Long-term consequences of undernutrition on physical and cognitive development

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Introduction

Undernutrition, or protein-energy malnutrition as it is often called, may occur at any stage in life. Most often, however, it is a symptom of developmental impairment that results from causes primarily operating during intrauterine life and the first three years. It kills nearly half of all children in developing countries and leaves those who survive functionally disadvantaged for life. In this paper, the focus is on studies from developing countries that assess effects of childhood undernutrition on physical and cognitive development at school age, adolescence and adulthood. This is not because effects during early childhood are unimportant or unknown. Rather, it is because less is known and written about long-term consequences.

Nature of undernutrition

Protein-energy malnutrition (PEM) is understood generally to include severe clinical malnutrition (kwashiorkor, marasmus, and mixed forms) and the common forms of growth failure, often called mild and moderate forms of malnutrition. The term undernutrition is preferable to PEM because its causes include more than just deficiencies in energy and protein. UNICEF’s conceptual framework recognizes three underlying classes of causal factors of undernutrition that operate at the level of household or family: i) food, i.e. insufficient access to food; ii) health, i.e. poor water, deficient sanitation, and inadequate health services; and iii) care, i.e. inadequate maternal and child care practices [1]. These underlying causal factors lead to undernutrition through two immediate mechanisms at the level of the individual: inadequate dietary intakes and disease. Dietary intakes may be deficient in amount and/or quality. Most often, deficiencies in energy will occur in conjunction with deficiencies in essential amino acids, vitamins and minerals. Moreover, diarrhoeal and respiratory infections, common problems among children in poor countries, interact with inadequate dietary intakes to cause undernutrition, disability and death.

In poor countries, newborns begin life compromised. In the least developed countries, around 23% of newborns have low birth weight (LBW, <2500 g) compared to about 6% of newborns in industrialized countries [1]. In South Asia, including countries such as India and Bangladesh, around 33% of newborns have LBW. The principal cause of LBW in developed countries is prematurity; however, the greater LBW prevalence of developing countries is explained by the greater frequency of intrauterine-growth-retarded (IUGR) infants [2]. IUGR babies are full-term neonates who suffered from chronic undernutrition in utero, a condition whose causes include small maternal body size, poor dietary intakes during pregnancy, and perhaps anaemia [3].

During the first two to three years of life, children are at great risk of experiencing undernutrition, including growth failure, anaemia, and vitamin A deficiency [4]. Several reasons explain this propensity. Young children have greater relative (i.e. per unit of mass) nutritional needs, in part because they are growing faster than at any other later point in life, including adolescence. At the same time, young children have relatively small
gastric capacities and thus require frequent meals with rich energy and nutrient concentrations; however, in many poor countries, children receive infrequent, dilute or bulky diets of low nutritional value. Immature immunological systems and high exposure to infectious agents account for the high frequency of diarrheal and respiratory infections.

Another reason for the vulnerability of small children is their complete dependence on others for care. The absence of breast-feeding in settings of poverty and poor environmental sanitation, denies the child the psychological, nutritional and anti-infective advantages of human milk over other modes of feeding and represents an extreme form of poor care. These factors, along with IUGR, account for the fact that many nutritional problems are at their peak of incidence and severity in early childhood.

The role of infections as causes of mortality has been recognized historically and that of nutrition ignored. Fortunately, undernutrition is now recognized to be associated with over half of child deaths in developing countries [5]. It is the combined influence of undernutrition and infection that explains the high mortality found in developing countries.

A number of basic causes of undernutrition operate at the societal level and these include the many dimensions of what is commonly called “poverty” [1]. These may include low incomes, illiteracy, discrimination and low social status, particularly of women. This context of poverty often magnifies the effects of undernutrition on human development. For this reason, the effects of undernutrition on development cannot be understood in isolation from poverty.

**Measuring cognitive development**

Whereas it used to be thought that undernutrition affected cognition largely by damaging the brain during sensitive periods of development, many other mechanisms, in addition to organic damage, are now recognized as important [6]. For example, nutrition may affect cognitive development in children by limiting physical activity and reducing interaction with other people and with the environment. Pollitt et al. [7] propose that an important factor in child development may be the lowered expectations of adults caused by an undernourished child’s younger appearance, the result of retarded physical growth and delayed development of motor skills. Timing is crucial for most of the proposed mechanisms which link undernutrition to poor cognition. Damage to the brain is more likely during the rapid phases of growth that occur during the prenatal period and infancy. Apathy and withdrawal, whether caused by diet, infection or both are more of a problem in young children. Finally, growth retardation occurs primarily in early childhood.

