

# Stunting, Wasting, and Micronutrient Deficiency Disorders



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Undernutrition and micronutrient deficiencies contribute substantially to the global burden of disease (Ezzati and others 2002). Impoverished communities experience high rates of undernutrition and increased exposure to infectious diseases caused by crowding and inadequate sanitation. Women of reproductive age and children experience devastating health consequences as a result of limited resources, cultural influences, and biological vulnerabilities. Undernutrition and infectious diseases exist in a baleful synergy: undernutrition reduces immunological capacity to defend against diseases, and diseases deplete and deprive the body of essential nutrients. Undernutrition and infectious diseases further exacerbate poverty through lost wages, increased health care costs, and—most insidiously—impaired intellectual development that can significantly reduce earning potential. Health experts have recently recognized the long-term effects of early undernutrition and inadequate infant feeding for obesity and chronic diseases, including diabetes and cardiovascular diseases. This chapter summarizes the problems of undernutrition and vitamin A, iron, zinc, and iodine deficiencies in young children and current programmatic efforts to prevent and treat them.

## NATURE, CAUSES, AND BURDEN OF UNDERNUTRITION

The following section describes the magnitude, distribution, and etiology of growth faltering and specific micronutrient deficiencies in young children.

## Growth Faltering

Because nutritional inputs are necessary for children's growth, undernutrition is generally characterized by comparing the weights or heights (or lengths) of children at a specific age and sex with the distribution of observed weights or heights in a reference population of presumed healthy children of the same age and sex and then calculating *z*-scores, that is, the difference between a child's weight or height and the median value at that age and sex in the reference population, divided by the standard deviation (SD) of the reference population. A child whose height-for-age is less than  $-2$  SD is considered stunted, because the chances of the child's height being normal are less than 3 percent. A child whose weight-for-age is less than  $-2$  SD is considered underweight, and one whose weight-for-height is less than  $-2$  SD is deemed wasted. *Stunting* results from chronic undernutrition, which retards linear growth, whereas *wasting* results from inadequate nutrition over a shorter period, and *underweight* encompasses both stunting and wasting. Typically, growth faltering begins at about six months of age, as children transition to foods that are often inadequate in quantity and quality, and increased exposure to the environment increases their likelihood of illness.

Although knowledge about the prevalence of stunting and wasting is preferred, information about underweight is more available globally. The high correlation between stunting and underweight and the low prevalence of wasting mean that the prevalence of underweight directly describes the magnitude of the problem of growth faltering and stunting in young children. About 130 million children under the age of five are

underweight, with the highest prevalences in South Asia and Sub-Saharan Africa (table 28.1). The prevalence of stunting, underweight, and wasting is decreasing in most areas of the world; however, in most of Africa, stunting is increasing.

Childhood malnutrition diminishes adult intellectual ability and work capacity, causing economic hardships for individuals and their families. Malnourished women tend to deliver premature or small babies who are more likely to die or suffer from suboptimal growth and development (Allen and Gillespie 2001). Poor early nutrition leads to poor school readiness and performance, resulting in fewer years of schooling, reduced productivity, and earlier childbearing. Thus, poverty, undernutrition, and ill-health are passed on from generation to generation. Undernutrition impedes economic progress in all developing countries.

Undernutrition raises the likelihood that a child will become sick and will then die from the disease. Morbidity and mortality are highest among those most severely malnourished; however, given the high prevalence of mild to moderate underweight, the mildly or moderately underweight individuals experience

the greatest total burden of disease (Fishman and others 2004). Children whose weight-for-age is less than  $-1$  SD are also at increased risk of death, and undernutrition is responsible for 44 to 60 percent of the mortality caused by measles, malaria, pneumonia, and diarrhea. Overall, eliminating malnutrition would prevent 53 percent of deaths in young children, with most of those deaths occurring in South Asia and Sub-Saharan Africa (table 28.2).

Morbidity attributable to undernutrition depends on the nature of the illness. Susceptibility to a highly infectious disease such as measles is unlikely to be affected by nutritional status: all individuals are equally likely to become infected if they are unvaccinated and naive. However, 5 to 16 percent of pneumonia, diarrhea, and malaria morbidity is attributable to moderate to severe underweight (Fishman and others 2004). As table 28.3 shows, the number of disability-adjusted life years (DALYs) attributable to undernutrition is high and, as with mortality, is concentrated in South Asia and Sub-Saharan Africa. The tremendous costs associated with the care and treatment of childhood diseases that could be partially

**Table 28.1** Estimated Prevalence of Selected Nutritional Deficiencies in Children Ages Birth through Four, by Region (percent)

| Region                          | Weight-for-age less than $-2$ SD | Weight-for-age $-2$ SD through less than $-1$ SD | Vitamin A deficiency | Iron deficiency anemia | Zinc deficiency |
|---------------------------------|----------------------------------|--|----------------------|------------------------|-----------------|
| East Asia and the Pacific       | 18                               | 29   | 11                   | 40                     | 7               |
| Eastern Europe and Central Asia | 6                                | 21   | <1                   | 22                     | 10              |
| Latin America and the Caribbean | 6                                | 23   | 15                   | 46                     | 33              |
| Middle East and North Africa    | 21                               | 35   | 18                   | 63                     | 46              |
| South Asia                      | 46                               | 44   | 40                   | 76                     | 79              |
| Sub-Saharan Africa              | 32                               | 38   | 32                   | 60                     | 50              |
| High-income countries           | 2                                | 14   | 0                    | 7                      | 5               |

Sources: Underweight: Fishman and others 2004; vitamin A: Rice, West, and Black 2004; iron: Stoltzfus, Mullany, and Black 2004; zinc: Caulfield and Black 2004.

**Table 28.2** Estimated Deaths of Children Ages Birth through Four Attributable to Selected Nutritional Deficiencies by Region (thousands)

| Region                          | Weight-for-age less than $-1$ SD <sup>a</sup> | Vitamin A deficiency | Iron deficiency anemia <sup>b</sup> | Zinc deficiency |
|---------------------------------|---|----------------------|-------------------------------------|-----------------|
| East Asia and the Pacific       | 125   | 11                   | 18                                  | 15              |
| Eastern Europe and Central Asia | 14  | 0                    | 3                                   | 4               |
| Latin America and the Caribbean | 22  | 6                    | 10                                  | 15              |
| Middle East and North Africa    | 305   | 70                   | 10                                  | 94              |
| South Asia                      | 870   | 157                  | 66                                  | 252             |
| Sub-Saharan Africa              | 1,334   | 383                  | 21                                  | 400             |
| High-income countries           | 0   | 0                    | 6                                   | 0               |

Sources: Underweight: Fishman and others 2004; vitamin A: Rice, West, and Black 2004; iron: Stoltzfus, Mullany, and Black 2004; zinc: Caulfield and Black 2004.

a. In high-income countries, the percentage of children at each weight-for-age criterion are those expected in a healthy population.

b. Considers only deaths directly attributable to iron deficiency anemia in children. Does not include perinatal deaths attributable to maternal iron deficiency anemia.

**Table 28.3** Estimated DALYs Lost by Children Ages Birth through Four Attributable to Selected Nutritional Deficiencies by Region (thousands)

| Region                          | Weight-for-age less than $-1$ SD | Vitamin A deficiency | Iron deficiency anemia <sup>a</sup> | Zinc deficiency | Iodine deficiency |
|---------------------------------|----------------------------------|----------------------|-------------------------------------|-----------------|-------------------|
| East Asia and the Pacific       | 5,777                            | 994                  | 241                                 | 1,004           | 66                |
| Eastern Europe and Central Asia | 489                              | 1                    | 66                                  | 149             | 409               |
| Latin America and the Caribbean | 725                              | 218                  | 109                                 | 587             | 83                |
| Middle East and North Africa    | 10,308                           | 2,403                | 109                                 | 3,290           | 381               |
| South Asia                      | 27,879                           | 4,761                | 704                                 | 8,510           | 366               |
| Sub-Saharan Africa              | 45,131                           | 13,552               | 596                                 | 14,094          | 748               |
| High-income countries           | 0                                | 0                    | 40                                  | 2               | 2                 |

Sources: Underweight: Fishman and others 2004; vitamin A: Rice, West, and Black 2004; iron: Stoltzfus, Mullany, and Black 2004; zinc: Caulfield and Black 2004.

a. Only considers DALYs directly attributable to iron deficiency anemia. Not included are DALYs due to perinatal deaths attributable to maternal iron deficiency anemia.

prevented through improvements in child nutrition have not been quantified.

