al. 1971; Ghai 1975; Hoorweg and Stanfield 1976; Reyes et al. 1990; Stoch and Smythe 1976), motivation (Stoch and Smythe 1967), Piagetian conservation tasks (Galler and Ramsey 1987) and perceptual-motor function (Grantham-McGregor et al. 1997). Poor academic performance and behavioural learning disabilities have also been recognized (Galler et al. 1984, 1990; Richardson et al. 1973).

Studies that assessed general measures of cognitive function among school-age children have observed significantly lower overall developmental and intelligence scores associated with early malnutrition (Berkman et al. 2002; Bhat et al. 1973; Birch et al. 1971; Botha-Antoun et al. 1968; Cabak and Najdanvic 1965; Cravioto et al. 1971; Fisher 1972; Galler et al. 1983, 1987a, 1987b; Hertzig et al. 1972; Ivanovic et al. 2000; McLaren et al. 1973; Mehta et al. 1975; Mendez and Adair 1999; Parekh et al. 1974; Pek et al. 1967; Sigman et al. 1991; Srikantia and Sastri 1971; Stoch and Smythe 1967, 1976; Udani et al. 1976). IQ differentials among these studies were predominantly in the order of 8 to 18 points. Studies in South Africa (Evans et al. 1971, 1980), however, observed no significant IQ difference between early kwashiorkor cases and non-hospitalized siblings. In Jamaica, Richardson et al. (1978) observed that height-for-age or weight-for-height among severely malnourished infants had no significant effect on IQ scores at school age after adjusting for social and environmental factors.

Among most of the studies showing long-term deficits, it could not be distinguished whether the observed impairment was related to the severe episode of acute malnutrition or to underlying or subsequent chronic undernutrition. In Jamaica, Grantham-McGregor (1982) reported that initial height-for-age, but not weight-for-height or presence of oedema, significantly predicted developmental quotients one month after hospitalization, suggesting the presence of chronic malnutrition was more closely associated with cognitive function than acute malnutrition (Grantham-McGregor 1982; Grantham-McGregor et al. 1989a, 1989b). In a long-term longitudinal study by Walker et al. (2000) height and head circumference in the first 24 months of life were found to be more significantly predictive of IQ at age 11 years than were anthropometric measurements taken at or near the time of cognitive testing, even after controlling for age, sex and socioeconomic factors, suggesting chronic malnutrition at an early age could have enduring effects on intelligence despite subsequent improvements in growth (Grantham-McGregor et al. 2000). In Peru, school-age children who were severely stunted in the second year of life scored 10.0 points lower than those moderately stunted or not stunted in the second year of life, after adjusting for socioeconomic status and schooling (Berkman et al. 2002). In the Philippines, severe stunting at 2 years of age was significantly associated with cognitive deficits at age 8 years, adjusting for schooling, socioeconomic status, sex and other covariates, but the differences were not significant by age 11 years (Mendez and Adair 1999). In Jamaica, Richardson (1976), observed that the long-term cognitive effect of early acute malnutrition was more heavily determined by chronic undernutrition and social background factors such as caretaker capability, presence of electricity and appliances, and child's access to toys, radio or stories. For the children having adequate growth and an advantageous social back-

ground, malnutrition during infancy was associated with an average IQ only 2 points lower than those not malnourished during infancy; for those experiencing both an unfavourable background and inadequate growth, the difference in IQ between children with and without malnutrition during infancy was 9 points by age 6–10 years (Richardson 1976).

A large number of correlational studies have examined the association between current anthropometric status and cognitive function. Low height-for-age was significantly associated with low IQ or poor school achievement in Brazil (Paine et al. 1992), China (Jamison 1977), Guatemala (Johnston et al. 1987), India (Agarwal et al. 1987), Nepal (Moock and Leslie 1986) and the Philippines (Florencio 1988), but not Chile (Colombo et al. 1988). Deficits in other developmental scores were observed among stunted children in Chile (Monckeberg 1972), Guatemala (Lasky et al. 1981), India (Agarwal et al. 1989), Jamaica (Powell and Grantham-McGregor 1985) and Nigeria (Ashem and Janes 1978). Low weight-for-age has been associated with delayed motor development (Agarwal et al. 1992; Groos 1991; Heywood et al. 1991; Sathy et al. 1991; Vazir et al. 1998), delayed language development (Agarwal et al. 1992; Vazir et al. 1998) and poor performance on conservation tasks (Agarwal et al. 1989). Lower scores on aggregate measures such as IQ have been observed among underweight children in Ethiopia (Aboud and Alemu 1995), India (Agarwal et al. 1992; Gupta et al. 1975; Kalra et al. 1980; Lahiri et al. 1994; Sathy et al. 1991; Singh and Sidhu 1987; Upadhyay et al. 1989), Indonesia (Pek 1967), Kenya (Sigman et al. 1989) and Thailand (Rajatasilpin et al. 1970), with evidence of progressively increasing cognitive impairment associated with decreasing weight-for-age (Agarwal et al. 1992; Kalra et al. 1980; Lahiri et al. 1994; Sathy et al. 1991; Singh et al. 1976; Upadhyay et al. 1989). The differences in mean IQ between normal and underweight children in these correlational studies ranged between 7 and 31 points.

Among the more recent of these studies to report IQ, Agarwal et al. (1992) found Indian children aged 36 months with weight-for-age below 70% had a mean IQ approximately 8.7 points lower than adequately nourished children with weight-for-age of 80% or above; the relative risk for IQ below 80 points associated with underweight status (weight-for-age <80%) was 2.33 (95% CI 1.46–3.74). Upadhyay et al. (1989) reported a difference of 7.2 points between children below 75% and those above 90%. Lahiri et al. (1994) observed a significant trend toward lower IQ scores with decreasing weight-for-age (chi-square = 6.78, $P < 0.05$) among children aged 3–6 years from low socioeconomic levels in rural India, but the effect was not statistically significant when adjusted for age and educational exposure.

Neuroanatomical changes observed in animal models of PEM offer a theoretical foundation for the possibility that severe early malnutrition, particularly during the period of rapid brain growth and myelination in the first two years of life, presents a permanent structural insult to

brain function, leading to irreversible intellectual impairment (Strupp and Levitsky 1995). PEM has also generated abnormalities in neurotransmitter activity and receptor number (Levitsky and Strupp 1995). However, it is widely held now that neurobiological changes, on their own, are not sufficient to explain the complicated, multifactorial relationship between undernutrition and cognition, particularly among mild-to-moderately undernourished cases (Pollitt 1987). Increasing attention has been paid to environment, social context and experiential factors as potential modifiers or confounders of the nutrition–cognition relationship. Lack of energy, impaired psychomotor function and poor social interaction may prevent a child from exploring her surroundings as fully as other children, leading to less stimulation and slower acquisition of skills (Grantham-McGregor et al. 1989a, 1989b). The poverty associated with malnutrition is an environment of scarce resources, overcrowding, poor sanitation, illiteracy, few adequate educational opportunities and high morbidity, lacking in constructive forms of cognitive stimulation. Households tend to have few toys or books, social contacts are limited, and parents may offer less care and attention because of poor health, low income, low intelligence and education levels or large family sizes (Grantham-McGregor 1995). Returning to such an environment following severe malnutrition in infancy, for example, may explain persisting cognitive deficits observed in follow-up studies of infant malnutrition. Saco-Pollitt et al. (1985) offered a *cumulative deficit hypothesis* that suggests cognitive deficits increase as children are continuously exposed to environments that fail to meet their physiological, emotional and educational requirements (Pollitt 1987). Experimental studies have utilized interventions based on some of these neurobiological and psychosocial concepts.

Intervention studies to prevent or remedy impairments have focused on two general types of treatment: nutritional supplements provided to very young children and, in some cases, pregnant women; and psychosocial stimulation with or without nutritional supplementation provided to very young children. Evidence from nutritional interventions among high-risk or undernourished children has suggested early (<2 years of age) supplementary feeding improves their developmental scores, with some indication of long-term benefits. Short-term (90 days) and long-term (2–3 years) supplementation programmes in Colombia (Waber et al. 1981), Guatemala (Pollitt et al. 1993), Indonesia (Hussaini et al. 1991) and Jamaica (Grantham-McGregor et al. 1991) have improved motor development among infants. Concurrent improvements in other developmental outcomes have been less consistent, but Waber et al. (1981) in Colombia observed significantly higher scores in personal–social, speech and language, and performance subscales in addition to locomotor and eye–hand coordination subscales among infants supplemented until 3 years of age. Long-term follow-up identified improvements in certain achievement-related abilities, but not basic cog-

nitive skills by 6 years of age (Gorman et al. 1995). In India, Elizabeth and Sathy (1997) reported larger gains in developmental quotient and higher overall IQ (+2.6 points) among malnourished infants by the end of a two-year nutritional management and supplementation intervention. In Guatemala, children exposed to prenatal and early postnatal supplementation demonstrated long-term cognitive benefits, performing significantly better at ages 13–19 years on general intelligence tests and achievement-related subtests of numeracy, general knowledge, reading and vocabulary, even after adjusting for socioeconomic factors and educational experience; some information-processing tasks showed improvement as well (Pollitt et al. 1995). In Indonesia, infants who began 90 days of supplementation before 18 weeks of age demonstrated a positive effect on memory skills at 8 years of age, but differences in other cognitive processes were not significant (Pollitt et al. 1997).

Intervention programmes based on early psychosocial stimulation have improved developmental scores among moderately to severely malnourished infants and preschool-age children. Various studies have employed educational day care programmes (McKay et al. 1978), clinic-based therapy and education (Elizabeth and Sathy 1997) and weekly home visits with mothers and infants (Grantham-McGregor et al. 1994; Waber et al. 1981). In Jamaica, demonstrating play techniques to mothers and severely malnourished infants at home over three years was associated with both immediate and long-term improvements in global intelligence and development scores relative to a non-intervened control group (Grantham-McGregor et al. 1994). At 14 years follow up, mean scores on WISC for the intervened group were 8.6 points higher than the non-intervened group, but both intervened and non-intervened groups still performed significantly worse than an adequately nourished, generally higher socioeconomic status control group (9.7 and 18.3 IQ points below, respectively). Similar results were observed in a facility-based study in India among malnourished infants provided nutritional management with or without cognitive stimulation (Elizabeth and Sathy 1997). At the end of two years, both interventions were associated with significant improvements in developmental quotients, with the final IQ of stimulated infants 8.3 points higher than those receiving nutritional management alone, but both treatment groups ultimately performed below an adequately nourished/higher mean socioeconomic status control group by 5.4 to 13.7 IQ points. In a community-based component of the study, stimulated infants achieved IQ scores 6.5 points higher than an untreated, comparable control group and 3.8 points higher than infants receiving nutritional management alone; it is too early to know long-term effects in this case.

In Colombia, McKay et al. (1978) assigned underweight children aged 3 years to varying durations of an integrated health, nutrition and education programme and found that earlier initiation and longer duration of intervention were associated with higher general cognitive scores.

However, by school age, the intervened groups continued to score below an adequately nourished, higher socioeconomic status control group, and the absence of a comparable malnourished/low socioeconomic status control group made the absolute effect of the intervention unclear. Waber et al. (1981) evaluated cognitive development of nutritionally at-risk Colombian infants according to their exposure to prenatal and postnatal nutritional supplementation and a maternal-and-child educational programme. Nutritional supplementation was associated with higher scores on tests of primarily motor skills, and maternal–child education was associated with better language performance.

In a randomized controlled trial among stunted infants in Jamaica, Grantham-McGregor et al. (1991, 1997) assessed the immediate and long-term developmental effects of nutritional supplementation with or without psychosocial stimulation. By the end of the two-year intervention, supplementation and stimulation each had a significant benefit on developmental quotients (by 7 and 8 points, respectively), with the combination of treatments having an additive effect. Follow-up at ages 7–8 years (Grantham-McGregor et al. 1997) and 11–12 years (Walker et al. 2000), indicated persistent benefits in overall IQ, vocabulary and reasoning ability associated with the early psychosocial stimulation, but less of an effect from nutritional supplementation. Supplementation alone was associated with a benefit of 4.2 IQ points (not statistically significant), stimulation alone was associated with 6.3 points, and both treatments combined were associated with 6.1 points relative to a control group of untreated, stunted children; all three groups performed below a non-stunted, untreated control group.