An eclectic array of tests, both general and single-dimensional psychometric tests are used to measure intelligence in the studies reviewed for this report. General tests often include verbal and non-verbal items, yielding subscores on an overall score, that is a general factor (“g”), or intelligence quotient (IQ). Examples of general tests include the Weschsler and Stanford-Binet tests. General tests may involve defining words, completing a series of pictures, or identifying words that do not belong in a set [8]. Single-dimension tests include the Peabody Picture Vocabulary Test (PPVT), which measures verbal intelligence in children, and the Raven’s Progressive Matrices, a non-verbal, untimed test that requires inductive reasoning about perceptual patterns [8]. Although performance may be correlated across general tests and among subscores, it is difficult to make generalizations across studies that use different tests. These tests also contain bias, which means that cross-cultural equivalency of psychometric performance should not be assumed. However, intracultural relationships between performance and undernutrition, that are emphasized in studies of undernutrition and cognition, may be less subject to bias. Also, some researchers use achievement tests, such as reading, and others focus on behavior, particularly in classroom settings. Measures of behavior include level of activity in children, attention, and interpersonal skills and relationships.

**Sources of evidence**

Several kinds of studies are considered below, beginning with follow-up studies of survivors of severe, clinical malnutrition. These studies perhaps underestimate the full importance of undernutri-
tion because the comparison groups are siblings or playmates, who also are often undernourished but to a lesser extent. These studies are also difficult to interpret because of the difficulty of matching cases and controls on all key confounders. For this reason, experimental studies, where nutrition is manipulated to prevent undernutrition, offer the strongest kind of evidence but, unfortunately, few such studies exist. Of these studies, the most comprehensive is the Follow-up study of the Institute of Nutrition of Central America and Panama (INCAP) [9].

Other types of studies might be said to provide “indirect” evidence. This is because physical growth, the hallmark consequence of undernutrition, is used as a proxy of undernutrition. Although a good indicator of nutritional status in children, physical growth is a phenotypic expression of the interaction of genetics and the environment. The environment includes many factors other than nutrition. Hence the term “indirect.” Studies of this type include reviews of the consequences of IUGR and of postnatal growth retardation.

Finally, it is possible to obtain useful information from studies of the long-term status of children adopted into good environments. Such children typically suffer considerable psychological as well as nutritional insults prior to adoption. Thus attributing sequelae to undernutrition in these studies, rather than to psychological factors, is often difficult.

**Follow-up studies of severely malnourished children**

A minority of children, usually less than 5-10%, exhibits severe clinical malnutrition in developing countries. Such cases are viewed as the extreme form of malnutrition, the “tip of the iceberg,” with the “invisible” portion of the iceberg made up of growth-retarded children without clinical signs. Survivors of severe PEM almost always have deficits in cognitive functioning and in school achievement compared to matched neighbourhood children, classmates, or siblings [10-12]. The degree of deficit varies, but many studies show a difference of 15 or more IQ points. Deficits are reduced when cases are compared to siblings rather than to matched controls [12].

Recent reviews are less certain that marasmus leads to greater deficits than does kwashiorkor [10, 12]. It has been suggested that growth failure, rather than the clinical definition of marasmus or the presence of oedema, is the most important predictor of poor cognitive and behavioral development [13].

The Barbados study of Galler and colleagues [14, 15] and that of Richardson et al. [16] in Jamaica demonstrate that severe malnutrition affects the behavioral and cognitive development of children. Jamaican schoolboys who were severely malnourished in infancy were less liked by siblings and more unhappy at school than were classmates of the same age and sex. Affected children also behaved immaturely more often and were clumsier than their classmate controls. Children with a history of severe malnutrition were either more highly active or lethargic than their classmates and were more often withdrawn, solitary, or unsociable [16]. Previously severely malnourished children in both Barbados and Jamaica had poorer relationships with classmates and teachers, exhibited a greater degree of attention deficits, and received poorer grades.

Despite such findings, it is often difficult to attribute unequivocally cognitive, behavioral, or physical effects to severe malnutrition. Wide-ranging disadvantages in the homes and families of undernourished children make it nearly impossible to match index and comparison children [12]. Just as these factors may cause both malnutrition and poor development, other factors associated with malnutrition directly may influence cognition or behavior. For example, severe malnutrition often leads to hospitalization not controlled for in most studies. Hospitalization itself could influence cognition [12].