Evidence is accumulating that early malnutrition increases the risk of numerous chronic diseases later (Caballero 2001; Gluckman and Hanson 2004). Associations of early undernutrition with diabetes, hypertension, renal disease, and cardiovascular disease mean that child undernutrition also leads to high adult health care costs.

### Vitamin A Deficiency

Vitamin A deficiency (VAD) is a common cause of preventable blindness and a risk factor for increased severity of infectious disease and mortality (Rice, West, and Black 2004). One of the first symptoms of marginal VAD is night blindness. If VAD worsens, additional symptoms of xerophthalmia arise, eventually resulting in blindness. A child who becomes blind from VAD has only a 50 percent chance of surviving the year. Even if children survive, blindness severely diminishes their economic potential. VAD may cause anemia in some regions, but it does not appear to impair children's growth (Ramakrishnan and others 2004).

Increased mortality is associated with VAD, most likely because of the detrimental effects on the immune system, which result in increased severity of illness (Sommer and West 1996). According to Rice, West, and Black (2004), VAD is responsible for almost 630,000 deaths each year from infectious disease (table 28.2), accounting for 20 to 24 percent of the mortality from measles, diarrhea, and malaria (Rice, West, and Black 2004). Attributable fractions are highest where VAD is prevalent and mortality is high. Linking morbidity with VAD is far more difficult. Vitamin A supplementation decreases the severity of diarrhea and complications from measles, but in some trials, supplementation has been associated with increased lower respiratory infections.

VAD results from inadequate intakes of vitamin A because of low intakes of animal foods; inadequate intakes of nonanimal sources of carotenoids that are converted to vitamin A; and

inadequate intakes of fat, which facilitates the absorption of carotenoids. Dietary sources of preformed vitamin A include liver, milk, and egg yolks. Dark green leafy vegetables such as spinach, as well as yellow and orange noncitrus fruits (mangoes, apricots, papayas) and vegetables (pumpkins, squash, carrots), are common sources of carotenoids (vitamin A precursors), which are generally less bioavailable than preformed vitamin A but tend to be more affordable.

Table 28.1 shows recent estimates of the prevalence of VAD in young children (Rice, West, and Black 2004). Of those affected, 250,000 to 500,000 each year will lose their sight as a result. The overall prevalence of VAD is decreasing markedly because of increased awareness of VAD as a public health problem and increased measles immunization and vitamin A supplementation or fortification programs. However, the prevalence of VAD is increasing or is unknown in some regions because of political instability, high rates of infectious disease, and increasing poverty.

### Iron Deficiency

More than 2 billion people, mostly women and young children, are thought to be iron deficient (Stoltzfus and Dreyfuss 1998). Iron is found in all plant foods but is more plentiful and bioavailable in meat. Deficiency results from insufficient absorption of iron or excess loss. Absorption is tightly regulated in the intestines, depending on the iron status of the individual, the type of iron, and other nutritional factors. Once iron is absorbed, it is well conserved. Iron is depleted primarily through blood loss, including from parasitic infections such as schistosomiasis and hookworm.

Mainly found in hemoglobin, iron is essential for the binding and transport of oxygen, as well as for the regulation of cell growth and differentiation (Beard 2001). Iron deficiency is the primary cause of anemia, although vitamin A deficiency, folate deficiency, malaria, and HIV also result in anemia. Iron deficiency anemia is most prevalent in South Asia and Sub-Saharan

Africa, but it is not limited to developing countries (table 28.1). Iron deficiency results in neurological impairment, which may not be fully reversible (Grantham-McGregor and Ani 1999). Finally, iron deficiency is known to decrease immune function, but some investigators have also hypothesized that deficiency protects against infectious disease or that iron supplementation increases infectious disease (Caulfield, Richard, and Black 2004). Iron deficiency and anemia do not appear to contribute to growth faltering (Ramakrishnan and others 2004).

Stoltzfus, Mullany, and Black (2004) find that iron deficiency anemia was an underlying factor in 841,000 deaths per year resulting from maternal and perinatal causes, and it directly causes the deaths of 134,000 young children annually (table 28.2). Worldwide, iron deficiency is a substantial contributor to DALY losses (table 28.3).

### **Iodine Deficiency**

Iodine is necessary for the thyroid hormones that regulate growth, development, and metabolism and is essential to prevent goiter and cretinism. Inadequate intake can result in impaired intellectual development and physical growth. A range of impairments resulting from iodine deficiency are referred to as iodine deficiency disorders (IDD) (Hetzel 1983) and can include fetal loss, stillbirth, congenital anomalies, and hearing impairment. The vast majority of deficient individuals experience mild mental retardation. This decrease in mental ability and work capacity may have significant economic consequences. Iodine deficiency has not, however, been associated with the incidence or severity of infectious disease, and studies implicating deficiency as an underlying cause of mortality are limited. Because of this, few child deaths can be attributed to iodine deficiency, but the directly attributable DALY losses remain considerable (table 28.3).

The prevalence of iodine deficiency is often estimated from the prevalence of palpable goiter, but this method is not sensitive to milder expressions of deficiency. Iodine deficiency is thought to be a public health problem in a community if goiter is detected in more than 5 percent of the school-age population. A prevalence greater than 30 percent means that the deficiency is severe. According to World Health Organization (WHO) estimates, goiter rates among school-age children exceed 5 percent in 130 countries, putting 2,225,000 people at risk of IDD. A high prevalence of IDD occurs in Eastern Europe and Central Asia, the Eastern Mediterranean and North Africa, South Asia, and Sub-Saharan Africa (WHO 1999). Iodized salt programs are decreasing iodine deficiency in many regions; however, this reduction is offset by apparent increases in other regions, where public health officials are now aware of the problem because of increased surveillance.

Switzerland and the United States embarked on iodine fortification programs in earnest in the early 1920s. Success resulted in enthusiastic political and financial support for increased

global coverage, and control of IDD through salt iodation represents a great achievement in international public health. Nevertheless, significant numbers of people remain at risk.

### **Zinc Deficiency**

Zinc is ubiquitous within the body and is vital to protein synthesis, cellular growth, and cellular differentiation. Studies in children have demonstrated important roles for zinc in relation to immune function, growth, and development (Brown and others 2002; Shankar and Prasad 1998).

Zinc deficiency results from inadequate intakes and, to some extent, increased losses. Only animal flesh, particularly oysters and shellfish, is a good source of zinc, and fiber and phytates inhibit absorption. Thus, as with iron deficiency, populations consuming a primarily plant-based diet are susceptible. Deficiency can also result from losses during diarrheal illness.

Consensus is currently lacking on how to measure zinc deficiency in individuals. The International Zinc Nutrition Consultative Group recommended using serum or plasma zinc concentrations to identify the risk of deficiency at the population level. In addition, the group used information on absorbable zinc in the food supplies of 176 countries to estimate the proportion of each national population at risk of inadequate intake (table 28.1). This information was used to calculate the burden of disease (table 28.2) associated with zinc deficiency in young children. Prevalence is not expected to decrease unless the implementation of zinc-related interventions increases substantially (Caulfield and Black 2004).