Unfortunately, few studies (and no individually randomized controlled trials) assessed the cognitive benefit of sustained nutritional supplementation among school-age children in developing countries. In Guatemala, a year of supplementation had no consistent effect on children aged 5–7 years (Pollitt et al. 1993). A two-year school-based supplementation programme in India observed marginal but significant effects on IQ scores and Piagetian tasks, but treatment-related differences in school attendance may have influenced the results (Agarwal et al. 1989). Several studies have examined the short-term effects of breakfast programmes (Chandler et al. 1995) or breakfast omission (Lopez et al. 1993; Simeon and Grantham-McGregor 1989) on cognitive performance among schoolchildren. In Jamaica, controlled breakfast omission was associated with impaired performances in short-term memory, idea generation and problem-solving ability among stunted or previously malnourished children, but not among adequately nourished children (Simeon and Grantham-McGregor 1989). A subsequent randomized controlled trial found that only the undernourished children responded to breakfast supplementation, showing improvement in idea generation capacity but not memory or problem-solving skills (Chandler et al. 1995). In Chile, controlled breakfast omission had no significant effect on short-term visual

memory, problem-solving capacity, or attention, regardless of pre-existing nutritional status (Lopez et al. 1993).

As with other health outcomes, the close relationship between poverty and malnutrition raises the possibility of confounding. While many of the correlational and matched case–control studies attempted to control or adjust for potential socioeconomic and environmental differences, those variables were frequently defined in broad terms and may have overlooked subtler, as-yet-unidentified, non-nutritional determinants of cognitive function. Educational status could have played a role in some findings, as well; for example, initial school enrolment may be delayed among stunted children because they appear or act younger than their chronological age (Brown and Pollitt 1996). Undernutrition could influence cognition indirectly through higher morbidity; illness could reduce activity and social interaction, or contribute to higher school absentee and drop-out rates (Mendez and Adair 1999; Neumann et al. 1991; Pollitt 1983).

Another important consideration in estimating the effect of low weight-for-age on cognitive function is possible confounding by deficiencies of iron and other micronutrients (Pollitt 1995). Iron deficiency has been associated with developmental delays (Lozoff et al. 1991; Soewondo et al. 1989; Walter 1993) and iron supplementation studies have demonstrated improvements in cognitive function among iron-deficient subjects (Pollitt 1995; Pollitt and Metallinos-Katsaras 1990; Seshadri and Gopaldas 1989). Further, studies of iron deficiency and growth have observed significant covariance between the two (Peragallo-Guarda 1984; Pollitt 1995), with evidence that iron supplementation improves both growth velocity (Chwang et al. 1988) and weight (Lawless et al. 1994) among anaemic children. Zinc deficiency may also play a role in cognitive ability (Golub et al. 1995). Several intervention studies described above (Elizabeth and Sathy 1997; Husaini et al. 1991; McKay et al. 1978; Waber et al. 1981) provided micronutrient-rich indigenous foods or supplements between experimental groups differentially and most observational studies did not control for other nutritional factors.

4.5 Risk reversibility

Risk reversibility refers to the extent to which an increased relative risk persists among a previously exposed group after that exposure is removed. There has been some investigation of the lasting effect of fetal malnutrition on immune function of children and adults in Africa (Moore et al. 1999, 2001). However, there is insufficient evidence at this time to state that past underweight status during childhood increases the mortality or morbidity risk of malaria, measles, pneumonia or diarrhoea among those no longer underweight. Therefore, the increased risk of mortality and morbidity associated with low weight-for-age is considered completely reversible. Likewise, the risk of neonatal death attribut-

able to low maternal BMI is considered completely reversible once underweight status is corrected.

5. Estimates of attributable burden

Estimates of the attributable fraction, attributable mortality and attributable burden of underweight status in the age group 0–4 years were calculated for mortality due to measles, malaria, diarrhoea, pneumonia and perinatal causes based upon the above estimates of underweight prevalence, relative risk and reversibility. Attributable fractions and attributable burdens were also calculated for morbidity due to malaria, pneumonia and diarrhoea. Because we were unable to calculate relative risks of morbidity for the proportion of children falling in the weight-for-age category of –1 SD to –2 SDs, the mortality and morbidity estimates are based on different anthropometric thresholds. The mortality estimates represent the cause-specific deaths that are attributable to weight-for-age less than –1 SD, and the morbidity estimates refer to the cause-specific episodes of illness attributable to weight-for-age less than –2 SDs. The burden estimates for mortality from perinatal causes are based on maternal pre-pregnancy BMI ≤20 kg/m^2 and infant deaths attributed to low birth weight. Results are listed in Tables 2.31 through 2.35

Table 2.31 Attributable burden of low weight-for-age on measles infection among children aged 0–4 years, by subregion

	Mortality (WA <–1 SD)			*Incidence (WA <–2 SDs)*	
Subregion	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*	*Attributable fraction (%)*	*Attributable burden DALYs (000s)*
AFR-D	45.2	94.7	3 302.1	0.0	0.0
AFR-E	44.2	64.2	2 238.0	0.0	0.0
AMR-A	0.0	0.0	0.0	0.0	0.0
AMR-B	9.3	0.0	0.0	0.0	0.0
AMR-D	24.3	0.0	0.0	0.0	0.0
EMR-B	16.5	0.0	0.6	0.0	0.0
EMR-D	39.2	26.3	915.8	0.0	0.0
EUR-A	0.0	0.0	0.0	0.0	0.0
EUR-B	15.5	0.7	25.4	0.0	0.0
EUR-C	1.5	0.0	0.0	0.0	0.0
SEAR-B	39.9	9.2	320.4	0.0	0.0
SEAR-D	53.8	59.6	2 073.9	0.0	0.0
WPR-A	5.7	0.0	0.1	0.0	0.0
WPR-B	29.3	6.5	226.0	0.0	0.0
World	44.8	261.3	9 102.1	0.0	0.0

Table 2.32 Attributable burden of low weight-for-age on malaria infection among children aged 0–4 years, by subregion

	Mortality (WA <–1 SD)			*Morbidity/incidence (WA <–2 SDs)*	
Subregion	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*	*Attributable fraction (%)*	*Attributable burden DALYs (000s)*
AFR-D	57.7	251.5	8 480.9	8.4	115.9
AFR-E	56.7	235.6	7 982.9	8.1	115.6
AMR-A	0.0	0.0	0.0	0.0	0.0
AMR-B	13.8	0.1	1.7	0.8	0.1
AMR-D	33.7	0.0	0.9	3.0	0.1
EMR-B	23.7	0.0	0.0	1.8	0.1
EMR-D	51.3	23.3	787.3	6.6	7.4
EUR-A	0.0	0.0	0.0	0.0	0.0
EUR-B	22.3	0.0	0.0	1.6	0.0
EUR-C	2.3	0.0	0.0	0.1	0.0
SEAR-B	52.0	1.7	56.0	6.8	1.8
SEAR-D	66.4	35.9	1 223.1	11.8	11.4
WPR-A	8.5	0.0	0.0	0.5	0.0
WPR-B	39.8	1.2	40.1	4.1	0.5
World	57.3	549.2	18 572.7	8.2	253.0

Table 2.33 Attributable burden of low weight-for-age on pneumonia/ALRI among children aged 0–4 years, by subregion

	Mortality (WA <–1 SD)			*Morbidity/incidence (WA <–2 SDs)*	
Subregion	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*	*Attributable fraction (%)*	*Attributable burden DALYs (000s)*
AFR-D	54.6	171.1	5 729.9	20.1	38.2
AFR-E	53.6	200.9	6 739.8	19.5	42.4
AMR-A	0.0	0.0	0.0	0.0	0.0
AMR-B	12.6	3.4	113.6	2.2	6.7
AMR-D	31.2	6.6	221.5	7.9	4.8
EMR-B	21.7	4.5	151.2	4.7	5.0
EMR-D	48.2	108.2	3 661.4	16.1	51.6
EUR-A	0.0	0.0	0.0	0.0	0.0
EUR-B	20.4	9.8	328.2	4.3	1.9
EUR-C	2.1	0.2	5.2	0.3	0.0
SEAR-B	49.0	20.5	692.7	16.5	37.7
SEAR-D	63.4	432.0	14 606.4	26.9	262.3
WPR-A	7.7	0.0	0.9	1.3	0.0
WPR-B	37.1	85.9	2 884.4	10.4	77.8
World	52.3	1 042.9	35 135.0	16.5	528.3

Table 2.34 Attributable burden of low weight-for-age on diarrhoea infection among children aged 0–4 years, by subregion

	Mortality (WA <–1 SD)			*Morbidity/incidence (WA <–2 SDs)*	
Subregion	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*	*Attributable fraction (%)*	*Attributable burden DALYs (000s)*
AFR-D	62.7	118.0	3 960.1	6.4	12.4
AFR-E	61.7	198.6	6 698.3	6.2	14.0
AMR-A	0.0	0.0	0.0	0.0	0.0
AMR-B	16.1	4.2	140.0	0.6	0.9
AMR-D	38.1	7.3	247.1	2.3	0.7
EMR-B	27.1	4.0	133.8	1.3	0.7
EMR-D	56.3	138.1	4 657.9	5.0	9.0
EUR-A	0.0	0.0	0.0	0.0	0.0
EUR-B	25.5	3.8	128.0	1.2	0.5
EUR-C	2.8	0.1	1.7	0.1	0.0
SEAR-B	57.1	15.2	507.2	5.1	4.8
SEAR-D	71.1	295.5	9 975.4	9.1	47.6
WPR-A	10.0	0.0	0.1	0.3	0.0
WPR-B	44.5	31.3	1 050.5	3.0	8.0
World	60.7	815.9	27 500.1	5.3	98.6

Table 2.35 Attributable burden of low pre-pregnancy BMI on mortality due perinatal conditions, by subregion

	Mortality (BMI ≤20 kg/m²)		
Subregion	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*
AFR-D	8.0	10.1	358.5
AFR-E	6.7	9.0	318.2
AMR-A	1.7	0.1	4.8
AMR-B	3.2	2.5	87.6
AMR-D	1.9	0.3	10.0
EMR-B	2.6	0.3	11.9
EMR-D	8.7	17.5	637.8
EUR-A	1.4	0.1	2.7
EUR-B	1.6	0.3	10.6
EUR-C	1.5	0.1	3.3
SEAR-B	3.5	1.3	46.2
SEAR-D	15.5	104.5	3 792.0
WPR-A	2.5	0.0	0.9
WPR-B	2.4	2.4	85.8
World	10.5	148.4	5 370.1

Table 2.36 Total burden of underweight status among children aged 0–4 years

	Mortality (WA <–1 SD)			*Morbidity/incidence (WA <–2 SDs)*	
Disease	*Attributable fraction (%)*	*Attributable mortality (000s)*	*Attributable burden DALYs (000s)*	*Attributable fraction (%)*	*Attributable burden DALYs (000s)*
Protein–energy malnutrition	100.0	153.6	5 252.7	100	9 632.5
Perinatal conditions[a]	10.5	148.4	4 967.0	0.0	0.0
Pneumonia/ ALRI	52.3	1 042.9	35 135.0	16.5	528.3
Diarrhoea	60.7	815.9	27 500.1	5.3	98.6
Malaria	57.3	549.2	18 572.7	8.2	253.0
Measles	44.8	261.3	9 102.1	0.0	0.0
Other	53.1	776.9	26 355.8	—	—
Total	34.7	3 748.2	126 885.4	—	10 512.2

— No data.