In the first long-term follow-up of severely malnourished children who participated in a programme to stimulate cognitive development, Grantham-McGregor and colleagues [17] controlled for hospitalization by selecting three groups of children for the study: eighteen nonintervened, severely malnourished children (NIM); twenty-one intervened, severely malnourished children (IM); and twenty-one nonmalnourished children with acute diseases, the control group (C). All children had birthweights of more than
2,500 g and all were hospitalized in the University Hospital of the West Indies, Kingston, Jamaica, between 6 and 24 months of age.

The children were tested up to 14 years after their discharge from the hospital using the Griffiths Test (up to 6 years after discharge), Stanford-Binet Test (7, 8 and 9 years), and the WISC (14 years) (Fig. 1). Although the control children (C) performed low in comparison with children from industrial countries, they were clearly superior to children from the two formerly malnourished groups NIM and IM. Children of the IM group, who had participated in a stimulation programme, showed performance levels between children of the C and NIM groups. Similar differences showed up in reading, spelling, arithmetic, and global scores on the Wide Range Achievement Test (WRAT) and in the Peabody Picture Vocabulary Test.

This Jamaican study showed that severe malnutrition had a very marked effect on cognitive development and that this was not due to hospitalization. This study also demonstrated that an intervention using psychosocial stimulation during home visits every 1 or 2 weeks for 3 years improved intellectual performance and school achievement. Stimulation included toy demonstrations and encouragement to mothers to increase verbal interaction. However, stimulation alone was not able to erase all of the effects of severe malnutrition.

The INCAP food supplementation trial

The INCAP studies have taken place in four villages located in the “Ladino”, or Spanish-speaking, eastern part of Guatemala. In 1968, two pairs of matched villages were selected from among dozens; one pair of villages each had about 900 people and the other about 500 each [9].

A longitudinal supplementation trial between 1969 and 1977 randomly assigned one large and one small village to receive a nutritious supplement; the other two villages received a low-energy drink. All four villages received similar medical care from auxiliary nurses under the supervision of a physician. The nutritious supplement (Atole) was a high-protein, high-energy gruel served hot, twice a day in cups and each provided 163 kcal and 11.5 g of high-quality protein. Anyone in the village could participate, but consumption, including additional servings and leftovers, was recorded carefully only for women who were pregnant or breast-feeding and for children 7 years old or younger.

To investigate the effect of protein supplementation on mental development, it was necessary to control for the socialization effects of attending a feeding center. This involved duplicating the setting in the control villages, including measurement of consumption. Control villages received a low-energy drink (Fresco). It had no protein but contained sugar (a cup provided 59 kcal). A number of vitamins and minerals were added in equal concentrations to both types of supplement.

The investigators recorded the household structure, composition, and socio-economic status for every family involved in the longitudinal study. Women were monitored during pregnancy, and data were collected on maternal characteristics and pregnancy outcomes, including birthweight. Data were collected periodically from children on a variety of aspects including growth and maturation, home diet consumption, frequency and duration of illness, and psychological development. Data on psychological development included batteries of infant and preschool tests [18].
A follow-up of participants in the longitudinal study was conducted during 1988-89 [9]. The subjects of the Follow-up study were longitudinal participants who had been 7 years old or younger at any point during the feeding experiment. Out of 2,169 such subjects, 1,574 (74%) were examined at ages ranging from 11 to 27 years. Of those examined, 81% were nonmigrants (n = 1,278), and 19% were migrants (n = 296). Data collection included social, economic, and demographic information about subjects and their families as well as subjects’ body size and composition, skeletal maturation, hand strength, work capacity, school attendance history, and intellectual performance.

Many publications address the effects of supplementation on preschool development in the INCAP studies [7, 18]. Children who received Atole prenatally and up to 24 months of age had significantly higher scores on the motor scale at 24 months than did children who received Fresco; no interactions with treatment were detected. A factor analysis applied to the preschool battery generated two factors, general and memory [7]. After adjustment for sex, attendance and socioeconomic status, Atole subjects performed better in the general factor at 4 and 5 years of age; no differences were observed in the memory factor. Atole had a greater effect on low-socioeconomic status subjects.