The health consequences of severe zinc deficiency have been elucidated over the past 40 years, whereas the health risks of mild to moderate deficiency have been described only recently. Clinical presentations of severe deficiency include growth retardation, impaired immune function, skin disorders, hypogonadism, anorexia, and cognitive dysfunction. Mild to moderate deficiency increases susceptibility to infection, and the benefits of zinc supplementation on the immune system are well documented (Shankar and Prasad 1998). Zinc can prevent and palliate diarrhea and pneumonia (Zinc Investigators' Collaborative Group and others 1999, 2000) and also may reduce malaria morbidity in young children (Caulfield, Richard, and Black 2004). Improvements in growth have been demonstrated (Brown and others 2002), which may operate directly or indirectly through increased immune function and decreased infectious disease.

Zinc deficiency is estimated to be responsible for about 800,000 deaths annually from diarrhea, pneumonia, and malaria in children under five (table 28.2). Sub-Saharan Africa, the Eastern Mediterranean, and South Asia bear the heaviest attributable burden of pneumonia and diarrhea, with Sub-Saharan Africa accounting for nearly the entire attributable malaria burden.

## INTERVENTIONS

Clearly, growth faltering and micronutrient deficiency disorders are prevalent, have deleterious consequences for children's health and development, and are primary contributors to the global burden of disease. Economic development is not the only path to solving childhood undernutrition. Improvements in family income may not translate into increased food intakes because the income elasticity for caloric intake is relatively low. The effects on micronutrient deficiencies might be greater if the food sources of those nutrients (meat, seafood, eggs, fortified food products) were more sensitive to income increases and if children had access to those foods. Price subsidies may reduce undernutrition in young children if targeted to foods consumed by them; the potential contribution of price subsidies to family nutrition is discussed elsewhere (see chapter 11). This chapter focuses on specific public health measures that are intended to address the problems directly. Progress has been made in some areas, but the current magnitude of the problems and of the associated disease burden underscore the need for more investment in nutritional interventions.

### Growth Faltering and Childhood Stunting

Infants and young children falter in their growth because of inadequate dietary intakes and recurrent infectious diseases, which reduce appetite, increase metabolic requirements, and increase nutrient loss. Even though this problem is understood, progress to reduce malnutrition has been slow. Over time, thinking on how to reduce growth faltering and childhood stunting has shifted. Whereas previous efforts focused almost exclusively on identifying and rehabilitating severely malnourished children, current efforts emphasize prevention through combined nutritional and disease prevention and treatment interventions.

Initially, these efforts to prevent undernutrition focused on diseases rather than on improved child feeding practices as such. However, according to Becker, Black, and Brown (1991), despite the devastating effects of illness on nutritional status, improving dietary intakes is more effective than disease prevention efforts in reducing undernutrition. Because of dramatic reductions in appetite during illness, efforts to improve dietary intakes initially focused on maintaining energy intakes despite anorexia and on increasing intakes during recuperation, when appetite may be normal or high. More recent interventions aim at feeding healthy children optimal diets, which includes paying attention to dietary quality. Finally, some have argued that, for nutritional advice to be effective, it needs to be provided alongside growth monitoring and promotion; however, it is increasingly recognized that messages for prevention are largely universal and that integrated growth monitoring and promotion are not the only model for service delivery.

**Promotion of Optimal Feeding of Infants and Young Children.** Much of the early focus on optimal feeding was on breastfeeding, which should be immediate and exclusive until six months of age. At that time nutritious and safe foods should be added to a diet that is still based on breast milk until early in the second year of life. A consensus has been reached that six months is the recommended duration of exclusive breastfeeding (WHO 2002) and that the total duration is a decision left to the mother.

Multiple approaches exist to promote the initiation of breastfeeding and to prolong exclusive breastfeeding—health education; professional support; lay support; health sector changes (for example, infant friendly hospitals); and media campaigns—through health facilities and community programs. A recent Cochrane review estimates the potential effectiveness of these approaches (Sikorski and others 2002). Women who received any form of support for breastfeeding were 22 percent less likely to stop exclusive breastfeeding, and women who received lay support, in particular, were 34 percent less likely to stop exclusive breastfeeding. Substantial evidence indicates that interventions can be effective in prolonging breastfeeding and exclusive breastfeeding and that operational research is needed for program implementation and sustainability. If such programs were fully successful, they would reduce deaths in children under five by 13 percent (Jones and others 2003).

Complementary feeding is the process of introducing other foods and liquids into the child's diet when breast milk alone is no longer sufficient to meet nutritional requirements. According to Brown, Dewey, and Allen (1998), complementary feeding practices are suboptimal from several perspectives:

- Complementary foods are introduced too early or too late.
- Foods are served too infrequently or in insufficient amounts, or their consistency or energy density is inappropriate.
- The micronutrient content of foods is inadequate to meet the child's needs, or other factors in the diet impair the absorption of foods.
- Microbial contamination may occur.

In addition, because children often do not eat all the food offered to them, interaction between the caregiver and the child, along with other psychosocial aspects of care during feeding, requires attention. The amount of complementary food a child needs depends on breast milk intake. Guidelines are available for determining energy and nutrient intakes from complementary foods, given breast milk intakes (Dewey and Brown 2003).

Several reviews of the multiple approaches to improving infant and young child feeding practices are available (Allen and Gillespie 2001; Caulfield, Huffman, and Piwoz 1999; Dewey 2002; Hill, Kirkwood, and Edmond 2004; Swindale and others 2004). Caulfield, Huffman, and Piwoz (1999) review

16 programs in 14 countries to improve dietary intakes of infants 6 to 12 months of age. The programs were designed to promote exclusive breastfeeding and appropriate feeding during illness up to age three, and the content and approaches reflected current thinking regarding nutrition and behavior change. The approaches employed included using the mass media to reach both caregivers and the population as a whole to change cultural norms about complementary feeding and using one-on-one or small group interactions with community health workers to provide individualized information and support.

Most of the projects achieved good coverage (50 to 70 percent), with rates varying depending on the communication strategy. They resulted in large shifts in maternal knowledge and attitudes and changes in infant feeding practices. In the few programs assessing dietary intakes, intakes improved by 70 to 165 kilocalories per day. Differences in nutritional status at 12 months indicated weight-for-age and height-for-age gains of 0.24 to 0.87 SD. Even with a 50 percent overestimation of the effects, the effect of such programs could translate into tangible reductions in malnutrition and attributable mortality. In addition, these calculations do not consider the cumulative reduction in malnutrition from programs that benefit children's growth into the second and third years of life. Jones and others (2003) use the results of the analysis, along with knowledge of the relationship between underweight and child mortality, to estimate that programs to promote complementary feeding could reduce by 6 percent the deaths of children under five in developing countries.

Many programs provide supplemental food to participants either to provide them an incentive for participating in other activities (to offset time costs and increase consumer demand for preventive services) or to rehabilitate severely malnourished children. Although the latter approach is traditionally considered for supplemental food programs, the former approach is more common. Indeed, India's Integrated Child Development Services Program, the world's largest supplemental food program, plans to shift from rehabilitation to the use of supplemental food as a "magnet" for providing other integrated child development services (Kapil 2002). No consensus exists on when or how to include supplemental food to reduce undernutrition, and inefficient targeting is frequently a key constraint to effectiveness. Swindale and others' (2004) review of the effectiveness of food-assisted child survival programs concludes that such programs are reducing malnutrition by 2.0 to 2.5 percent per year.

Despite evidence of the effectiveness of nutritional interventions in improving feeding practices and preventing undernutrition, few programs take a comprehensive approach toward optimizing infant feeding, perhaps because of a lack of consensus on the key components of a comprehensive strategy. In 2002, participants at a WHO consultation developed 10 guiding

principles for optimal feeding of the breastfed child (PAHO and WHO 2003). These principles, outlined in box 28.1, build on lessons from previous programmatic efforts such as those reviewed here and provide a basis for designing comprehensive programs to reduce malnutrition. The international public health community faces the challenge of implementing and evaluating these approaches.