[a] "Perinatal conditions" include low birth weight, birth asphyxia and trauma, neonatal sepsis, maternal and placental complications, respiratory distress, fetal blood loss, fetal haematological disorders, anaemia, perinatal infections, maternal diabetes and various other conditions. The category does not include congenital anomalies, neonatal tetanus or syphilis. The burden estimates represent infant deaths due to low birth weight only.

for both sexes combined according to outcome and subregion. Table 2.36 summarizes the total burden estimates associated with underweight status, including estimates of mortality directly attributable to PEM and "other" indeterminate deaths in the residual category. The total attributable fraction of mortality among children aged <5 years due to maternal and child undernutrition was 34.7% (Table 2.36). This was calculated from the sum of the cause-specific attributable mortality estimates divided by the estimate of total deaths among children aged 0–59 months. This fraction includes deaths directly and indirectly attributable to undernutrition. In determining the fraction of deaths indirectly caused by undernutrition, the analysis differentiated postneonatal from neonatal deaths, with maternal undernutrition considered as the risk factor for neonatal deaths.

6. Discussion

The results of these analyses indicate that undernutrition in young children contributes significantly toward the global burden of disease. We

estimate that undernutrition in children aged <5 years, as reflected in underweight or low weight-for-age, causes 3 599 800 deaths, including 815 900 diarrhoea deaths, 1 042 900 pneumonia deaths, 261 300 measles deaths and 549 200 malaria deaths. The estimates also illustrate that the burden of undernutrition in children begins *in utero*, with approximately 148 400 neonatal deaths from low birth weight attributable to maternal underweight status. The 3 748 200 total deaths attributable to maternal and child undernutrition constitute 34.7% of all deaths among children aged <5 years. The loss in DALYs associated with these deaths is staggering—over 126 million. Among the principal causes of death in young children, 60.7% of deaths due to diarrhoea, 52.3% of deaths due to pneumonia, 44.8% of deaths due to measles and 57.3% of deaths due to malaria are attributable to undernutrition. These attributable fractions are large because undernutrition substantially increases a child's risk of dying from common childhood illnesses, and because undernutrition is still highly prevalent in many regions of the world. In particular, the subregions SEAR-D, AFR-D and AFR-E respectively account for 32%, 22% and 24% of the total DALYs lost from undernutrition, followed by EMR-D (12%) and WPR-B (6%).

Undernutrition contributes to the morbidity burden among children as well. Our analyses indicate that having a weight-for-age less than –2 SDs places a child at increased risk of developing pneumonia, diarrhoea or malaria. We estimate that 16.5% of pneumonia, 5.3% of diarrhoea illness and 8.2% of malarial attacks are attributable to low weight-for-age, with an associated loss of DALYs amounting to 528 300 for pneumonia, 98 600 for diarrhoea and 253 000 for malaria. Available evidence suggests that undernutrition does not influence a child's risk of contracting measles and thus none of the measles morbidity burden is attributed to this risk factor.

The relatively greater burden of death as compared to illness attributable to undernutrition is understandable, given the known synergy between illness and malnutrition. This synergy was first described by Scrimshaw et al. (1968): "The simultaneous presence of malnutrition and infection results in an interaction that is more serious for the host than would be expected from the combined effect of the two working independently". Undernutrition potentiates the risk of mortality by increasing the likelihood that the illness will be prolonged or become severe; and more prolonged or severe illness is more likely to negatively affect the nutritional status of the child, placing her at ever-increasing risk of future and more prolonged or severe illness episodes. This has been most clearly demonstrated in the literature for diarrhoeal illnesses (Black et al. 1984), and our analysis makes the extension to three other principal causes of death among young children: pneumonia, malaria and measles. Such findings underscore the need to prioritize the improvement of the nutritional status of children, including within disease control programmes (Becker et al. 1991).

In attempting to quantify the relationship between underweight status and disease, it is important to consider that undernutrition, as assessed by the underweight indicator (or a stunting indicator), is commonly associated with deficiencies of micronutrients (Bhan et al. 2001). In fact, some of these deficiencies, such as zinc, may themselves contribute to poor growth (Brown 2002). These deficiency conditions, especially of vitamin A, iron and zinc, put the child at risk of adverse outcomes, including morbidity and mortality from infectious disease and cognitive impairment. The consequences of these deficiency states can be estimated separately, as has been done in other chapters in this book. The association of underweight condition with these deficiencies means that some of the risk attributed to being underweight may be, in fact, due to specific micronutrient deficiencies. On the other hand, these other deficiencies can occur in children who are not considered underweight, so the risk of adverse outcomes is not entirely encompassed in the subset of the population who are underweight. In fact, the effect of vitamin A supplementation on reducing mortality in vitamin A-deficient populations has been found to be similar in children who were more or less well nourished at the beginning of the trial (Sommer 1986; West 1991). Likewise, zinc supplementation in populations that are presumably zinc deficient has had similar effects on reducing infectious disease morbidity in children who were classified as both "wasted" or "not wasted" (Zinc Investigators' Collaborative Group 1999). Therefore, the effects of undernutrition as identified by being underweight and those of deficiencies of vitamin A, iron and zinc cannot be simply added to determine the overall burden of disease due to nutritional risk factors, nor is it appropriate to assume that all of the consequences of micronutrient deficiencies are subsumed in the underweight calculation. Additional work is needed to determine the joint prevalence distributions of being underweight and being deficient in each of these micronutrients and to assess the joint effects of these aspects of undernutrition.

The outcomes described here represent some of the fundamental global health challenges facing populations at greatest risk for undernutrition. However, the burden of undernutrition on human health and well-being no doubt extends beyond the relatively narrow focus of this analysis. We presented findings to suggest a role for maternal malnutrition as a risk factor for maternal mortality, as well as a role for undernutrition in disability due to poor cognitive development, but the research base thus far does not allow for the calculation of burden estimates. Other considerations relating to undernutrition may include the short-term or long-term effects of underweight status on work capacity or the effect of IUGR on childhood development and morbidity. Many areas are still largely unknown or speculative, such as the role of early or ongoing undernutrition in potentiating chronic disease (Barker 1995;

Law et al. 2001; Stein et al. 1996) or the effect of undernutrition on susceptibility to infection among adult and elderly populations—questions that will only gain in importance as developing countries continue to go through demographic and epidemiological transitions.

Several reports suggest that through current programmatic efforts, rates of undernutrition among children are declining at 1% per year. In contrast, our projections indicate that over the coming decades we can expect variable changes in these prevalences. Because our analyses clearly indicate the overwhelming magnitude of the disease burden associated with child malnutrition, effective strategies to reduce child undernutrition are urgently needed. Energy supplementation for pregnant women, counselling and promotion of breastfeeding and adequate complementary food intake, and child growth monitoring are some of the more effective and affordable interventions for preventing low birth weight and improving child growth (ACC/SCN 2001; WHO 2002). Micronutrient supplementation or fortification, the use of oral rehydration therapy and childhood immunizations, have also been key elements to improving nutritional status and preventing severe childhood disease. Further innovations will be necessary in order to meet the challenge of undernutrition in all populations.

7. Projections of exposure: trends in child underweight status from 2000 to 2030

The estimates for 2010 and 2020 are directly derived from the model as outlined in section 2.2, based on estimates of underweight prevalence in individual countries at multiple points in time. To extend these model estimates to 2030, we applied the lowest delta between last available trend intervals, i.e. for AFR-D and AFR-E, we used 2010–2015 and for the other subregions the deltas 2015–2020; the respective difference was multiplied by 2 (as they refer to a five-year interval) and then subtracted from or added to the estimate of 2020 to derive the value for 2030 (Table 2.37).

Table 2.37 Estimated prevalence of underweight among children aged 0–4 years from 2000 to 2030, by subregion

	% having weight-for-age below –2 SDs			
Subregion	*2000*	*2010*	*2020*	*2030*
AFR-D	32.2	34.2	36.2	38.2
AFR-E	31.0	37.3	44.1	50.7
AMR-A	2.3	1.5	0.7	0.0[a]
AMR-B	5.0	3.3	2.2	1.1
AMR-D	12.4	9.5	7.2	5.0
EMR-B	8.1	4.3	2.3	0.5
EMR-D	25.1	20.4	16.4	12.6
EUR-A	2.3	1.5	0.7	0.0[a]
EUR-B	7.6		Overall stagnation[b]	
EUR-C	2.6		Overall declining trend[b]	
SEAR-B	25.8	18.7	13.2	8.0
SEAR-D	45.9	37.6	30.0	22.6
WPR-A	3.8	2.5	1.2	0.0[a]
WPR-B	16.0	11.8	8.6	5.6

[a] AMR-A, EUR-A and WPR-A, comprising the developed countries, are estimated to move further towards overweight and thus the underweight levels are forecasted to decrease stepwise to reach 0% by 2030. In WPR-A the estimated trend is driven by Japan (comprising 77% of the population aged 0–4 years in this subregion) and a national survey from 1978–81 already resulted with a low prevalence of underweight (3.7%). Similarly the 1996 national survey in Australia (contributing 16% of the population aged 0–4 years in this subregion) reported 0% underweight. Appendix C lists by country in alphabetical order publications backing the estimated trend towards overweight in AMR-A, EMR-A and WPR-A.

[b] There are very scarce empirical data on the nutritional status of children aged <5 years in EUR-B and EUR-C. Based on the little information available to date (see Appendix D) the estimated trends of underweight until 2030 for these two subregions are expected to stagnate and decline, respectively. These overall trend estimations for EUR-B and EUR-C, however, are to be taken with caution.

(i) EUR-B

The available data for this subregion show a diverse pattern. There are countries with decreasing rates in child underweight and parallel there are others where increase in the national prevalence of child underweight can be observed. With the information available to date it is very difficult to estimate a trend in child underweight from 2000 to 2030. The trend pattern is expected to be driven by Turkey, which has 40% of the total <5 population in this subregion. However, given the available trend data for other countries in this subregion and the unfavourable conditions (demographic and economic transition associated in parts with political instability) in some of them an overall stagnation of the underweight prevalence from 2000 to 2030 is to be expected. Appendix E(a) lists references by country in alphabetical order to document the forecasted trend in this subregion.

(ii) EUR-C

Given that any trend in this subregion is likely to be dominated by Russia (which has 56% of the total population of children aged <5 years in this subregion) and that current evidence shows raising rates in overweight in this country we would estimate an overall declining trend of underweight. However, given the enduring unstable situation in some of the countries in EUR-C and the uncertainty about support and investments from developed countries, a quantification of the expected decline cannot be made. Appendix E(b) lists references by country in alphabetical order derived from the WHO Global Database, Medline and the Internet which contain additional information to document the forecasted trend in this subregion.

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Notes

1 See preface for an explanation of this term.

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Appendix A

National surveys included in prevalence estimates

Country	*Author(s)*[a]	*Reference*
Afghanistan		(1998) *Afghanistan 1997 multiple indicator baseline (MICS). Report to UNICEF*. Centro de Investigacion de Enfermedades Tropicales (CIET), Acapulco.[b]
Algeria	Kellou K	(1987) Etat nutritionnel des enfants algériens de 0 à 10 ans et niveaux d'urbanisation d'après les résultats préliminaires de l'enquête épidémiologique sur la malnutrition protéino-énérgétique en 1987. Institut National de Santé Publique, Algiers.
Algeria	République Algérienne Démocratique et Populaire	(1992) *Enquête algérienne sur la santé de la mère et de l'enfant*. Office national des statistiques. (PAPCHILD Surveys.) The League of Arab States, Cairo.
Algeria	Ministère de la Santé et de la Population	(1996) *Enquête nationale sur les objectifs de la mi-décennie, "MDG Algérie", 1995*. Algiers.[b]
Angola		(1999) *Inquerito de indicadores multiplos (MICS) 1996*. Instituto Nacional de Estatistica, Gabinete de Monitorização das Condições de Vida da População. Luanda.[b]
Argentina	Lejarraga H, Krupitzky S, Gimenez E et al.	(1997) The organisation of a national survey for evaluating child psychomotor development in Argentina. *Paediatric and Perinatal Epidemiology*, **11**:359–373.[b]
Armenia		(1998) *The health and nutritional status of children and women in Armenia*. National Institute of Nutrition, Italy.[b]
Azerbaijan	Branca F, Burkholder B, Hamel M, Parvanta I, Robertson A	(1996) *Health and nutrition survey of internally displaced and resident population of Azerbaijan—April 1996*. Baku.[b]
Bahrain	Ministry of Health	(1992) *Bahrain child health survey 1989*. Manama.
Bangladesh	Helen Keller International, Institute of Public Health	(1985) *Bangladesh nutritional blindness study, 1982–83: nutritional findings*. Dhaka.[b]
Bangladesh	Government of the People's Republic of Bangladesh	(1987) *Report of the child nutrition status module, Bangladesh household expenditure survey 1985–86*. Bangladesh Bureau of Statistics, Dhaka.