Effects of supplementation on physical growth also were found. Severe failure in growth (height 3 or more standard deviations below the reference mean) among children 3 years of age declined in Atole villages (n = 451), from 45% in 1969 to 20% during 1976-77 when the supplementation programme ended; in Fresco villages (n = 429), however, the prevalence of severe failure in growth remained about 45% throughout the period [19]. Multivariate analyses demonstrated that only Atole exposure and intake in the first 3 years of life improved growth rates. Supplementation after 3 years did not affect growth [20]. A possible reason for the different effects by age is that growth in this population appears to fail only in the first 3 years of life. After 3 years, children who received no previous supplement grew as well as children in the US reference population.

Long-term effects of the nutrition intervention included effects on body size and composition [21]. Adolescents who were supplemented with Atole during the first three years of life were taller and had a greater fat-free mass than those who received Fresco; these effects were greatest in females. However, there was some attenuation of the effects observed at age three. In other words, there was a small degree of catching up in Fresco villages with respect to Atole villages. The cut-off point of less than 149 cm, equivalent to a height of 4 ft 11 in, is often used as a criterion of obstetric risk in women [22]. Of those females over 16 years of age who had been exposed to the supplements from birth to three years of age, 49% of the Fresco subjects had very short stature compared to 34% of Atole subjects. Little or no growth in height occurs in Guatemalan adolescents after 16 years [23].

Differences in fat-free mass also were found. Females from Atole villages had 2.1 kg more fat-free mass than females from Fresco villages. These differences are equivalent to an effect size of about 0.5, i.e. equal to a positive shift of 0.5 SD. Work capacity was improved significantly in subjects who were exposed to the supplements in their first three years of life, but only in males [24]. Males who received Atole had maximal oxygen consumptions (VO₂ max) that were 0.3 l/min greater than those of subjects who received Fresco; the difference is equivalent to about 0.7 SD. No effects on maturation were found. Menarche was 13.8±1.2 years in Atole villages compared to 13.7±1.4 in Fresco villages [25].

In contrast to the weak findings observed in the preschool period in regards to cognitive development, supplementation with Atole had strong and positive effects in adolescence and young adulthood on the results of psychoeducational tests of knowledge, numeracy, reading, and vocabulary [7, 26]. The Atole-Fresco differences found in preschool children were less than 0.2 SD compared to differences of around 0.6 SD in adolescence. Exposure to Atole also was associated with a faster reaction time for information processing tasks. One of the most interesting findings was the strong interaction between supplementation and socioeconomic status and education. Indeed, Atole had a greater impact than Fresco on low-socioeconomic status children and its benefits were magnified when children attended school.
Other intervention studies

The Cali, Colombia, study found cognitive ability in 7-year-old children improved due to stimulation and food supplementation [27]; two years after the study, effects of the intervention were still evident, although reduced [28]. A study in Mexico suggested that food supplementation leads to better, long-term performance [29]. Despite a weak design, small samples, and deficient analyses, the 24-year duration of the study and the breadth of the information collected are impressive. The study began in 1968 in the village of Tezonteopan. Unsupplemented women and newborns were recruited during the first year of the study. Supplemented cases were women who became pregnant during the second and third years. A nutritious drink was given twice daily to mothers as the food supplement. Children were supplemented from 12 weeks to 10 years. The type of supplement changed with age; for example, milk was given to young children, and milk and sandwiches were given to children starting at 4 years. Supplementation benefitted infant behavior, child IQ, behavior at school, school performance, and adult body size.

Intrauterine growth retardation

Intrauterine growth retardation has long-lasting effects on physical size. Curiously, differences in adult body size between IUGR and non-IUGR subjects are similar in developed and developing countries, about 5 cm in height and 5 kg in weight [30].

According to Hack [31], studies of the long-term effects of IUGR on mental performance and behavior provide varying and often conflicting results. The many reasons include the heterogeneity of the populations that have been studied, variations in the quality of neonatal care provided, and the fact that there are many postnatal conditions that may confound outcomes.

Follow-up studies generally report overall normal intelligence with a trend to lower IQ scores among IUGR subjects [31]. This is estimated to be about 6 IQ points, or a third of a standard deviation [32]. Hack notes that “the rates of minimal cerebral dysfunction, as evidenced by learning and subtle neurological and behavioral problems in IUGR children with normal intelligence”. Outcomes are poorer if IUGR is extreme and accompanied by reduced brain growth and if hypoxic ischemic injury occurs [31].

All of the studies reviewed by Hack [31] are from developed countries. The only report from a developing country is from the INCAP Follow-up study. Its findings were that growth during infancy, rather than birthweight, was related to adolescent performance on cognitive and achievement tests [33].