**Disease Control and Prevention.** Interventions to prevent or decrease malnutrition or infectious disease are expected to decrease child mortality, and interventions that accomplish both will have the greatest effect (Pelletier, Frongillo, and Habicht 1993). This subsection considers the potential for disease control and prevention efforts to reduce undernutrition in young children.

Malaria is responsible for a large portion of childhood mortality in Sub-Saharan Africa. The effect of undernutrition on susceptibility to malaria has been discussed at length elsewhere (Caulfield, Richard, and Black 2004), but the nutritional deficiencies resulting from malaria have been insufficiently explored. Insecticide-treated bednets have been shown to prevent clinical episodes of malaria and decrease the prevalence of anemia in children (Lengeler 2003). Improvements in growth have also been documented.

Water, sanitation, and hygiene interventions decrease childhood malnutrition primarily by preventing diarrheal disease (Checkley and others 2004). Hand-washing interventions can reduce the risk of diarrheal diseases by about 45 percent. Hand-washing interventions can be included in water and sanitation programs or can exist as a single intervention, and they are both effective and cost-effective (Borghi and others 2002).

### **Vitamin A Deficiency**

Even though the consequences of VAD had been defined by 1920, it was 1986 when vitamin A interventions were rigorously studied in a large, controlled community trial (Sommer and West 1996). A number of other community trials soon also demonstrated a significant decrease in child mortality with vitamin A supplementation (Beaton and others 1993). Supplementation can alleviate acute VAD quickly, whereas long-term strategies incorporate fortification and dietary diversification.

Supplementation can be either a curative or a preventive measure. If an individual presents with ocular symptoms of VAD, supplementation is part of the usual standard of care. Beyond the use of supplementation for symptoms that result directly from deficiency, its use as part of the treatment regimen for measles or severe malnutrition can improve health outcomes. In deficient areas, high-dose oral supplementation is recommended every four to six months for children under five and is highly efficacious in reducing ocular effects as well as

## Box 28.1

### Guiding Principles for Complementary Feeding of the Breastfed Child

1. Practice exclusive breastfeeding from birth to six months of age and introduce complementary foods at six months of age (180 days) while continuing to breastfeed.
2. Continue frequent, on-demand breastfeeding until two years of age or beyond.
3. Practice responsive feeding, applying the principles of psychosocial care. Specifically, do the following:
  - Feed infants directly and assist older children when they feed themselves, being sensitive to their hunger and satiety cues.
  - Feed slowly and patiently; encourage children to eat, but do not force them.
  - Experiment with different food combinations, tastes, textures, and methods of encouragement if children refuse many foods.
  - Minimize distractions during meals if the child loses interest easily.
  - Remember that feeding times are periods of learning and love, and talk to children during feeding, including making eye contact.
4. Practice good hygiene and proper food handling:
  - Wash hands before food preparation and eating (both caregivers and children).
  - Store foods safely and serve foods immediately after preparation.
  - Use clean utensils to prepare and serve food.
  - Use clean cups and bowls when feeding children.
  - Avoid the use of feeding bottles, which are difficult to keep clean.
5. Start at six months of age with small amounts of food and increase the quantity as the child gets older, while maintaining frequent breastfeeding. According to average breast milk intakes in developing countries, infants' needs from complementary foods are approximately 200 kilocalories per day at 6 to 8 months, 300 kilocalories per day at 9 to 11 months, and 550 kilocalories per day at 12 to 23 months.
6. Increase food consistency and variety gradually as the infant gets older, adapting to the infant's requirements and abilities. Infants can eat pureed, mashed, and semisolid foods beginning at six months. By eight months most infants can also eat finger foods—that is, snacks that they can eat unaided. By 12 months, most children can eat the same types of foods that the rest of the family consumes, keeping in mind the need for nutrient-dense foods. Avoid foods that cause choking.
7. Increase the frequency with which the child is fed complementary foods as he or she gets older. The appropriate number of feedings depends on the energy density of local foods and the usual amounts consumed at each feeding. For the average healthy, breastfed infant, meals should be provided two or three times a day at 6 to 8 months of age and three or four times a day at 9 to 23 months of age, with additional snacks.
8. Feed a variety of foods to ensure that nutrient needs are met. The child should eat meat, poultry, fish, or eggs daily, or as often as possible. Vegetarian diets cannot meet nutrient needs at this age unless nutrient supplements or fortified products are used.
9. Use fortified complementary foods or vitamin and mineral supplements for the infant, as needed. In some populations, breastfeeding mothers may also need vitamin and mineral supplements or fortified products for their own health and to ensure normal concentrations of certain nutrients in their breast milk.
10. Increase fluid intake during illness, including more frequent breastfeeding, and encourage the child to eat soft, varied, appetizing, favorite foods. After illness, give food more often than usual, and encourage the child to eat more.

Source: PAHO and WHO 2003.

mortality (Sommer and West 1996). A meta-analysis of controlled trials in children demonstrated a 23 percent reduction in mortality (Beaton and others 1993). High-dose vitamin A supplements are considered safe for infants younger than six months. Several studies suggest that giving vitamin A within 48 hours of birth reduces mortality in the first three months by 21 to 74 percent (D. Ross 2002).

A variety of foodstuffs have been fortified with vitamin A, including oil, monosodium glutamate, butter, wheat flour, sugar, and rice. Fortified white sugar has been successful in reducing VAD prevalence in Central America. In El Salvador and Guatemala, where fortified sugar is the primary source of vitamin A, it accounts for approximately 30 percent of the recommended dietary intake (RDI). Fortification of monosodium

glutamate with vitamin A has been demonstrated to be biologically efficacious. Even though program implementation was flawed by unacceptable cost, discoloration of the monosodium glutamate, and packaging problems, indicators of VAD declined significantly during periods of fortification in both Indonesia and the Philippines (Dary and Mora 2002).

Vitamin A intakes can also be improved through dietary diversification, either by educating communities about important sources of vitamin A and beta-carotene that are available in the local diet or by increasing economic prosperity so that individuals have additional funds to spend on a wider variety of food. Education alone has not been demonstrated to affect the degree of VAD in a community, but it can be a powerful tool when incorporated in a broader strategy that also includes supplementation and fortification (Sommer and West 1996).

### **Iron Deficiency**

Despite the public health community's enduring interest in preventing and treating iron deficiency anemia, little evidence suggests that the problem has been reduced. Indeed, in some regions the opposite may be true. From the 1970s to the 1980s, the iron density of people's diets decreased in every region except the Near East and North Africa as iron-poor cereals displaced legumes. During much of this period, iron deficiency anemia increased in South Asia and Sub-Saharan Africa, where the problem is most severe (Stoltzfus, Mullany, and Black 2004). Goals for reducing iron deficiency anemia were articulated for the 1990s at the 1990 World Summit for Children, and many countries adopted policies for providing supplementation for young children; however, few large programs have been developed to eliminate the problem.

The explanations for this failure to act include doubts among both scientific program planners and policy makers about the causes and consequences of iron deficiency and anemia; lack of political commitment; inadequate program planning, including mobilization and training of health staff members; insufficient community involvement; and, in particular, inherent difficulties with prolonged adherence to daily supplementation (Stoltzfus, Mullany, and Black 2004). Despite this bleak picture, guidelines for supplementation have been formulated for children ages 6 to 24 months and for low birth-weight infants beginning at 2 months (Stoltzfus and Dreyfuss 1998). Also, various scientific documents synthesize and communicate current knowledge about the consequences of iron deficiency anemia and programming efforts.