Bangladesh	Government of the People's Republic of Bangladesh	(1991) *Report of the child nutrition status survey 1989–90*. Bangladesh Bureau of Statistics, Dhaka.
Bangladesh	Ministry of Planning	(1994) *Child nutrition survey of Bangladesh 1992*. Bangladesh Bureau of Statistics, Dhaka.
Bangladesh		(1997) *Bangladesh demographic and health survey 1996–97*. (Demographic and Health Surveys.) National Institute for Population Research and Training, Dhaka.[b]
Barbados		(1986) *National nutrition survey of Barbados, 1981*. Caribbean Food and Nutrition Institute, Jamaica.[b]
Belize	Ministry of Health	(1992) *Assessment of the food, nutrition and health situation of Belize*. (INCAP Publication DC1/002.) Institute of Nutrition of Central America and Panama, Kingston.
Benin	Kodjogbé N, Mboup G, Tossou J et al.	(1997) *Enquête démographique et de santé 1996*. (Demographic and Health Surveys.) Ministère du Plan, de la Restructuration Economique et de la Promotion de l'Emploi, Cotonou.
Bhutan		(1989) *Bhutan Directorate of Health Services. Report on the national nutrition survey*. Bhutan.
Bhutan	Ministry of Health and Education	(1999) *National anthropometric survey of under five children in Bhutan*. Division of Health Services, Thimphu.[b]
Bolivia	Government of Bolivia	(1982) *Bolivia national nutritional status survey, 1981: summary report*. National Institute for Food and Nutrition, La Paz.
Bolivia	Ministerio de Planeamiento y Coordinacion	(1990) *Encuesta nacional de demografia y salud 1989*. (Demographic and Health Surveys.) La Paz.[b]
Bolivia	Ministerio de Planeamiento y Coordinacion	(1992) *Situacion alimentaria y nutricional de Bolivia 1992*. Instituto Nacional de Alimentacion y Nutricion, La Paz.
Bolivia	Ministerio de Desarrollo Sostenible y Medio Ambiente	(1994) *Encuesta nacional de demografia y salud 1994*. (Demographic and Health Surveys.) La Paz.[b]
Bolivia	Ministerio de Desarrollo Humano	(1994) *Bolivia: mapa de la desnutricion 1990–1992*. La Paz.
Bolivia	Gutierrez Sardan M	(1997) *Encuesta nacional de multiples indicadores 1996 (MICS)*. Ministerio de Desarrollo Humano, La Paz.[b]

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National surveys included in prevalence estimates (*continued*)

Country	*Author(s)*[a]	*Reference*
Bolivia	Ministerio de Haciendo, Instituto Nacional de Estadistica	(1998) *Encuesta nacional de demografia y salud 1998*. (Demographic and Health Surveys.) La Paz.[b]
Botswana		(1999) *The 1996 Botswana family health survey III*. Central Statistics Office, Gaborone.
Brazil	Monteiro CA, Benicio MH, Gouveia NC	(1991) *Growth and nutritional status of the Brazilian children: findings from the 1989 National Health and Nutrition Survey*. Country Studies on Nutritional Anthropometry (NUT/ANTREF/1/91.) World Health Organization, Geneva.[b]
Brazil	Monteiro CA, Benicio MH, Iunes R, Gouveia NC, Taddei JA, Cardoso MA	(1992) Nutritional status of Brazilian children: trends from 1975 to 1989. *Bulletin of the World Health Organization*, **70**:657–666.[b]
Brazil	Ministry of Health	(1996) *Pesquisa nacional sobre demografia e saùde 1996 (relatorio preliminar)*. (Demographic and Health Surveys.) Rio de Janeiro.[b]
Burkina Faso	Konaté DL, Sinaré T, Seroussi M	(1994) *Enquête démographique et de santé, Burkina Faso 1993*. (Demographic and Health Surveys.) Ouagadougou.[b]
Burundi	Segamba L, Ndikumasabo V, Makinson C, Ayad M	(1988) *Enquête démographique et de la santé au Burundi, 1987*. (Demographic and Health Surveys.) Gsitega.[b]
Cambodia	Ministry of Planning	(1997) *Socioeconomic survey of Cambodia 1996. Volume 1. Summary results*. National Institute of Statistics, Phnom Penh.[b]
Cameroon	The Government of Cameroon	(1978) *United Republic of Cameroon national nutrition survey*. USAID, Washington, DC.
Cameroon	Balépa M, Fotso M, Barrère B	(1992) *Enquête démographique et de santé Cameroun, 1991*. (Demographic and Health Surveys.) Yaoundé.[b]
Cameroon	Fotso M, Ndonou R, Libité PR et al.	(1999) *Enquête démographique et de santé, Cameroun 1998 (DHS)*. Bureau Central des Recensements et des Etudes de Population, Ministère des Investissements Publics et de l'Aménagement du Territoire, Yaoundé.[b]
Cape Verde	Reitmaier P, Dupret A, Cutting WAM	(1987) Better health data with a portable microcomputer at the periphery: an anthropometric survey in Cape Verde. *Bulletin of the World Health Organization*, **65**:651–657.[b]

Cape Verde	Wennberg A	(1988) Anthropometric assessment of the nutritional status of preschool children in Cape Verde. *Bulletin of the World Health Organization*, **66**:375–386.
Cape Verde	Ferreira Medina JB, Skard T, Sobhy S, America Ungaretti M	(1996) *A saúde das crianças menores de cinco anos em Cabo Verde*. Ministério de Saúde e Promoção Social and UNICEF, Cape Verde.
Central African Republic	Ministère de la Santé Publique et de la Population	(1995) *Etat nutritionnel de la population. Rapport préliminaire de l'enquête de nutrition mai–juillet 1995*. Bangui.
Central African Republic	Ndamobissi R, Mboup G, Nguélébé EO	(1995) *Enquête démographique et de santé, République Centrafricaine 1994–95*. (Demographic and Health Surveys.) Bangui.[b]
Chad		(1998) *Enquête démographique et de santé, Tchad 1996–97*. (Demographic and Health Surveys.) Bureau Central du Recensement, Direction de la Statistique, des Etudes Economiques et Démographiques, N'Djamena.[b]
Chile	Ministerio de Salud	(1986) *Estado nutricional de la poblacion en control de salud. Sistema de vigilancia alimentaria y nutricional*. Departamento de Control y Evaluacion, Santiago.
Chile	Avila B, Garcia F, Vera G	(1988) *Situacion alimentaria nutricional de Chile, periodo 1984–87*. Universidad de Chile, Santiago.
Chile	Ministerio de Salud, SISVAN	(1987) *Estado nutricional de la poblacion infantil, 1986*. Santiago.
Chile	Monckeberg F, Valienta S, Mardones F	(1987) Infant and pre-school nutrition: economical development versus intervention strategies—the case of Chile. *Nutrition Research*, **7**:327–342.
Chile	Ministerio de Salud	(1994) *National health service system*. Nutrition Unit, Santiago.
Chile	Castillo CL, Atalah E, Castro R	(1996) Alimentacion del menor de 18 meses: relacion con el estado nutricional. *Revista Chilena de Pediatria*, **67**:22–28.
Chile	Ministerio de Salud	(1995) *National health service system*. Santiago.
Chile	Ministerio de Salud	(1997) *National health service system*. Santiago.
Chile	Ministerio de Salud	(1999) *Boletin anual de vigilancia nutricional, año 1998*. Departamento Coordinacion e Informatica, Santiago.
China	Ge K	(1995) *The dietary and nutritional status of Chinese population (1992 national nutrition survey)*. Institute of Nutrition and Food Hygiene, Beijing.[b]

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
Colombia	Mora JO	(1982) *Situacion nutricional de la poblacion Colombiana en 1977–80. Volumen I: Resultados antropometricos y de laboratorio; Comparacion con 1965/66*, Ministerio de Salud, Instituto Nacional de Salud, Bogota.
Colombia	Mora JO, de Paredes B, de Navarro L, Rodriguez E	(1992) Consistent improvement in the nutritional status of Colombian children between 1965 and 1989. *Bulletin of PAHO*, **26**:1–13.
Colombia	Ministerio de Salud	(1988) *Tercera encuesta nacional de prevalencia del uso de anticonceptivos y primera de demografia y salud, 1986.* (Demographic and Health Surveys.) Institute for Resource Development, Bogota.[b]
Colombia		(1995) *Encuesta nacional de demografia y salud 1995*. (Demographic and Health Surveys.) Bogota.[b]
Comoros	Ministère de la Santé Publique et de la Population	(1995) *Rapport sur l'état nutritionnel et les facteurs impliqués chez les enfants de moins de deux ans en République Fédérale Islamique des Comores 1991*. Direction de la Santé Familiale, Comoros.
Comoros	Mondoha KA, Schoemaker J, Barrère M	(1997) *Enquête démographique et de santé, Comores 1996*. (Demographic and Health Surveys.) Centre National de Documentation et de Recherche Scientifique, Moroni.
Congo	Cornu A, Delpeuch F, Simondon F et al.	(1990) *Enquête nationale sur l'état nutritionnel des enfants d'age préscolaire au Congo*. (Collection Etudes et Thèses.) ORSTOM, Institut Francais de Recherche Scientifique pour le Développement en Coopération, Paris.
Costa Rica	Ministerio de Salud	(1982) *Encuesta nacional de nutrition 1982*. Departamento de Nutricion, San José.
Costa Rica	Ministerio de Salud	(1994) *Analisis del estado nutricional de la poblacion Costarricense 1992*. Departamento de Nutricion y Atencion Integral, Seccion Vigilancia Nutricional, San José.
Costa Rica	Ministerio de Salud	(1996) *Estado nutricional de preescolares atendidos por el programa de atencion primaria*. Departamento de Nutricion, Seccion de Vigilancia Nutricional, San José.
Costa Rica	Ministerio de Salud	(1996) *Encuesta nacional de nutricion: I fasciculo antropometria*. San José.
Côte d'Ivoire	Sahn DE	(1990) *Malnutrition in Côte d'Ivoire, prevalence and determinants*. (Working Paper No. 4.) World Bank, Washington, DC.[b]
Côte d'Ivoire	Sombo N'Cho, Kouassi L, Kouamé Koffi A et al.	(1995) *Enquête démographique et de santé, Côte d'Ivoire 1994*. (Demographic and Health Surveys.) Abidjan.[b]