Postnatal growth retardation

Postnatal growth retardation in developing countries typically begins at about 3 to 6 months and ends around 2 to 3 years of age. After this age, physical growth, even in poor societies such as rural India, is likely to be as observed in well-nourished children [23]. Figures 2 and 3, which use data from the INCAP Follow-up study, make the point that postnatal growth retardation is almost exclusively a problem of early childhood. Figure 2 shows the median lengths for Guatemalan boys compared to US reference children [34] as well as to children of Mexican origin living in

Fig. 2: Medial length in males 0.5-36 months old: rural Guatemalans and Mexican-Americans compared with US reference population (from Martorell et al. [23]).
the United States. This US reference is used worldwide, as recommended by the World Health Organization [35]. At birth, values are well below the 50th percentile, reflecting the effects of intrauterine growth failure. The median lengths of Guatemalan boys decline rapidly after 3 months of age and end up well below the 5th percentile of the reference population by 36 months of age. The fact that Mexican-American children, who are of the same ethnic origin as Guatemalans, have values close to the 50th percentile indicates that differences with respect to the reference population are due to environmental and not genetic differences.

Cross-sectional data on these same children at older ages are compared to values from US [36] and Mexican-American populations in figure 3. Values at ages 11 through 25 of Guatemalan males continue to be below the 5th percentile of the US distribution. Interestingly, median heights of Mexican-Americans decline during puberty and end up near the 25th percentile at young adulthood. Because malnutrition and infection are unlikely to explain the patterns in growth of children of Mexican-Americans living in the US, genetic causes may be responsible. If this is the case, some degree of catch-up in height can be said to occur in Guatemalans.

Follow-up studies from India suggest that there is some catch-up in growth during adolescence but that, as in Guatemala, most of the growth retardation in height observed in early childhood persists into adulthood [37-39]. Also, in unusual populations with extremely delayed maturation, the degree of catch-up in growth may be large enough to erase most of the pre-pubertal deficit [40, 41].

The INCAP Follow-up study is the only study available from developing countries which can provide information about the long-term consequences of postnatal growth failure [42]. The distribution of height at three years of age was divided into three groups depending upon the degree of stunting as measured in standard deviation units (Z-scores): severe, below –3; moderate, between –3 and –2; and mild, above –2. Among adults, defined as 18 years or older, the differences between adults with histories of severe and mild stunting as children, that is between the extremes in early childhood, were 10.4 cm in height, 8.5 kg in body weight and 7 kg in fat-free-mass. For women, the corresponding values were 8.3 cm, 5.9 kg and 5.0 kg, respectively. Differences were also found in terms of muscle circumferences, frame size and hand strength. Z-scores in height at age 3 were related to intellectual performance measures of adolescents and adults. Stronger relationships were found among males. In males, growth failure at 3 years of age was associated with late entry into school, fewer years of schooling, and with less intelligence and poorer performance in tests of general knowledge, numeracy, reading and vocabulary. Significant relationships in females were with age at school entry and reading. These analyses controlled for maternal schooling, family socio-economic characteristics and other variables. Clearly, stunting at three years of age leads to reduced body size and strength among adults. For reasons that are not clear to us, stunting is powerfully associated with intellectual competence only among males.

Adoption studies

Follow-up studies of developmentally retarded children adopted into good environments are the last type of evidence reviewed. Two studies of Korean orphans adopted by US families illustrate...
that survivors of malnutrition perform better if their fortunes improve [43, 44]. The definition of malnutrition in these studies was solely anthropometric. The height and weight of girls (boys were excluded from the study because fewer were available) were compared to Korean reference data, and three study groups were formed: “malnourished” (below the 3rd percentile), “moderately malnourished” (3rd through the 24th percentile), and “well nourished” (at or above the 25th percentile). Both height and weight had to agree in classification for the children to be considered.

The first study included children who were adopted before the age of 2 years, and the second study focused on children adopted between 2 and 5 years of age. A portion of the children were tested at school age (grades 1-8) several years after adoption. The results, shown in figure 4, indicate that adopted children performed better as a group than the general US population on standardized tests of intelligence. This finding shows that severe growth failure in early childhood did not necessarily lead to completely irreversible low performance. In both studies, performance was a function of former nutritional status; for example, the difference between “malnourished” and “well-nourished” girls, the extreme nutrition groups, was 10 IQ points in both studies. Performance also depended on the child’s age at adoption; for example, children adopted at younger ages performed 6 IQ points better than children adopted at ages 2-5 years. Overall, these two studies suggest that: i) adoption leads to performance improvements, despite severe growth failure; ii) early undernutrition has an important residual effect on performance; and iii) the earlier adoption occurs, the better the results will be.