Ample evidence indicates that iron deficiency is the principal cause of anemia in children; that iron supplements are efficacious in preventing and treating iron deficiency anemia, increasing hemoglobin concentrations by about 1 gram per deciliter on average in controlled trials; and that supplements reduce severe anemia even in malarious areas. The contribution of parasitic

infections such as malaria and hookworm to anemia does not negate the usefulness of iron supplements; rather it underscores the need for multiple inputs to prevent severe anemia, given the risks of transfusion. Although current recommendations indicate daily supplements, less frequent delivery, such as intermittently or weekly, is commanding interest. Beaton and McCabe's (1999) meta-analysis concludes that both daily and weekly supplementation are efficacious if adherence is good.

In many countries, iron fortification of foods is the principal strategy for reducing iron deficiency and anemia. Fortified foodstuffs include wheat and maize flours, noodles, sugar, condiments, and complementary foods and milk for infants and children. Efficacy studies indicate the potential of fortification to increase iron intakes and reduce anemia, and effectiveness trials in Chile (dry milk for infants), Ghana (complementary food for young children), Guatemala (sugar), India (salt), Mexico (fortified weaning food and dry milk), and República Bolivariana de Venezuela (maize and wheat) have found improvements in hemoglobin concentration or reductions in anemia prevalence (Allen and Gillespie 2001; Rivera and others 2004). Nevertheless, few national iron fortification programs have evaluation results that are without controversy. Yip and Ramakrishnan (2002) argue that the strongest examples of the potential for fortification are found in the Chilean program of fortified dry milk for infants and in the U.S. program of iron-fortified infant cereals. A randomized trial in Mexico of a poverty alleviation program that distributes a complementary food fortified with multiple micronutrients, including iron, found positive effects on anemia rates (Rivera and others 2004). Evaluations of newly implemented iron fortification programs should gauge their contribution to anemia prevention.

Newer strategies, such as sprinkles (powders), spreads, or foodlets (a hybrid of a food and a tablet), appear promising, particularly for regions where the infrastructure will not support more traditional forms of fortification (Zlotkin and others 2003). Processed complementary foods and beverages offer additional vehicles for reducing iron and other micronutrient deficiencies and promoting well-being (Solon and others 2003). Implementing such strategies and documenting their cost-effectiveness are important activities for the next few years.

In many settings, promoting iron-rich organ meats and animal products and undertaking other food-based strategies may increase iron intakes and contribute to anemia reduction. Such approaches have been promoted for many years, but research is still needed to document their efficacy and effectiveness (Ruel and Levin 2000).

### **Iodine Deficiency Disorders**

Interventions to diminish iodine deficiency using either supplementation or fortification are both efficacious and inexpensive, and WHO, the United Nations Children's Fund, and



the International Council for the Control of Iodine Deficiency Disorders have pledged to eliminate iodine deficiency and the spectrum of IDD.

For regions with severe endemic iodine deficiency, high-dose iodine supplementation is indicated while longer-term solutions are put into place. Iodized oil and iodide tablets are the most common means of direct administration. Injections of iodized oil have been used with much success to decrease the prevalence of IDD and have been shown to be effective for three to four years, depending on the dosage (Hetzel 1989). Although injected oil is effective, it is also expensive, requires trained personnel to administer, and carries the risk of infectious disease transmission from contaminated needles. Because of those drawbacks, researchers began exploring oral administration as an alternative. Oral administration of iodized oil in liquid and tablet form has been successful in the long-term correction of clinical deficiency, and in Indonesia, oral administration was associated with a reduction in infant mortality (Cobra and others 1997).

Iodized or iodated salt is the primary strategy for correcting iodine deficiency because of the nearly universal consumption of salt regardless of socioeconomic status; the lack of an effect on consistency, color, or taste from the addition of iodine; and the limited number of producers in many countries. Large-scale salt fortification has been highly successful in many countries, and of the 130 countries with iodine deficiency, 75 percent have laws mandating salt iodization. The goal of universal salt iodization for consumption by both humans and livestock in all countries with endemic iodine deficiency was set at the 1990 World Summit for Children (WHO, UNICEF, and ICCIDD 2001). Some populations do not easily embrace salt iodization because of cultural preferences or because they have an ample supply of unprocessed salt, so other means of fortification are needed. One promising option is to add potassium iodate to irrigation water.

### Zinc Deficiency

Although zinc deficiency is likely widespread and even mild deficiency probably has significant health consequences, few interventions have been developed to combat it in developing countries. Possible interventions include supplementation, fortification, and dietary diversification or modification. The strong evidence that the use of zinc supplements given during and for a short time after diarrhea improves the outcome of that episode and prevents future episodes has led to the recommendation that zinc, along with increased fluids and continued feeding, be used to treat all episodes of acute diarrhea (WHO and UNICEF 2004). Substantial efforts are under way to initiate programs in developing countries. Prophylactic zinc supplementation also improves growth and reduces diarrhea incidence (International Zinc Nutrition Consultative Group 2004).

Fortification interventions include the traditional method of adding zinc to a commercial food, consumer fortification using sprinkles, and plant-breeding techniques. For example, Mexico has introduced several large-scale programs, including the fortification of maize and wheat flours and the distribution of fortified complementary food and fortified milk to low-income children (Rivera and Sepulveda 2003). Researchers are investigating the possibility of home fortification of food using sprinkles containing iron and zinc (Zlotkin and others 2003), but further research is needed to determine whether sprinkles are a viable option. Through plant breeding and genetic engineering, staple crops may be made to contain more zinc or less phytate, resulting in increased zinc bioavailability (Ruel and Bouis 1998). Other dietary strategies target food preparation techniques, such as fermentation of unrefined flour to increase zinc bioavailability.

### INTERVENTION COSTS AND COST-EFFECTIVENESS

Multiple strategies exist for preventing malnutrition in young children in the short and long term. This section considers the costs and cost-effectiveness of these interventions for preventing malnutrition or deaths attributable to each nutritional problem. Table 28.4 presents a compendium of cost information, including, where possible, the costs of preventing a child death or saving a DALY.

Horton and others (1996) use data from Brazil, Honduras, and Mexico to estimate the costs and cost-effectiveness of hospital-based programs to promote breastfeeding. Using standard costing methods, they examine the costs of breastfeeding promotion activities in each program and the additional inputs, as well as the savings. Savings accrued from the removal of infant formula where it was currently used. Using data on infant feeding practices and morbidity and mortality from Brazil, they estimated the costs of the programs per birth, per diarrhea case averted, and per death averted. As table 28.4 shows, the costs of such programs range from US\$0.30 to US\$0.40 per child, and from US\$100 to US\$200 per death averted, making them comparable in cost-effectiveness to measles and rotavirus vaccination. Assuming that deaths would otherwise have occurred around age one, and using average Latin American life expectancy at that age, yields a cost per DALY gained of only US\$3 to US\$7.