Democratic Republic of the Congo	Ministère du Plan et Reconstruction Nationale	(1996) *Enquête nationale sur la situation des enfants et des femmes au Zaire en 1995*. Kinshasa.
Djibouti	Ministère de la Santé Publique et des Affaires Sociales	(1990) *Enquête couverture vaccinale malnutrition. République de Djibouti*. Djibouti.
Djibouti		(1997) *Enquête djiboutienne auprès des ménages indicateurs sociaux (EDAM-IS 1996)*. Ministère du Commerce et du Tourisme, Direction Nationale de la Statistique, Djibouti.
Dominican Republic		(1987) *Encuesta demografica y de salud 1986*. (Demographic and Health Surveys.) Consejo Nacional de Poblacion y Familia, Santo Domingo.[b]
Dominican Republic		(1992) *Encuesta demografica y de salud 1991*. (Demographic and Health Surveys.) Santo Domingo.[b]
Dominican Republic		(1997) *Encuesta demografica y de salud 1996*. (Demographic and Health Surveys.) Centro de Estudios Sociales y Demograficos, Asociacion Dominicana Pro Bienestar de la Familia, Oficina Nacional de Planificacion, Santo Domingo.[b]
Ecuador	Freire W, Dirren H, Mora J et al.	(1988) *Diagnostico de la situacion alimentaria, nutricional y de salud de la poblacion ecuatoriana menor de cinco años*. Ministerio de Salud Publica y Consejo Nacional de Desarrollo, Quito.
Egypt	Ministry of Health[a]	(1978) *National nutrition survey, 1978*. Cairo.[b]
Egypt	Abdel-Sayed H, Osman M, El-Zanaty F, Way A	(1989) *Egypt demographic and health survey 1988*. (Demographic and Health Surveys.) Egypt National Population Council, Cairo.[b]
Egypt		(1992) *Egyptian maternal and child health survey*. (PAPCHILD Surveys.) Agency for Public Mobilisation and Statistics, Cairo.[b]
Egypt	El-Zanaty FH, Sayed HAA, Zaky HHM, Way AA	(1993) *Egypt demographic and health survey 1992*. (Demographic and Health Surveys.) Cairo.[b]
Egypt		(1995) *National survey for assessment of vitamin "A" status in Egypt*. Nutrition Institute, Cairo.
Egypt	El-Zanaty F, Hussein EM, Shawsky GA, Way AA, Kishor S	(1996) *Egypt demographic and health survey 1995*. (Demographic and Health Surveys.) National Population Council, Cairo.[b]

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
Egypt		(1998) *Egypt demographic and health survey 1997*. (Demographic and Health Surveys.) El-Zanaty and Associates, Cairo.
El Salvador	Trowbridge FL	(1984) *Center for Disease Control Survey (CDC): Data on El Salvador, 1975*. Atlanta, GA.
El Salvador		(1990) *Evaluacion de la situacion alimentaria nutricional en El Salvador (ESANES-88)*. Ministerio de Salud Publica y Asistencia Social, San Salvador.
El Salvador	Salvadoran Demographic Association	(1994) *National family health survey 1993 (FESAL-93)*. Government of El Salvador, San Salvador.[b]
Eritrea	Ministry of Finance and Development	(1994) *Children and women in Eritrea: situation analysis, 1994*. Government of the State of Eritrea and UNICEF, Asmara.
Eritrea		(1997) *Eritrea demographic and health survey 1995*. (Demographic and Health Surveys.) Asmara.
Ethiopia		(1993) *Report on the national rural nutrition survey, core module*. (Statistical Bulletin No 113.) Transitional Government of Ethiopia, Central Statistical Authority, Addis Ababa.
Fiji		(1995) *1993 National nutrition survey—main report*. National Food and Nutrition Committee, Suva.
Gambia		(1997) *Report of the progress of the mid-decade goals in the Gambia (MICS), 1996 (draft)*. Central Statistics Department, Banjul.
Ghana	Alderman H	(1989) *Nutritional status in Ghana and its determinants*. (Working Paper No. 3.) World Bank, Washington, DC. (And additional analysis on the Living Standards Survey, Ghana 1987–88.)
Ghana		(1989) *Ghana demographic and health survey 1988*. (Demographic and Health Surveys.) Ghana Statistical Service, Accra.[b]
Ghana		(1994) *Ghana demographic and health survey 1993*. (Demographic and Health Surveys.) Ghana Statistical Service, Accra.[b]
Ghana		(1999) *Ghana demographic and health survey 1998*. (Demographic and Health Surveys.) Ghana Statistical Service, Accra.[b]
Guatemala	Delgado H, Hidalgo E, Giron EM, Pareja G	(1989) *Encuesta nacional de salud materno infantil 1987*. (Demographic and Health Surveys.) Ministerio de Salud Publica y Asistencia Social, Guatemala.[b]

Guatemala		(1996) *Encuesta nacional de salud materno infantil 1995*. (Demographic and Health Surveys.) Guatemala City.[b]
Guatemala		(1999) *Encuesta nacional de salud materno infantil 1998–1999*. (Demographic and Health Surveys.) Guatemala City.[b]
Guyana	Government of Guyana	(1979) *The national food and nutrition survey of Guyana 1971*. (Scientific Publication No. 323.) Georgetown.
Guyana	Caribbean Food and Nutrition Institute	(1981) *Guyana nutritional status survey 1981*. Caribbean Food and Nutrition Institute, Kingston.[b]
Guyana	Guyana Bureau of Statistics	(1994) Household income and expenditure survey 1993 and Guyana living standards measurement survey. In: *Guyana strategies for reducing poverty*. (Report No. 12861-GUA, pp. 50–67) World Bank, Washington, DC.
Haiti	Graitcer PL, Gedeon MA, de Beausset I, Duckett EM	(1980) Haiti nutrition status survey, 1978. *Bulletin of the World Health Organization*, **58**:757–765.[b]
Haiti	Ministry of Public Health and Population	(1993) *Haiti's nutrition situation in 1990: A report based on anthropometric data of the 1990 nutrition surveys*. Port-au-Prince.
Haiti	Cayemittes M, Rival A, Barrère B, Lerebours G, Gédéon MA	(1995) *Enquête mortalité, morbidité et utilisation des services (EMMUS-II) Haiti 1994/95*. (Demographic and Health Surveys.) Pétionville.[b]
Honduras	Barahona F, Soto RJ, Tronconi E, Maradiaga A, O'Connor G, Corrales G	(1988) *Encuesta nacional de nutricion, Honduras, 1987. Cuadros de frequencias por regiones de salud y nacionales*. Ministerio de Salud Publica, Tegucigalpa.[b]
Honduras	Ministerio de Salud Publica	(1993) *Encuesta nacional de epidemiologia y salud familiar (ENESF), 1991/92*. Tegucigalpa.[b]
Honduras		(1996) *National survey of socioeconomic indicators 1993/94*. Tegucigalpa.[b]
Honduras	Ministry of Health	(1997) *National micronutrient survey Honduras 1996*. Tegucigalpa.[b]
India	National Institute of Nutrition	(1993) *National Nutrition Monitoring Bureau, 1991–92 (8 States pooled data)*. Hyderabad.[b]
India	National Institute of Nutrition	(1993) *National Nutrition Monitoring Bureau, 1974–79 (8 States pooled data)*. Hyderabad.[b]
India	National Institute of Nutrition	(1993) *National Nutrition Monitoring Bureau, 1988–90 (8 States pooled data)*. Hyderabad.[b]

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
India	Vijayaraghavan K, Hanumantha Rao D	(1998) Diet and nutrition situation in rural India. *Indian Journal of Medical Research*, **108**:243–253.[b]
India	International Institute for Population Sciences	(1995) *National family health survey, India 1992–93*. (Demographic and Health Surveys.) Bombay.[b]
Indonesia		(1992) *National socioeconomic survey 1987 (SUSENAS-1987)*. Central Bureau of Statistics, Jakarta.[b]
Indonesia		(1997) *Indonesia multiple indicator cluster survey (MICS) 1995*. UNICEF, Jakarta. (Preliminary results provided by the Centers for Disease Control and Prevention.)
Iran (Islamic Republic of)	Undersecretary for Public Affairs, Ministry of Health and Medical Education	(1996) *Cluster survey for evaluation of mid decade goal indicators (MICS)*. Teheran.[b]
Iran (Islamic Republic of)		(2000) *The nutritional status of children, October–November 1998 (ANIS)*. Ministry of Health and Medical Education and UNICEF, Teheran.
Iraq	International Study Team	(1992) *Infant and child mortality and nutritional status of Iraqi children after the Gulf conflict: results of a community-based study*. Center for Population and Development Studies, Harvard University, Cambridge, MA.
Jamaica	Ministry of Health and Environmental Control	(1978) *Unpublished data*. The Nutrition Unit, Kingston.[b]
Jamaica		(1990) *Jamaica living standards and measurement survey 1989*. World Bank, Kingston.[b]
Jamaica		(1992) *Jamaica survey of living conditions—report 1991*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jamaica		(1994) *Jamaica survey of living conditions 1992*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jamaica		(1995) *Jamaica survey of living conditions 1993*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jamaica		(1996) *Jamaica survey of living conditions, 1994*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jamaica		(1997) *Jamaica survey of living conditions, 1995*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jamaica		(1997) *Jamaica survey of living conditions, 1996*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]

Jamaica		(1998) *Jamaica survey of living conditions, 1997*. The Planning Institute and the Statistical Institute of Jamaica, Kingston.[b]
Jordan	Zou'bi A, Poedjastoeti S, Ayad M	(1992) *Jordan population and family health survey 1990*. (Demographic and Health Surveys.) Ministry of Health, Amman.[b]
Jordan		(1998) *Jordan population and family health survey 1997*. (Demographic and Health Surveys.) Department of Statistics, Amman.[b]
Kazakhstan	National Institute of Nutrition	(1996) *Kazakhstan demographic and health survey 1995*. (Demographic and Health Surveys.) Almaty.[b]
Kenya		(1991) *Fourth rural child nutrition survey, 1987*. Central Bureau of Statistics, Ministry of Planning and National Development, Nairobi.
Kenya		(1994) *Kenya demographic and health survey 1993*. (Demographic and Health Surveys.) Central Bureau of Statistics, Nairobi.[b]
Kenya	Central Bureau of Statistics	(1995) *Fifth child nutrition survey, 1994. Welfare monitoring survey*. Nairobi.
Kenya	National Council for Population and Development	(1999) *Kenya demographic and health survey 1998*. (Demographic and Health Surveys.) Central Bureau of Statistics, Nairobi.
Kiribati	Ministry of Health and Family Planning	(1990) *National nutrition survey, 1985* (draft version). Government of Kiribati, South Tarawa.
Kuwait	Bayoumi A, Moussa MAA	(1985) Kuwait nutritional survey: comparison of the nutritional status of Kuwaiti children, 0–5 years with the NCHS/CDC reference. *World Health Organization Bulletin,* **63**:521–526.
Kuwait	Amine EK, Al-Awadi FA	(1996) Nutritional status survey of preschool children in Kuwait. *Eastern Mediterranean Health Journal*, **2**:386–394.
Kyrgyzstan		(1998) *Kyrgyz Republic demographic and health survey 1997*. (Demographic and Health Surveys.) Bishkek City.[b]
Lao People's Democratic Republic		(1995) *Diagnostic de la situation nutritionnelle et consommation alimentaire au Laos. Rapport complet de l'étude sur l'état nutritionnel de la population laotienne*. (ESNA: TCP/LAO/2354.) Food and Agriculture Organization of the United Nations, Rome.
Lao People's Democratic Republic	Ministry of Public Health	(1994) *Women and children in the Lao People's Democratic Republic. Results from the Lao social indicator survey (LSIS)*. Mother and Child Institute, Vientiane.