A study of Romanian children adopted by British families also points to the importance of age at adoption as well as to the resilience of young children [45]. These children suffered an extreme degree of psychological privation as well as severe growth failure (i.e. height Z-scores were about –2 at entry in the UK) prior to adoption. Catch-up in cognitive development was nearly complete at 4 years of age for those children who came to the UK before the age of 6 months. For those children placed between 6 and 24 months of age, the cognitive deficit was about 1 standard deviation. Both groups had much reduced rates of retardation in height at 4 years of age but head circumferences remained smaller. It is difficult in this study to disentangle psychological from nutritional privation. The fact that age, but not weight at entry, was a predictor of developmental outcomes suggests to the authors that psychological privation is more important. Longer-term follow-up of these children and inclusion of children adopted after 24 months will enhance the contribution of the research about this population. Nonetheless, the results to date show remarkable degrees of recuperation in children adopted at a young age.

A small study in Chile confirms that adoption leads to substantial gains in performance for children who were severely malnourished at an early age. At 8-9 years of age, previously malnourished children who were adopted, mostly by middle-class families, had an IQ of 97 compared to values of 81 in children who remained institutionalized and 83 in children who were returned to their biological families. Differences were consistent across WISC subscales. Years earlier, the develop-
mental quotient had been similar across groups at admission and discharge from a nutritional recuperation center [46].

Adoption at older ages poses two problems. One is that there may be less ability to recuperate cognitive function as suggested by the Korean studies. A second concern, particularly at ages 4-9 years in girls, is that the rapid increase in weight which usually occurs after adoption may lead to increased “circulating levels of free active steroids which could prime the hypothalamic centers and cause precocious stimulation of the hypothalamic-pituitary-gonadal axis” [47]. Precocious puberty is reported by Indian girls adopted by families in Italy [45] and Sweden [48]. By accelerating maturation, the duration of growth is shortened and final height may end up being no different than observed in rural India, despite the initial period of catch-up growth.

Conclusions

Any consideration of the long-term consequences of undernutrition on physical growth and cognitive development must take into consideration the following aspects: i) the causes of undernutrition are multiple and include dietary and non-dietary factors; ii) these causes operate primarily during critical periods of development which include intrauterine life and the first two to three years of life; and iii) undernutrition occurs within a social context of poverty.

Does undernutrition affect the development of children growing up in the poor countries of the world? The answer is clearly yes. IUGR is associated with a deficit of about 6 IQ points and severe, clinical malnutrition is associated with a deficit of as much as 15 IQ points. Behavior is also affected in subtle ways that adversely affect relationships with classmates and teachers and that inhibit learning. Growth failure, a marker of undernutrition along the severity spectrum, from severe to mild, is associated with deficits in intellectual performance.

These effects on cognitive development can be reversed through interventions early in life. Enrichment of the home environment reverses some of the dysfunction associated with severe malnutrition and adoption leads to remarkable degrees of recuperation. The literature suggests that the earlier the adoption occurs in life, the better the results will be. It is not known, on the other hand, what happens if adoption occurs after age five. Finally, the INCAP Follow-up study provides evidence that nutritional improvements in early childhood lead to improved intellectual performance in adolescence, particularly among poorer children and among those who go to school.

What is the significance of the effects of childhood undernutrition? Intellectual performance is a key component of “human capital” in all societies [32]. It predicts acceptance of new ideas and innovations, choice of occupation and income. Work capacity and strength are assets for the arduous lifestyles of agricultural societies [24]. In women, stunting is associated with increased risk of cephalopelvic disproportion and delivery complications [22, 42]. Thus undernutrition definitely limits physical and intellectual capacity in the adolescent and the adult. These limitations have deleterious consequences for individuals, families and societies. The prevention of undernutrition precludes those adverse outcomes and thus enables long-term economic benefits and increases the welfare of nations.

References

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24. Haas JD, Martínez EJ, Murdock S, et al. Nutritional supplementation during the preschool years and physical work capacity in adolescent and young adult Guatemalans. J Nutr 1995;125:1078-89S.