In many community-based strategies, multiple organizations work through a variety of communication channels to promote exclusive breastfeeding. Two studies in Ghana and Madagascar provide cost estimates for such programs (Chee, Makinen, and Sakagawa 2002; Chee and others 2003). The programs cost US\$4 to US\$16 per child, and given the effect on mothers' practices, the cost ranged from US\$5 to

**Table 28.4** Costs and Cost-Effectiveness of Nutrition Interventions

| Type of deficiency and intervention             | Source                                      | Year | Country                  | Costs (US\$)   |                      |                 |
|---|---|------|--------------------------|--|----------------------|-----------------|
|   |   |      |                          | Per child or per outcome                                     | Per death averted    | Per DALY gained |
| <i>Underweight</i>                              |   |      |                          |  |                      |                 |
| Breastfeeding support                           | Horton and others 1996                      | 1996 | Brazil, Honduras, Mexico | 0.30–0.40 per birth; 0.65–1.10 per diarrhea case averted     | 100–200              | 3–7             |
| Breastfeeding promotion                         | Ross, Loening, and Mbele 1987               | 1987 | Mali                     | 2–3 per child  | 282                  | 11              |
| Breastfeeding promotion                         | Chee, Makinen, and Sakagawa 2002            | 2002 | Ghana                    | 16 per child; 5–58 per adopter of exclusive breastfeeding    | 203 <sup>a</sup>     | 7.80            |
|   | Chee and others 2003                        | 2003 | Madagascar               | 4.41 per child; 10–17 per adopter of exclusive breastfeeding | —                    | —               |
| Child survival program with nutrition component | J. Ross 1997; WHO 2002                      | 1997 | Across programs          | 76–101 per undernourished child averted                      | 1,200                | 41–43           |
| Nutrition programs                              |   |      |                          |  |                      |                 |
| Less intensive                                  |   |      |                          | 2–5 per child  |                      |                 |
| More intensive                                  |   |      |                          | 5–10 per child   |                      |                 |
| Growth monitoring and counseling                | Fiedler 2003                                | 2003 | Honduras                 | 4 per child; 20 per undernourished child averted             | 240–320 <sup>b</sup> | 8–11            |
| <i>Vitamin A deficiency</i>                     |   |      |                          |  |                      |                 |
| Capsule distribution                            | Rassas, Hottor, and others 2004             | 2004 | Ghana                    | 0.90 per child   | 277                  | 11              |
|   | Rassas, Nakamba, and others 2004            | 2004 | Zambia                   | 1.23 per child   | 162                  | 6–7             |
|   | Fiedler 2000                                | 2000 | Nepal                    | 1.25 per child   | 327                  | 11–12           |
|   | Fiedler and others 2000                     | 1994 |                          |  |                      |                 |
| Fortification                                   | Institute of Medicine 1998; World Bank 1994 |      | Guatemala                | 0.17 per child   | 1,000                | 33–35           |
| Sugar   |   |      |                          | 0.05–0.15 per child  |                      |                 |
| Other   |   |      |                          |  |                      |                 |
| <i>Iron deficiency</i>                          |   |      |                          |  |                      |                 |
| Supplements                                     | Institute of Medicine 1998; World Bank 1994 | 1994 |                          | 3.17–5.30 per child  | —                    | —               |
| Fortification                                   |   |      |                          |  |                      |                 |
| Salt  | World Bank 1994                             | 1994 | India                    | 0.12 per child   | —                    | —               |
| Sugar   | World Bank 1994                             | 1994 | Guatemala                | 0.20–1.00 per child  | —                    | —               |
| Cereal  | World Bank 1994                             |      |                          | 0.09 per child   | 2,000                | 66–70           |
| <i>Iodine deficiency</i>                        |   |      |                          |  |                      |                 |
|   | Institute of Medicine 1998; World Bank 1994 |      |                          |  |                      |                 |
| Oil injection                                   |   | 1994 | Peru<br>Zaire            | 2.75 per child<br>0.80 per child<br>1.25 per child           | —<br>—<br>—          | —<br>—<br>—     |
| Fortification                                   |   |      |                          |  |                      |                 |
| Water   |   |      | Indonesia                | 0.05 per child   |                      |                 |
| Salt  |   |      | Italy                    | 0.02–0.05 per child  | 1,000                | 34–36           |
| Salt  |   |      | India                    | 0.05 per child   | —                    | —               |
| <i>Zinc deficiency</i>                          |   |      |                          |  |                      |                 |
| Supplements with oral rehydration salts         | Robberstad and others 2004                  | 2004 | n.a.                     | 0.47 per child   | 2,100                | 73              |

— = Source did not include data from which to estimate deaths averted (and DALYs gained).

Note: Deaths prevented by promoting or supporting breastfeeding are assumed to occur around age one. Deaths prevented by other programs to reduce underweight and all programs to reduce micronutrient deficiency are assumed to occur between ages one and five. Authors' estimates of costs per DALY (in parentheses) using region-specific life expectancies at ages one and five, reflect this range.

a. Assumes that all the DALY gains come from preventing deaths.

b. Assumes that an undernourished child has a chance of 1 in 16 to 1 in 12 (6 to 8 percent) of dying before age five, the same as estimated for child survival programs.

US\$58 per adopter of exclusive breastfeeding. In Ghana, an estimated 883 deaths were averted, yielding a program cost of US\$7.80 per DALY gained or US\$203 per death prevented. The range of costs within each program depended on the baseline prevalence of the behavior, the population density, and the characteristics of the implementing organizations themselves. Programs will be more cost-effective when the baseline prevalence is lower; the population density is higher; and the organizations involved are focused, highly motivated, and well organized.

Less information is available on the costs of community-based nutrition programs to prevent growth faltering, to control morbidity, and to improve survival. The costs of a program in Mali (Ross, Loening, and Mbele 1987), which included promotion of breastfeeding, counseling, and education on optimal child feeding; prevention of diarrheal disease; and growth monitoring, were estimated to be US\$282 per death averted and US\$11 per DALY gained. This estimate is consistent with others that nutrition programs cost US\$2 to US\$10 per child, depending on the intensity of nutrition counseling, including Fiedler's (2003) study of the Integrated Community Child Care Program in Honduras, which had an estimated cost of US\$4 per child. (For a fuller analysis of such programs, including contextual and programmatic characteristics that affect outcomes, see chapter 56.)

In the past five years, investigators have undertaken several cost analyses of national programs to distribute vitamin A capsules. Two reports from Ghana and Zambia are particularly informative (Rassas, Hottor, and others 2004; Rassas, Nakamba, and others 2004). As table 28.4 shows, such programs cost US\$0.90 to US\$1.23 per child, with the costs per death averted ranging from US\$162 to US\$277. (Deaths from micronutrient deficiencies are assumed to occur between ages one and five, and estimates of cost per DALY ranging from US\$6 to US\$11 reflect this range, as well as region-specific life expectancies at those ages.) These costs are comparable with estimates of a vitamin A program in Nepal that cost US\$1.25 per child and US\$327 per death averted (Fiedler 2000). Ching and others (2000) examine the costs of incorporating vitamin A capsule distribution into immunization campaigns in 50 countries in 1998 and 1999. Their analysis finds that the total costs per death averted ranged from about US\$150 to US\$600, with the incremental costs for vitamin A distribution amounting to only about US\$30 to US\$150 per death averted. The costs per death averted depended on the country setting, the program's coverage, the delivery of vitamin A (one or two doses), and the underlying level of mortality. The incremental cost per DALY gained could be as low as US\$1 or as high as US\$6.

Fewer examples of vitamin A fortification programs are available, with the only clear example being sugar fortification

in Central America. In 1994, estimates indicated that a program in Guatemala cost US\$0.17 per child, and US\$1,000 per death averted. Counting only the losses from mortality, the cost of saving a DALY was US\$33 to US\$35. However, for each death prevented, there were probably several cases of eye damage prevented and of improved general health; thus, taking full account of nonfatal effects would reduce the cost per DALY somewhat.

Iron supplementation is more costly than distribution of vitamin A capsules, as it involves a daily supplement over an extended period. Estimates indicate that such programs cost US\$3.17 to US\$5.30 per child. Numerous cost estimates are available for iron fortification programs, because these programs have been the principal strategy to prevent and control iron deficiency anemia. Such programs have traditionally cost US\$0.09 to US\$1.00 per child, depending on the country and the vehicle for fortification. These estimates are based on elemental iron as the fortifier. Even though this is the cheapest form available, critics have questioned the bioavailability of elemental iron, and many researchers now advocate using other forms of iron.

Iodine fortification programs cost little, about US\$0.02 to US\$0.05 per child. Iodized oil injections are more costly at US\$0.80 to US\$2.75 per beneficiary, but these programs may be recommended for settings where people consume little commercialized and easily fortified food.