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National surveys included in prevalence estimates (*continued*)

Country	*Author(s)*[a]	*Reference*
Lebanon		(1996) *Lebanon maternal and child health survey (preliminary report)*. (PAPCHILD Surveys.) The League of Arab States, Cairo.
Lesotho		(1977) *Lesotho national nutrition survey*. The Government of Lesotho, Maseru.
Lesotho	Ministries of Health and Agriculture	(1992) *National nutrition survey report, May–June 1992*. Maseru.
Lesotho	Ministry of Health	(1994) *National survey on iodine, vitamin A and iron status of women and children in Lesotho*. Maseru.
Lesotho	Spring CA	(1996) *Mid-decade goals: progress towards the world summit, May 1996 (MICS)*. Bureau of Statistics and UNICEF, Maseru.
Liberia	Ministry of Health and Social Welfare	(1978) *Liberia national nutrition survey 1975–1976*. Monrovia.
Libyan Arab Jamahiriya		(1997) *Libyan maternal and child health survey*. (PAPCHILD Surveys.) The League of Arab States, Cairo.
Madagascar	Adrianasolo R	(1986) *Etude des aspects épidémiologiques de l'allaitement maternel à Madagascar*. Laboratoire Central de Nutrition MINSAN, Antananarivo.
Madagascar	Refeno G, Rabeza V, Mboup G, Schoemaker J	(1994) *Enquête nationale démographique et sanitaire 1992*. (Demographic and Health Surveys.) Centre National de Recherches sur l'Environnement, Antananarivo.[b]
Madagascar	Institut National de la Statistique	(1995) *Enquête permanente auprès des ménages—rapport principal, décembre 1995*. Antananarivo.
Madagascar	Institut National de la Statistique	(1996) *Enquête par grappes à indicateurs multiples (résultats préliminaires). Multiple Indicators Cluster Survey*. Antananarivo.
Madagascar		(1998) *Enquête démographique et de santé, Madagascar 1997*. (Demographic and Health Surveys.) Institut National de la Statistique, Antananarivo.[b]
Malawi	Malawi Government	(1984) *National sample survey of Agriculture 1980/81. Vol. III*. National Statistics Office, Zomba.
Malawi		(1993) *Malawi demographic and health survey 1992*. (Demographic and Health Surveys.) National Statistics Office, Zomba.[b]

Malawi	Ministry of Economic Planning and Development	(1996) *Malawi social indicators survey 1995. MICS surveys.* National Statistical Office and the Centre for Social Research, Lilongwe.
Malaysia	Ministry of Health	(1994) *Annual reports, 1990–1993. Family Health Information System*. Information and Documentation Unit, Kuala Lumpur.
Malaysia	Ministry of Health	(1996) *Annual reports, 1994 and 1995. Family Health Information System*. Division of Family Health Development, Kuala Lumpur.
Maldives	Ministry of Health and Welfare	(1994) *Nutritional status and child feeding practices of Maldivian children*. Department of Public Health, Male.
Maldives		(1996) *Maldives multiple indicator survey report (MICS).* United Nations Children's Fund, Male.[b]
Maldives	Ministry of Planning and National Development, United Nations Development Programme	(1999) *Vulnerability and poverty assessment 1998*. Male.[b]
Mali	Traoré B, Konaté M, Stanton C	(1989) *Enquête démographique et de santé au Mali, 1987*. (Demographic and Health Surveys.) Bamako.[b]
Mali	Coulibaly S, Dicko F, Traoré SM, Sidibé O, Seroussi M, Barrère B	(1996) *Enquête démographique et de santé Mali 1995–1996*. (Demographic and Health Surveys.) Cellule de Planification et de Statistique, Bamako.[b]
Mali		(1996) *Enquête à indicateurs multiples au Mali (EIM) 1996 (MICS). Rapport d'analyse*. Direction Nationale de la Statistique et de l'Informatique (DNSI), Bamako.
Mauritania	Elder JA	(1990) *The socioeconomic determinants of nutritional status among children under five in Mauritania.* (Social dimensions of adjustment surveys.) World Bank, Washington, DC.
Mauritania	Ministry of Planning	(1992) *Mauritania maternal and child health survey 1990–91*. (PAPCHILD surveys.) Nouakchott.[b]
Mauritania	Ministère du Plan, Direction des Ressources Humaines	(1996) *Enquête nationale sur les indicateurs des objectifs à mi-terme en Mauritanie (MICS).* Nouakchott.
Mauritius	Ministry of Health	(1988) *Mauritius national nutrition survey 1985: summary report.* Evaluation and Nutrition Unit, Port Louis.
Mauritius	Ministry of Health	(1996) *A survey on nutrition in Mauritius and Rodrigues, 1995 (final report)*. Port Louis.

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
Mexico		(1990) *Encuesta nacional de alimentacion en el medio rural, 1974*. Instituto de la Nutricion 'Salvador Zubiran', Comision Nacional de Alimentacion, Division de Nutricion, INNSZ, Tlalpan, Mexico City.
Mexico		(1990) *Encuesta nacional de alimentacion en el medio rural, 1979*. Instituto de la Nutricion 'Salvador Zubiran', Comision Nacional de Alimentacion, Division de Nutricion, INNSZ, Tlalpan, Mexico City.
Mexico	Sepulveda AJ, Lezana MA, Tapia Conyer R, Valdespino IL, Madrigal H, Kumate J	(1990) Estado nutricional de preescolares y las mujeres en Mexico: resultados de una encuesta probabilistica nacional. *Gaceta Medica de Mexico*, **126**:207–226.[b]
Mexico		(1990) *Encuesta nacional de alimentacion en el medio rural, 1989 (ENAL)*. Instituto de la Nutricion 'Salvador Zubiran', Comision Nacional de Alimentacion, Division de Nutricion, INNSZ, Tlalpan, Mexico City.[b]
Mongolia	Kachondham Y	(1992) *Report of a consultancy on the Mongolian child nutrition survey*. Institute of Nutrition, Nakornpathom.
Mongolia	Kachondham Y	(2000) *Report on the 2nd national child and nutrition survey, Mongolia 1999*. Institute of Nutrition and Faculty of Medicine, Ramathibodi Hospital, Mahidol University.[b]
Morocco	Azelmat M, Ayad M, Belhachmi H	(1989) *Enquête nationale sur la planification familiale, la fécondité et la santé de la population au Maroc (ENPS) 1987*. (Demographic and Health Surveys.) Ministère de la Santé Publique, Rabat.[b]
Morocco	Azelmat M, Ayad M, Housni El A	(1993) *Enquête nationale sur la population et la santé (ENPS-II) 1992*. (Demographic and Health Surveys.) Rabat.[b]
Mozambique	Government of Mozambique, UNICEF	(1996) *Multiple indicator cluster survey Mozambique—1995. MICS surveys*. Ministry of Planning and Finance, Maputo.
Mozambique		(1998) *Moçambique inquérito demográfico e de saúde 1997*. (Demographic and Health Surveys.) Instituto Nacional de Estatística, Maputo.[b]
Myanmar	Daw Cho Nwe Oo	(1981) *Feeding practices in infants and young children in Rangoon Division. 1980–1981*. Rangoon.
Myanmar	Daw Cho Nwe Oo	(1986) *Feeding practices in young children and infants*. Department of Medical Research, Rangoon.
Myanmar	Ministry of Health	(1991) *Nutrition situation of Myanmar children. Preliminary report of the national nutrition survey 1990*. Rangoon.

Myanmar	Ministry of Health	(1994) *Nutrition situation of Myanmar children. Report of the national nutrition nurvey 1991*. Rangoon.
Myanmar	Department of Health	(1995) *National nutrition survey, 1994*. National Nutrition Centre, Yangon.
Myanmar	Ministry of Health	(1995) *Monitoring progress toward the goals of the World Summit for Children through multiple indicator cluster survey (MICS)*. Yangon.
Myanmar	Ministry of Health	(2000) *National nutrition survey 1997*. National Nutrition Centre, Yangon.[b]
Namibia	Katjiuanjo P, Titus S, Zauana M, Boerma T	(1993) *Namibia demographic and health survey 1992*. (Demographic and Health Surveys.) Windhoek.[b]
Nepal	His Majesty's Government of Nepal	(1975) *Nepal nutrition status survey, January–May 1975*. Kathmandu.[b]
Nepal	National Planning Commission	(1996) *Nepal multiple indicator surveillance: cycle I, January–March 1995 health and nutrition—final report (MICS)*. Kathmandu.[b]
Nepal	Ajit Pradhan, Ram Hari Aryal, Gokarna Regmi, Bharat Baan, Pavalavalli Govindasamy	(1997) *Nepal family health survey, 1996*. (Demographic and Health Surveys.) Ministry of Health, Kathmandu.[b]
Nicaragua	Ministerio de Salud	(1988) *Enfoque de riesgo y estado nutricional de los ninos menores de 5 anos en la region III, 1988*. Centro de investigaciones y estudios de la salud, Managua.
Nicaragua	Ministry of Health	(1982) *Unpublished data*. Managua.
Nicaragua		(1997) *Nicaragua 1993 living standards measurement survey (LSMS)*. World Bank, Washington, DC.[b]
Nicaragua	Ministerio de Salud	(1999) *Encuesta nicaraguense de demografia y salud 1998*. (Demographic and Health Surveys.) Instituto Nacional de Estadisticas y Censos, Managua.[b]
Niger	Ministère de la Santé Publique et des Affaires Sociales	(1985) *Enquête nationale sur la morbidité et la mortalité, rapport No 1*. Cellule de Planification, Niamey.
Niger	Kourguéni IA, Garba B, Barrère B	(1993) *Enquête démographique et de santé, Niger 1992*. (Demographic and Health Surveys.) Niamey.[b]

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
Niger	Attama S, Seroussi M, Kourguéni AI, Koché H, Barrère B	(1998) *Enquête démographique et de santé, Niger 1998*. (Demographic and Health Surveys.) Care International, Niger and Macro International Inc., Calverton, MD.[b]
Nigeria		(1992) *Nigerian demographic and health survey 1990*. (Demographic and Health Surveys.) Federal Office of Statistics, Lagos.[b]
Nigeria	Federal Ministry of Health and Social Services	(1996) *National micronutrient survey, 1993*. Ibadan.[b]
Oman	Musaiger OA (On behalf of the Ministry of Health)	(1993) *National nutrition survey of the Sultanate of Oman*. On behalf of the Ministry of Health. UNICEF, Muscat.[b]
Oman	Ministry of Health	(1995) *National study on the prevalence of vitamin A deficiency (VAD) among children 6 months to 7 years*. Muscat.[b]
Pakistan		(1992) *Pakistan demographic and health survey 1990/91*. (Demographic and Health Surveys.) National Institute of Population Studies, Islamabad.[b]
Pakistan	Government of Pakistan	(1978) *Micronutrient survey of Pakistan*. Nutrition Cell, Planning and Development Division, Islamabad.[b]
Pakistan	Government of Pakistan	(1988) *National nutrition survey 1985–87 report*. National Institute of Health, Nutrition Division, Islamabad.[b]
Pakistan	Ministry of Health	(1996) *Multiple indicator cluster survey of Pakistan, 1995. MICS surveys*. Government of Pakistan, Islamabad.
Pakistan	Pakistan Medical Research Council	(1998) *National health survey of Pakistan (NHSP, 1990–94): Health profile of the people of Pakistan*. Islamabad.
Panama	Franklin DL, Harrell M, Tamaro J, Frazao B, Vial I, Parillon C	(1982) *Nutrition evaluation project: second annual report*. Research Triangle Institute, North Carolina.
Panama	Ministerio de Salud	(1992) *Encuesta nacional de Vitamina A, 1992*. Departamento de Nutricion y Dietetica, Panama.
Papua New Guinea	Smith T, Keig G, Marks J, Grau R	(1992) *Summary results by environmental zone from the 1982/83 National Nutrition Survey of Papua New Guinea*. Papua New Guinea Institute of Medical Research, Goroka.[b]

Papua New Guinea	Jenkins C, Zemel B	(1990) *Ancient diversity and contemporary change in the growth patterns of Papua New Guinea children*. (59th Annual Meeting of the American Association of Physical Anthropologists.) Miami, FL.
Paraguay	Carron JM, Loaiza E, Ochoa LH	(1991) *Encuesta nacional de demografia y salud 1990*. (Demographic and Health Surveys.) Asuncion.[b]
Peru	Ministerio de Salud	(1988) *Situacion nutricional en el Peru. Encuesta nacional de nutricion y salud (ENSSA), 1984*; and *Evaluacion nacional de nutricion por antropometria (ENPPE), 1975*. Ministerio de Salud, Lima.
Peru	Padilla A, Ochoa LH, Marckwardt AM	(1992) *Encuesta demografica y de salud familiar 1991/1992*. (Demographic and Health Surveys.) Lima.[b]
Peru		(1997) *Peru demographic and health survey 1996*. (Demographic and Health Surveys.) Instituto Nacional de Estadistica e Informacion, Lima.[b]
Philippines		(1975) *1971–75 unpublished data*. Department of Physiological Hygiene and Nutrition, Institute of Public Health, Manila.
Philippines	Food and Nutrition Research Institute	(1982) *Second nationwide nutrition survey, Philippines, 1982*. National Science and Technology Authority, Manila.
Philippines	National Economics and Statistics Section	(1991) *Regional updating of nutritional status of Filipino children, 1989–90*. Food and Nutrition Research Institute, Manila.[b]
Philippines	Department of Science and Technology	(1991) *Third national nutrition survey Philippines, 1987*. Food and Nutrition Research Institute. Manila.[b]
Philippines	Department of Science and Technology	(1994) *The 1992 regional nutrition survey*. Food and Nutrition Research Institute, Manila.[b]
Philippines	Department of Science and Technology	(1995) *The fourth national nutrition survey: Philippines 1993*. Food and Nutrition Institute, Manila.[b]
Qatar	Amine EK	(1996) *Nutritional assessment in Qatar*. (Assignment report EM/NUT169/E/R/01.96/27.) World Health Organization Regional Office, Alexandria.
Rwanda	Meheus A, Butera S, Dindinian O, Eylenbosch W	(1977) *Evaluation de l'état nutritionnel des enfants de 0 à 5 ans dans la République Rwandaise, 1976*. Université Instelling, Antwerpen.[b]

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National surveys included in prevalence estimates (*continued*)

Country	*Author(s)*[a]	*Reference*
Rwanda	Barrère B, Schoemaker J, Barrère M, Habiyakare T, Kabagwira A, Ngendakumana M	(1994) *Enquête démographique et de santé, Rwanda 1992*. (Demographic and Health Surveys.) Kigali.[b]
Rwanda	Ministère de la Santé	(1997) *National nutrition survey of women and children in Rwanda in 1996*. Kigali.
Saint Lucia	The Government of St Lucia	(1976) *The national food and nutrition survey of St. Lucia, 1974*. The Caribbean Food and Nutrition Institute, Kingston.[b]
Sao Tome and Principe	Ministerio de Saude	(1986) *Estado nutricional e cobertura vacinal des crianças menoras de 5 anos na*. Secçao de Nutriçao. Sao Tome.
Senegal	Ndiaye S, Sarr I, Ayad M	(1987) *Enquête démographique et de santé au Sénégal, 1986*. (Demographic and Health Surveys.) Dakar.[b]
Senegal	Senegal Bureau of Statistics	(1993) *Social dimensions of adjustment. Household priority survey 1991–92*. World Bank, New York.
Senegal	Ndiaye S, Papa Demba Diouf, Ayad M	(1994) *Enquête démographique et de santé au Sénégal (EDS-II) 1992/93*. (Demographic and Health Surveys.) Dakar.[b]
Senegal		(1996) *Evaluation des objectifs intermédiaires (MICS)*. UNICEF, Dakar.
Seychelles	Ministry of Health	(1989) *Nutritional status of Seychellois children* (unpublished data). Victoria.
Sierra Leone	Ministry of Health	(1975) *WHO global epidemiological surveys*. World Health Organization, Health Situation Unit, Geneva.
Sierra Leone	Government of Sierra Leone, USAID	(1978) *Sierra Leone national nutrition survey 1977–78*. USAID, Washington, DC.
Sierra Leone	Ministry of Health	(1990) *The Republic of Sierra Leone national nutrition survey*. Freetown.
Solomon Islands	Solomon Islands Government	(1970) *National nutrition survey* (unpublished data). South Pacific Health Service, Honiara.
Solomon Islands	Ministry of Health and Medical Services	(1990) *Solomon Islands national nutrition survey 1989*. Honiara.[b]
South Africa	The South African Vitamin A Consultative Group	(1995) *Children aged 6 to 71 months in South Africa, 1994: their anthropometric, vitamin A, iron and immunisation coverage status*. Johannesburg.[b]

Sri Lanka	Ministry of Health	(1976) *Sri Lanka nutrition status survey, 1976*. Colombo.[b]
Sri Lanka	Ministry of Plan Implementation	(1987) *Sri Lanka demographic and health survey 1987*. (Demographic and Health Surveys.) Colombo.[b]
Sri Lanka	Ministry of Plan Implementation	(1979) *National nutritional survey 1977–78 (resurvey)*. Colombo.[b]
Sri Lanka	Department of Census and Statistics	(1995) *Sri Lanka demographic and health survey 1993*. (Demographic and Health Surveys.) Colombo.
Sri Lanka	Ramanujam P, Nestel P	(1997) Preliminary report on the fourth national nutrition and health survey July–August, 1995. *The Ceylon Journal of Medical Science*, **40**:13–24.
Sudan		(1994) *Sudanese maternal and child health survey, 1993*. (PAPCHILD surveys.) The League of Arab States, Cairo.
Swaziland	Serdula MK, Aphane JM, Kuene PF et al.	(1987) Acute and chronic undernutrition in Swaziland. *Journal of Tropical Pediatrics*, **33**:35–42.[b]
Syrian Arab Republic		(1994) *Syrian maternal and child health survey*. (PAPCHILD surveys.) The League of Arab States, Cairo.
Syrian Arab Republic	Prime Minister's Council	(1996) *Multiple indicator cluster survey in the Syrian Arab Republic (MICS)*. Central Bureau of Statistics, Damascus.
Thailand	Chayovan N, Kamnuansilpa P, Knodel J	(1988) *Thailand demographic and health survey 1987*. (Demographic and Health Surveys.) Institute of Population Studies, Chulalongkorn University, Bangkok.[b]
Thailand	Kitvorapat W, Chaotilittakul N, Sinawat S, Wanaratana L	(1996) Random survey on nutritional status of children of ages under five. *Thailand Journal of Health Promotion and Environmental Health*, **19**:57–66.[b]
Togo	Togo Ministry of Rural Development	(1978) *Togo nutrition status survey, 1977*. Lomé.[b]
Togo	Agounké A, Assogba M, Anipah K	(1989) *Enquête démographique et de santé au Togo 1988*. (Demographic and Health Surveys.) Direction Générale de la Santé, Lomé.[b]
Togo	Kotokou K, Anipah K, Jondoh C	(1996) *Enquête nationale sur la situation des enfants au Togo en 1995 (MICS) (version préliminaire)*. Ministère du Plan et de l'Aménagement du Territoire, Lomé.

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National surveys included in prevalence estimates (*continued*)

Country	*Author(s)*[a]	*Reference*
Togo	Anipah K, Mboup G, Ouro-Gnao AM, Boukpessi B, Messan PA, Salami-Odjo R	(1999) *Enquête démographique et de santé, Togo 1998*. (Demographic and Health Surveys.) Ministère de la Planification et du Développement Economique, Direction de la Statistique, Lomé.[b]
Trinidad and Tobago	Gueri M, Andrews N, Jutsum P, Rawlins R	(1980) Nutritional status of young children in Trinidad and Tobago. *Journal of Tropical Pediatrics,* **26**:11–15.[b]
Trinidad and Tobago	Heath K, Da Costa-Martinez D, Sheon AR	(1988) *Trinidad and Tobago demographic and health survey 1987*. (Demographic and Health Surveys.) Family Planning Association of Trinidad and Tobago, Port-of-Spain.[b]
Tunisia	Forbes AL, Pelletier O, Lowenstein FW, Lane M, Keller W	(1976) *Preliminary report of the 1973–1975 Tunisian national nutrition survey*. Tunisian National Institute of Nutrition and Food Technology, Tunis.[b]
Tunisia	Aloui T, Ayad M, Fourati H	(1989) *Enquête démographique et de santé en Tunisie 1988*. (Demographic and Health Surveys.) Office National de la Famille et de la Population, Tunis.[b]
Tunisia	Ministère de la Santé Publique, Institut National de Nutrition et de Technologie Alimentaire	(1998) *Enquête nationale 1996–1997. Evaluation de l'état nutritionnel de la population Tunisienne: Rapport national.* Sotepa Grafic, Tunis.[b]
Turkey		(1994) *Turkey demographic and health survey, 1993*. (Demographic and Health Surveys.) Ankara.[b]
Turkey	Ministry of Health	(1996) *Multiple indicator cluster survey in Turkey 1995*. National Bureau of Statistics, Ankara.
Turkey		(1999) *Turkish demographic and health survey 1998*. (Demographic and Health Surveys.) Hacettepe University, Institute of Population Studies, Ankara.[b]
Uganda	Kaijuka EM, Kaija EZA, Cross AR, Loaiza E	(1989) *Uganda demographic and health survey 1988/89*. (Demographic and Health Surveys.) Ministry of Health, Entebbe.[b]
Uganda		(1996) *Uganda demographic and health survey 1995*. (Demographic and Health Surveys.) Entebbe.[b]

United Republic of Tanzania	Ngallaba S, Kapiga SH, Ruyobya I, Boerma JT	(1992) *Tanzania demographic and health survey 1991/92*. (Demographic and Health Surveys.) Dar es Salaam.[b]
United Republic of Tanzania	Bureau of Statistics	(1997) *Tanzania demographic and health survey 1996*. (Demographic and Health Surveys.) Dar es Salaam.[b]
Uruguay	Bove MI	(1987) *Uruguay: Situacion alimentario-nutricional, algunos factores condicionantes 1970–87*. (Document prepared for the ACC/SCN.) Montevideo.
Uruguay	Ministerio de Salud Publica	(1996) *Sistema de vigilancia epidemiologica nutricional (SISVEN)*. Departamento de nutricion, Montevideo
Uzbekistan	Ministry of Health	(1997) *Uzbekistan demographic and health survey 1996*. (Demographic and Health Surveys.) Institute of Obstetrics and Gynecology, Tashkent City.[b]
Vanuatu	Hung MM	(1983) *National nutrition survey report*. Department of Health, Port Vila.
Venezuela	Instituto Nacional de Nutricion	(1986) *Encuesta nacional de nutricion 1981–82*. Caracas.
Venezuela		(1995) *Proyecto Venezuela 1987*. Centro de estudios sobre crecimiento y desarrollo de la poblacion venezolana, Caracas.[b]
Venezuela	Oficina SISVAN	(1996) *Resultados de la evalucion antropometrica del componente menores de 5 años del Sistema de Vigilancia Alimentaria y Nutricional (SISVAN) segun los puntos de corte de la OMS: Venezuela 1990–1993*. Instituto Nacional de Nutricion, Caracas.
Venezuela	Oficina SISVAN	(1996) *Clasificacion antropometrica nutricional, Venezuela 1994. INN-SISVAN componente menores de 15 años*. Instituto Nacional de Nutricion, Caracas.
Venezuela	Oficina SISVAN	(1998) *Evaluacion antropometrica nutricional de los menores de cinco años, para comparacion internacional: Venezuela 1995–1997*. Instituto Nacional de Nutricion, Caracas.
Venezuela	Oficina SISVAN	(1999) *Evaluacion antropometrica nutricional de los menores de cinco años, para comparacion internacional. Venezuela 1990–1998*. Instituto Nacional de Nutricion, Caracas.
Viet Nam	Tu Giay, Ha Huy Khoi, Luong Tan Thanh	(1986) Some characteristics about protein energy nutritional status in Vietnamese children. In: *Applied nutrition, proceedings of the international conference on applied nutrition, Hanoi 25/26 April 1986*:201–208.
Viet Nam	Ministry of Health	(1991) *Report on reanalysed data collected by the General Nutrition Survey 1987–89*. Department of Planning, Hanoi.
Viet Nam	Bloem MW, Gorstein J	(1995) *Viet Nam: Xerophthalmia free; 1994 national Vitamin A deficiency and protein-energy malnutrition prevalence survey. Consultancy report 5–17 March 1995*. National Institute of Nutrition, Hanoi.