Currently, no examples of zinc intervention programs are available from which to estimate cost-effectiveness. However, Robberstad and others' (2004) simulation analysis examines the potential costs and cost-effectiveness of providing zinc as an adjunct to oral hydration salts in treating diarrhea in young children. Providing zinc as part of case management carries an estimated incremental cost of US\$0.47 per treatment, ranging from US\$0.33 to US\$0.62. Given the relationship between zinc provision and mortality risk, this addition to current management programs would cost, on average, US\$2,100 per death averted and US\$73 per DALY gained.

Despite the enormity of the nutritional problems, the associated loss of DALYS, and the existence of programs to combat malnutrition, surprisingly little data on the costs or cost-effectiveness of nutritional programs are available. This problem represents a serious gap in information for health planning, implementation, and advocacy. Nonetheless, considerable evidence indicates that when programs to promote breastfeeding or child growth or to correct micronutrient deficiencies are delivered to populations with a relatively high prevalence of malnutrition, the cost per participating child is usually so low that deaths can be averted at a cost per DALY that is less than US\$100, and often less than US\$10, even in regions with low life expectancy. Few health interventions are comparably cost-effective.

## ECONOMIC BENEFITS OF INTERVENTION

The previous sections outlined the costs to society in terms of deaths and disabilities resulting from growth faltering and micronutrient malnutrition, as well as the costs and cost-effectiveness of options for their alleviation and prevention; however, DALYs do not capture the full range of potential benefits to society from effective nutrition programs. For example, even though the effect of iron deficiency on mental retardation in children contributes to the attributable DALYs (Stoltzfus, Mullany, and Black 2004), the negative effects of iron deficiency on cognition that do not constitute retardation are not considered. Other effects of malnutrition on cognitive and physical functioning that ultimately affect labor productivity are also not considered, nor are other long-term health consequences of child undernutrition. Finally, because undernutrition increases the frequency and severity of disease, undernutrition is associated with considerable health care costs, which are also not captured in burden estimates.

### Malnutrition and Human Capital Formation

Researchers have studied cognitive function using global measures of development and intelligence, such as IQ, along with school performance and more narrowly defined intellectual, psychomotor, and behavioral skills. A large body of research has examined whether undernutrition causes lasting cognitive deficits in later life and whether potential deficits are amenable to subsequent nutritional interventions. Acute malnutrition is associated with negative neuroanatomical, emotional, and behavioral effects on children's development. After recovery, results of behavioral and developmental tests generally improve, but the long-term developmental implications remain unclear. Many studies find IQ scores 8 to 18 points lower in children who suffered from severe malnutrition (Fishman and others 2004). Studies of chronic undernutrition also report deficits in IQ and school performance with stunting during early childhood. Evidence from nutritional interventions among high-risk or undernourished children suggests that early supplementary feeding (but no sooner than two years of age) improves developmental scores during the intervention, with some evidence of long-term benefits. For example, follow-up of Guatemalan children exposed to prenatal and early postnatal supplementation demonstrated long-term cognitive benefits even after adjusting for socioeconomic factors and educational experience (Pollitt and others 1995). These results argue strongly for preventing acute severe malnutrition and generalized growth faltering that leads to stunting in children.

Nutritional interventions may preserve or improve cognitive function through mechanisms other than preventing growth faltering or acute malnutrition. For example, breastfeeding confers some cognitive benefits. Anderson, Johnstone,

and Remley's (1999) meta-analysis estimates gains of 3.5 IQ points, adjusting for important covariates.

Iron deficiency has long been associated with developmental delays, and iron supplementation studies have demonstrated improvements in cognitive function. Whether the negative effects of iron deficiency and anemia on development are reversible remains controversial, which implies the need for strong preventive measures. More research is needed to learn about the effects of iron deficiency on development and to develop measures for evaluating programs that provide iron.

Multiple lines of evidence indicate that zinc influences development (Black 1998). Despite a clear biological role, epidemiological studies provide insufficient evidence to draw conclusions on the gain in human capital if zinc deficiency were reduced through public health interventions. Research to address this gap is under way.

The public health community has long recognized that iodine deficiency is the most common cause of preventable mental retardation. Even though the problem of maternal iodine deficiency and cretinism in the offspring is well recognized, evidence also suggests that deficiency in children is negatively associated with cognitive abilities. Bleichrodt and Born's (1994) meta-analysis finds losses of 13.5 IQ points in those with iodine deficiency. Some of these effects occur in the absence of goiter, the hallmark of IDD. More research is needed to fully understand the human consequences of milder forms of iodine deficiency that are probably still prevalent in developing countries.

### Malnutrition and Loss of Productivity

Abundant evidence demonstrates that both anemia and iron deficiency decrease fitness and capacity for aerobic work by decreasing oxygen transport and respiratory efficiency in muscles. The consequences of iron deficiency are thus measurable in terms of loss of economic productivity. Aguayo, Scott, and Ross's (2003) case study in Sierra Leone estimates that anemia among women is associated with agricultural productivity losses of US\$19 million per year. For children, the economic costs are not as clear, but those costs may be substantial depending on the children's ages and the types of work they perform.

Growth faltering that leads to stunting in early childhood translates into shortened adult stature. Adult height is related not only to total food consumption but also to protein intake (Jamison, Leslie, and Musgrove 2003), which reinforces the importance of dietary quality. Multiple levels of evidence link adult stature and worker productivity (Martorell 1996). Haddad and Bouis (1991) estimate that a 1 percent decrease in adult stature is associated with a 1.4 percent decrease in productivity. Others find that a 1 percent increase in adult stature is associated with a 2.0 to 2.4 percent increase in wages or earnings. Other things being equal, current programs to prevent

stunting in early childhood can deliver about a third to a half of that 1 percent increase in adult stature. Thus, a lifetime of economic loss results from a failure to prevent stunting in early childhood and accompanying deficits in adult stature, and strategies to reduce this tremendous loss are available.

In addition, the impacts of malnutrition on cognitive development translate indirectly into deficits in productivity in adulthood. Children who are malnourished are more likely to start school late, to perform less well, and to stay in school for a shorter time (Behrman, Alderman, and Hoddinott 2004). Studies suggest that improvements in nutrition within the current range of benefits of programs for young children can lead to substantial increases in rates of school initiation and to more years of schooling (Alderman, Hoddinott, and Kinsey 2003; Alderman and others 2001; Behrman and others 2003). Both years of schooling and school performance affect wages and economic productivity. Alderman, Hoddinott, and Kinsey (2003) calculate that the effects of malnutrition during early childhood, with the accompanying effects on schooling, lead to a 12 percent reduction in lifetime earnings in Zimbabwe. Current programming could restore a significant proportion of those lost wages.

### **Resource Allocation**

Malnutrition increases the likelihood that a child will be sick and, when sick, will become seriously ill. Thus, resources must be allocated to health care services to deal with the increased frequency and severity of illness caused by undernutrition and micronutrient deficiencies. To our knowledge, this increase in likelihood and severity of illness has never been quantified, but it is likely to be high, considering not only the costs of health care infrastructure, but also the time costs and costs of lost wages or schooling borne by the family for each episode of illness. Furthermore, to the extent that undernutrition or micronutrient deficiencies lead to deficits in cognitive development, resources need to be allocated to special education, rehabilitation, and vocational services. The costs associated with not providing such services are ultimately paid in mortality and economic statistics.

### **Adult Disease and Disability**

In the past 10 years, a growing literature has identified associations between small size at birth; early patterns of postnatal growth; and adult conditions as diverse as diabetes, cardiovascular disease, and schizophrenia. More research is needed to provide evidence of causality for such associations and to create the evidence base for attributing those effects to malnutrition in burden-of-disease calculations. Given current knowledge, health care budgets in developing countries will likely be strained to deal with the burden of chronic diseases of adulthood caused by the failure to prevent maternal and child undernutrition.