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NATIONAL SURVEYS INCLUDED IN PREVALENCE ESTIMATES (*continued*)

Country	*Author(s)*[a]	*Reference*
Viet Nam		(1998) *Viet Nam living standards survey 1992–93 (VNLSS)*. World Bank, Washington, DC.[b]
Viet Nam	Dibley MJ, Khoi HH, Khan NC et al.	(1999) *National protein energy malnutrition survey, Viet Nam 1998*. National Institute of Nutrition, Hanoi; and Centre for Clinical Epidemiology and Biostatistics, Newcastle, Australia.[b]
Viet Nam	Khoi HH, Khan NC, Tuyen LD, Ngu T, Xuan TT	(2000) *1999 Viet Nam—child nutrition situation. The national goal for child malnutrition control*. Medical Publishing House, Hanoi.[b]
Yemen	Ministry of Health	(1992) *Yemen maternal and child health survey*. (PAPCHILD Surveys.) Sana'a.[b]
Yemen		(1996) *Yemen multiple indicator cluster survey (March 1996): Final results*. Ministry of Planning and Development, Sana'a.[b]
Yemen		(1998) *Yemen democratic and maternal and child health survey 1997*. (Demographic and Health Surveys.) Central Statistical Organization, Sana'a.
Zambia	Wenlock RW	(1980) Nutritional risk and the family environment in Zambia. *Ecology of Food and Nutrition*, **10**:79–86.[b]
Zambia	Gaisie K, Cross AR, Nsemukila G	(1993) *Zambia demographic and health survey 1992*. (Demographic and Health Surveys.) Central Statistical Office, Lusaka.[b]
Zambia	Cogill B, Zaza M	(1990) *Report of the nutrition module as part of the crop forecasting survey—Rural Zambia 1990*. Ministry of Health, Lusaka.
Zambia		(1997) *Zambia demographic and health survey 1996*. (Demographic and Health Surveys.) Central Statistical Office, Lusaka.[b]
Zimbabwe	Ministry of Health	(1987) *Report of the nutrition component of the national health information system*. Harare.
Zimbabwe	Ministry of Finance, Economic Planning and Development	(1989) *Zimbabwe demographic and health survey, 1988*. (Demographic and Health Surveys.) Harare.[b]
Zimbabwe		(1995) *Zimbabwe demographic and health survey 1994*. (Demographic and Health Surveys.) Harare.[b]

[a] Where no authors are listed, documents have been written by multiple authors such as organizations, institutions and governments.

[b] Survey data have been reanalysed either by responsible national authorities or by WHO.

Appendix B

Numbers of countries and population coverage of children aged <5 years for underweight, by subregion

Subregion	*No. of countries/total*	*% population aged <5 years covered*
AFR-D	23/26	99.1
AFR-E	20/20	100
AMR-A	1/3	87.9
AMR-B	19/26	99.8
AMR-D	6/6	100
EMR-B	10/13	81.6
EMR-D	8/9	96.7
EUR-A	2/26	20.5
EUR-B	5/16	58.9
EUR-C	3/9	69.8
SEAR-B	3/3	100
SEAR-D	6/7	98.4
WPR-A	2/5	92.3
WPR-B	13/22	97.4

Appendix C

Trends in underweight status; references for AMR-A, EUR-A, and WPR-A

Country	*Author(s)*	*Reference*
Australia	Margarey AM, Daniels LA, Boulton TJ	(2001) Prevalence of overweight and obesity in Australian children and adolescents: reassessment of 1985 and 1995 data against new standard international definitions. *Medical Journal of Australia*, **174**:561–564.
Australia	Booth ML, Wake M, Amstrong T, Chey T, Hesketh K, Mathur S	(2001) The epidemiology of overweight and obesity among Australian children and adolescents, 1995–97. *Australian and New Zealand Journal of Public Health*, **25**:162–169.
Canada	Hanley AJG, Harris SB, Gittelsohn J, Wolever TMS, Saksvig, Zinman B	(2000) Overweight among children and adolescents in a native Canadian community: prevalence and associated factors. *American Journal of Clinical Nutrition*, **71**:693–700.
France	Deheeger M, Rolland-Cachera MF, Labadie MD, Rossignol C	(1994) Etude longitudinale de la croissance et de l'alimentation d'enfants examinés de l'âge de 10 mois à 8 ans. *Cahiers de Nutrition et de Diététique*, **XXIX**:16–23
France	Lehingue Y, Miginiac M, Locard E, Mamelle N	(1993) Birth weight and obesity at the age of 6. Study from the growth curves of a population of schoolchildren. *Pediatrie*, **48**:623–632.
Germany	Schaefer F, Georgi M, Wuhl E, Scharer K	(1998) Body mass index and percentage fat mass in healthy German school children and adolescents. *International Journal of Obesity Related Disorders*, **22**:461–469.
Germany	Kromeyer-Hauschild K, Zellner K, Jaeger U, Hoyer H	(1999) Prevalence of overweight and obesity among school children in Jena (Germany). *International Journal of Obesity Related Disorders*, **23**:1143–1150.
Greece	Mamalakis G, Kafatos A, Manios Y, Anagnosto-poulou T, Apostolaki I,	(2000) Obesity indices in a cohort of primary school children in Crete: a six year prospective study. *International Journal of Obesity & Related Metabolic Disorders*, **24**:765–771.
Japan	Mitsunori Murata	(2000) Secular trends in growth and changes in eating patterns of Japanese children. *American Journal of Clinical Nutrition*, **72**:S1379–1383.
Netherlands	Cole TJ, Roede MJ	(1999) Centiles of body mass index for Dutch children aged 0–20 years in 1980—a baseline to assess recent trends in obesity. *Annals of Human Biology*, **26**:303–308.
Netherlands	Fredriks AM, van Buuren S, Burgmeijer RJF et al.	(2000) Continuing positive secular growth change in the Netherlands 1955–1997. *Pediatric Research*, **47**:316–323.

Netherlands	Hirasing RA, Fredriks AM, van Buuren S, Verloove-Vanhorick SP, Wit JM	(2001) Increased prevalence of overweight and obesity in Dutch children and the detection of overweight and obesity using international criteria and new reference diagrams. *Nederlands Tijdschrift voor Geneeskunde*, **145**:1303–1308.
Spain	Proyecto Universitario	(1984) *Paidos'84, estudio epidemiologico sobre nutricion y obesidad infantil.* Graficas Jomagar, Mostoles, Madrid.
Spain	Moreno LA, Sarria A, Fleta J, Rodriguez G, Bueno M	(2000) Trends in body mass index and overweight prevalence among children and adolescents in the region of Aragon (Spain) from 1985 to 1995. *International Journal of Obesity and Related Metabolic Disorders*, **24**:925–931.
Spain	Rios M, Fluiters E, Perez Mendez LF, Garcia-Mayor EG, Garcia-Mayor RV	(1999) Prevalence of childhood overweight in northwestern Spain: a comparative study of two periods with a ten year interval. *International Journal of Obesity and Related Metabolic Disorders*, **23**:1095–1098.
Spain	Moreno LA, Sarria A, Fleta J, Rodriguez G, Gonzalez JM, Bueno M	(2001) Sociodemographic factors and trends on overweight prevalence in children and adolescents in Aragon (Spain) from 1985 to 1995. *Journal of Clinical Epidemiology*, **54**:921–927.
United Kingdom	Chinn S, Hughes JM, Rona RJ	(1998) Trends in growth and obesity in ethnic groups in Britain. *Archives of Disease in Childhood*, **78**:513–517.
United Kingdom	Reilly JJ, Dorosty AR.	(1999) Epidemic of obesity in UK children. *The Lancet*, **354**:1874–1875.
United Kingdom	Reilly JJ, Dorosty AR, Emmett PM	(1999) Prevalence of overweight and obesity in British children: cohort study. *British Medical Journal,* **319**:1039.
United Kingdom	Chinn S, Rona RJ	(2001) Prevalence and trends in overweight and obesity in three cross-sectional studies of British children, 1974–94. *British Medical Journal*, **322**:24–26.
United Kingdom	Bundred P, Kitchiner D, Buchan I	(2001) Prevalence of overweight and obese children between 1989 and 1998: population based series of cross-sectional studies. *British Medical Journal*, **322**:313–314.
United Kingdom	Rudolf MC, Sahota P, Barth JH, Walker J.	(2001) Increasing prevalence of obesity in primary school children: cohort study. *British Medical Journal*, **322**:1094–1095.
USA	Overpeck MD, Hediger ML, Ruan WJ et al.	(2000) Stature, weight, and body mass among young US children born at term with appropriate birth weights. *Journal of Pediatrics*, **137**:205–213.
USA	Park MK, Menard SW, Schoolfield J	(2001) Prevalences of overweight in a triethnic pediatric population of San Antonio, Texas. *International Journal of Obesity and Related Metabolic Disorders*, **25**:409–416.
USA	Mei Z, Scanlon KS, Grummer-Strawn LM, Freedman DS, Yip R, Trowbridge FL	(1998) Increasing prevalence of overweight among US low-income preschool children: the Centers for Disease Control and Prevention pediatric nutrition surveillance, 1983 to 1995. *Pediatrics*, **101**:E12.

Appendix D

National survey data on trends of underweight (<–2 SD weight-for-age) in children aged <5 years in EUR-B and EUR-C countries

Country	*Year of survey*	*% <–2 SD weight-for-age*	*Overall trend*[a]	*pp/yr*[b]	*Pop. estimate*[c]
EUR-B					
Albania	1996–1998, 2000[d]	8.1, 14.3[d]	↑	2.07	309
Armenia	1998	3.3			207
Azerbaijan	1996, 2000[d]	10.1, 16.8[d]	↑	1.68	609
Bosnia and Herzegovina	2000[d]	4.1[d]			205
Bulgaria	—	—			317
Georgia	1999	3.1			299
Kyrgyzstan	1997	11.0			524
Poland	—	—			1 994
Romania	1991	5.7			1 137
Slovakia	—	—			289
Tajikistan	—	—			773
The former Yugoslav Republic of Macedonia	1999	5.9			145
Turkey	1993, 1995, 1998	10.4, 10.3, 8.3	↓	–0.42	7 108
Turkmenistan	—	—			602
Uzbekistan	1996	18.8			2 761
Former Yugoslavia	1996, 2000	1.6, 1.9	↔	–0.08	640
EUR-C					
Belarus	—	—			468
Estonia	—	—			61
Hungary	1980–1988	2.2			490
Kazakhstan	1995, 1999	8.3, 4.2	↓	–1.03	1 273
Latvia	—	—			93
Lithuania	—	—			186
Republic of Moldova	—	—			258
Russian Federation	1993, 1995	4.2, 3.0	↓	–0.60	6 362
Ukraine	2000[d]	3.0[d]			2 190

Key: ↑, Rising: ≥0.30 percentage points per year; ↔, Static: <0.30 or >–0.30 percentage points per year; ↓, Falling; ≥–0.30 percentage points per year.

— No data.

[a] For countries with no arrow, a trend could not be established.

[b] Percentage point change per year calculated by dividing the difference between the earliest and the last data points by the number of years between the two surveys. Trends are classified as rising, static or falling according to the cut-offs listed above.

[c] *World population prospects: the 2000 revision*; estimates refer to total number (000s) of children aged <5 years in 2000, sexes combined.

[d] MICS end-decade draft results.

Appendix E

Trends in underweight status; references for EUR-B and EUR-C

(a) References for EUR-B

Country	*Author(s)*[a]	*Reference*
Georgia		(2000) *Georgia multiple indicator cluster survey 1999 (MICS)*. Tibilisi.[b]
Hungary	Nemeth A, Eiben OG	(1997) Secular growth changes in Budapest in the 20th century. *Acta Medica Auxologica*, **29**:5–12.

[a] Where no authors are listed, documents have been written by multiple authors such as organizations, institutions and governments.

[b] Survey data have been reanalysed either by responsible national authorities or by WHO.

(b) References for EUR-C

Country	*Author(s)*	*Reference*
Russian Federation	Popkin BM, Richards MK, Monteiro CA	(1996) Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *Journal of Nutition*, **126**:3009–3016.
Russian Federation	Popkin BM et al.	Russia longitudinal monitoring survey, http://www.cpc.unc.edu/projects/rlms/
Russian Federation	Khandy MV	(1997) Dynamics of growth and development of rural children in the Republic of Sakha (Yakutia) over a 70 year period. *Gigiena i Sanitariia*, **4**:30–31.
Russian Federation	Beliaev EN, Chiburaev VI, Ivanov AA, Plantonova AG, Markelova SV	(2000) Characteristics of actual nutrition and health of children in regions of the Russian Federation. *Voprosy Pitaniia*, **69**:3–7.