## **PROGRAM IMPLEMENTATION: LESSONS OF EXPERIENCE**

For decades, countries have implemented programs to alleviate growth faltering and micronutrient deficiencies in children; therefore, it is timely to consider what has been accomplished and what can be learned from successes and failures. The task is difficult, because nutrition programs are diverse, ranging from the simple fortification of salt with iodine to multifaceted programs to improve dietary intakes and prevent growth faltering. Nonetheless, some general statements about the state of programming in this area are possible.

Success in conceptualizing and implementing programs to reduce growth faltering by combining disease control strategies with the promotion of breastfeeding and optimal complementary feeding has been demonstrable. The focus has shifted away from growth monitoring and promotion (counseling) strategies to population-based assessment with more generalized dissemination of key messages for behavior change. Available data suggest high cost-effectiveness for such programs. Key challenges involve scaling up and sustainability, as well as strengthening of monitoring and evaluation systems. A gap in this knowledge concerns optimal feeding in the presence of HIV infection, and testing options and designing programs in such settings are of the highest priority.

Iodine fortification has been a clear success over decades, which underscores the need for continued and consistent funding and advocacy for such programs. Even when universal access to iodine becomes a reality, policy and programmatic supports will be necessary to maintain it.

The success of iodine fortification contrasts with other examples of fortification that have made slow and uneven progress. Fortification of foodstuffs with vitamin A is limited geographically, and even though many countries have embarked on iron fortification, these programs lag because of controversies about the effectiveness of existing programs, the evaluation methods used, and the lack of infrastructure for fortification in some settings. Concerted efforts to address the controversies and to provide evidence of the effectiveness of fortification in controlling iron deficiency are under way. In addition, recognition that fortification should address multiple micronutrient deficiencies, chiefly the B vitamins and zinc, has grown.

Programs to distribute vitamin A capsules twice a year are a reality in many areas characterized by VAD. Jones and others (2003) estimate that current coverage of supplementation for children living in areas with VAD is 55 percent. In the past few years, studies have provided solid data on the costs and cost-effectiveness of such programs in diverse settings.

In contrast, despite concerns about the health and developmental consequences of iron deficiency and anemia, few examples are available of even small-scale iron supplementation programs for young children. Supply and adherence continue to

constrain progress, with adherence depending on program workers' and families' perceptions of benefits and their reluctance to continue the long-term use in children of what are often considered to be medicines. Similar constraints apply to programs to provide iron supplements to pregnant women, but operational research has overcome many obstacles, and the hope is that the lessons learned can inform the design and implementation of iron supplementation programs for young children.

Food-based strategies, particularly dietary diversification and the promotion of specific food groups for preventing micronutrient malnutrition, are less advanced than other programs. In part, this lack of advancement reflects the diverse nature of the behaviors to be changed and of the available options. Given this diversity and the fact that such strategies are more setting specific than, for example, capsule distribution, the lack of summary estimates of effectiveness is not surprising. Consensus is growing that improving dietary intakes through agricultural innovations and dietary diversification represents long-term answers to micronutrient malnutrition, but progress is slow because of the urgency of alleviating deficiencies in the short term. More research is needed to define the policies that will promote these strategies.

Research over the past decade has articulated a strong case for interventions to prevent zinc deficiency, with supplementation and fortification identified as important approaches. Experiences with zinc supplementation or fortification programs are needed to provide estimates of costs and cost-effectiveness. If the costs of providing zinc supplements to young children are in line with those reported earlier, then such programs would be highly cost-effective, considering the prevalence and burden of disease associated with zinc deficiency.

Child malnutrition results from multiple factors, and even though each context has its own unique features, the etiology has many more commonalities. Thus, for program planners and policy makers intent on alleviating malnutrition to begin designing and implementing programs in their particular settings from scratch is strikingly inefficient. In the past decade, this point has been recognized, and documents that articulate processes for program implementation and evaluation have mushroomed. These "road maps" permit policy makers and program planners to capitalize rapidly on interest in addressing nutrition problems. The road maps also communicate a sense of feasibility by streamlining the complex processes of program design and evaluation. Thus, their use can reduce the likelihood that programs will be diffuse (too many inputs), will be culturally inappropriate, will have unrealistic expectations, and will have no possibility of sustainability and no plans for process or impact evaluation.

Demonstrating that nutrition programs are effective is key to translating scientific findings into policies and programs as well as to ensuring the continuity and expansion of funding.

Despite decades of nutrition programs, with identifiable successes, uncertainty about their effectiveness persists. The value of publishing solid process and outcome evaluations in the scientific literature in addition to project reports has only recently been recognized and cannot be overstated. Whereas outcome evaluations provide data on program effects, process evaluations provide key information to maintain quality assurance and to support the plausibility of key outcomes. Consensus is growing on the need to evaluate a package of services rather than use complex strategies to tease apart the effects of specific program elements. Well-designed programs with process evaluation efficiently provide this information.

Although following standard scientific approaches to establish program effectiveness has enabled progress in many interventions, alternative designs can and should be used for this purpose. Scientists traditionally argue that randomized controlled trials are needed to establish causal evidence of effectiveness and that multiple trials are needed in diverse settings, perhaps followed by pooled or meta-analyses to provide summary estimates. Others argue that designs that provide plausible evidence of program effects or adequate information to support continued funding should be recognized as valid by funders and publishers in refereed journals and should be implemented more broadly (Victora, Habicht, and Bryce 2004).

## RESEARCH AND DEVELOPMENT AGENDA

Despite progress, much work remains unfinished. Other chapters focus on research and development needs in relation to packaging services, scaling up, and ensuring sustainability. Here the focus is on research for strengthening the database for policy making.

Gaps in knowledge remain with respect to recognized strategies for intervention programs. Often information on intervention efficacy exists, yet little scientific literature on program effectiveness is available. Key gaps include the following:

- evaluation of the effectiveness of national iron fortification programs to reduce iron deficiency anemia
- implementation and evaluation of the effectiveness of iron supplementation programs for young children
- evaluation of the effect on child mortality of multifaceted programs to reduce child undernutrition
- evaluation of the effectiveness of programs based on the new guiding principles for reducing undernutrition and micronutrient malnutrition in young children
- implementation and evaluation of the effectiveness of food-based strategies to reduce micronutrient malnutrition
- implementation and evaluation of the effectiveness of early postnatal vitamin A supplementation to reduce infant mortality.

Costing studies should accompany the evaluations to allow estimates of cost-effectiveness for decision making.

Because of the logistical difficulties in developing fortification approaches in settings with little industry infrastructure, alternative fortification approaches are needed, such as micronutrient sprinkles or foodlets. In addition, operational research is needed to develop, implement, and evaluate programs to improve zinc status. Never has so much evidence been amassed on the consequences of a deficiency disorder without programmatic application. The challenge now is to develop and implement programs for preventing and treating zinc deficiency and to evaluate their effectiveness for child growth, health, and survival. The International Zinc Nutrition Consultative Group (2004) has laid out a research agenda with these aims in mind.

## CONCLUSIONS

Undernutrition is a major cause of death and disability in young children. When ranked among other causes, growth faltering and micronutrient deficiencies figure prominently, both because they are prevalent and because their consequences are devastating. Not included in the numbers, however, are the losses of lifetime productivity associated with early malnutrition and the resources that must be allocated to confront the developmental and morbidity consequences of child malnutrition, which last a lifetime.

Success has been achieved in preventing and controlling iodine deficiency, and palpable progress has been made in the past 20 years in correcting vitamin A deficiency and promoting breastfeeding; however, for iron, articulated goals have not been translated into programs, and the problem has remained the same or worsened. Zinc deficiency is now recognized as an important new challenge.

As shown here, solid evidence shows that nutrition programs can be effective at addressing nutritional problems in young children. Increasingly available cost data, when combined with outcome evaluations, demonstrate that nutritional interventions rank favorably in terms of cost-effectiveness when compared with competing interventions. The case that further investment in nutrition interventions is warranted is thus compelling.

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