Health, Human Capital and Economic Growth

Nutrition, Malnutrition and Economic Growth *

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Abstract: Malnutrition is widespread in the developing world. 12 or more million low-birth-weight births occur per year and 180 million children are characterized as malnourished. This chapter first assesses estimates of the extent of malnutrition in developing countries. It then surveys micro evidence about the productivity impact of improved nutrition in developing countries – from conception through infancy and childhood and into adolescence and adulthood. These gains may operate through many channels – increasing cognitive development, increasing physical stature and strength, inducing earlier school enrollment and more regular school attendance, inducing greater schooling and learning, and increasing adult productivity – as well as saving resources that otherwise would go towards dealing with diseases and other problems related to malnutrition. Finally the possible gains from different nutritional policy strategies and the policy bases for adopting such strategies are examined.

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1. Introduction

Malnutrition is widespread in many developing countries. It is estimated that at least 12 million low-birth-weight births occur per year and that around 180 million preschool children are malnourished. Redressing this serious health problem has long been justified on intrinsic grounds; better nutrition has been widely seen as being of value in its own right. Others also have emphasized the importance of health and nutrition for productivity and economic growth. That is, the emphasis is on the instrumental, rather than the intrinsic, value of good health and nutrition. This chapter adopts such an approach, but differs from many of the other contributions to this book in three ways. First, our focus is on the microeconomic rather than the macroeconomic evidence of the links between nutritional status and representations of productivity. We perceive that the microeconomic analysis is likely to be more informative about the possible causal effects of nutrition on productivity because of enhanced opportunities to address estimation problems as such endogeneity at the micro level and because aggregation problems may obscure non-linear relationships. Second, we emphasize that these links are multiple and heterogeneous. In particular, there are important links between health/nutrition and cognitive development and between health/nutrition and schooling so that in adulthood, health and nutritional status may have direct links to productivity as well as indirect links through their effects on education. Third, we emphasize that many outcomes that reflect nutritional status are cumulative and this has implications for the design of policies and interventions designed to ameliorate malnutrition.

This chapter begins with a brief explanation of the causes and measurement of malnutrition as well as estimates of the extent of malnutrition across developing
countries. It then surveys micro evidence about the productivity impact of improved nutrition in developing countries – from conception through infancy and childhood and into adolescence and adulthood. These gains may operate through many channels – increasing cognitive development, increasing physical stature and strength, inducing earlier school enrollment and more regular school attendance, inducing greater schooling and learning, and increasing adult productivity – as well as saving resources that otherwise would go towards dealing with diseases and other problems related to malnutrition. Finally the possible gains from different nutritional policy strategies and the policy bases for adopting such strategies are examined. Given links between improved nutrition and greater schooling as well as directly between nutrition and higher levels of productivity and incomes, such investments are instrumental in achieving additional outcomes valued by individuals and the societies within which they reside.

2. Understanding malnutrition

Indicators of nutritional status are measurements of body size, body composition, or body function that reflect single or multiple nutrient deficiencies. They can be divided into three broad groups depending on the type of information. The first group is anthropometry, the measurement of body size and gross body composition. The second group uses clinical examinations to detect signs and symptoms of advanced nutritional depletion. Examples are assessments of iodine deficiency by inspection and palpitation of enlarged thyroid glands. The third group uses laboratory methods to detect decreased levels of nutrients in body tissues or fluids, or decreased activity of an enzyme that is

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1 These are outcome indicators. In addition, measures of nutrient intake or access track some of the inputs that condition nutritional status.
nutrient-dependent. While malnutrition is often seen as a manifestation of some form of deprivation, it also refers to circumstances of excess such as obesity. Table 1, adapted from Morris (2001), describes the most common measures.

Table 1: Measures of nutritional status and their prevalence

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Interpretation</th>
<th>Most common means of reporting</th>
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<tr>
<td><strong>Group I: Anthropometric Indicators</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalence of low birthweight</td>
<td>An indicator of intrauterine growth retardation resulting from short maternal stature, poor maternal nutrition before or during pregnancy, infection and smoking.</td>
<td>Percentage of children with birthweights below 2500 grams</td>
</tr>
<tr>
<td>Prevalence of low height-for-age (stunting) in preschool or school-age children</td>
<td>Children’s skeletal (linear) growth compromised due to constraints to one or more of nutrition, health, or mother-infant interactions. This is an indicator of chronic nutritional deprivation.</td>
<td>Expressed as a z score or as the percentage of children stunted. Z scores are calculated by standardizing a child's height given age and sex against an international standard of well nourished children. A z score of -1 indicates that given age and sex, the child's height is one standard deviation below the median child in that age/sex group. Children with z scores below –2 are classified as stunted; with z scores below –3 are classified as severely stunted.</td>
</tr>
<tr>
<td>Prevalence of low weight-for-height (wasting) in preschool or school-age children</td>
<td>Children suffer thinness resulting from energy deficit and/or disease-induced poor appetite, malabsorption, or loss of nutrients. This is an indicator of transitory nutritional deprivation.</td>
<td>Expressed as a z score or as the percentage of children wasted. Z scores are calculated by standardizing a child's weight given height and sex against an international standard of well nourished children. Children with z scores below –2 are classified as wasted; with z scores below –3 are classified as severely wasted.</td>
</tr>
<tr>
<td>Prevalence of low weight-for-age (underweight) in preschool or school-age children</td>
<td>This is a composite measure of child nutritional status, reflecting both chronic and transitory nutritional deprivation. This is a Millennium Development Goal indicator.</td>
<td>Expressed as a z score or as the percentage of children underweight. Z scores are calculated by standardizing a child's weight given age and sex against an international standard of well nourished children. Children with z scores below –2 are classified as underweight; with z scores below –3 are classified as severely underweight.</td>
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2 This section draws heavily on ACC/SCN (2000), Gibson (1990) and Morris (2001) who provide very helpful introductions to these measures.
Prevalence of low body mass index in adults or adolescents

Adults suffer thinness as a result of inadequate energy intake, an uncompensated increase in physical activity, or (severe) illness. Expressed as Body Mass Index (BMI). BMI is calculated by dividing weight in kilograms by the square of height in meters. Individuals are considered to be chronically energy deficient if they have a BMI below 18.5, overweight if they have a BMI greater than 25, and obese if they have a BMI greater than 30.

<table>
<thead>
<tr>
<th>Group 2 Clinical Examinations or Group 3 Laboratory Methods</th>
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<tr>
<td>Prevalence of iodine deficiency</td>
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<tr>
<th>Group 3 Laboratory Methods</th>
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<tr>
<td>Prevalence of low hemoglobin (anemia) in preschool or school-age children</td>
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<tr>
<td>Prevalence of low hemoglobin (anemia) in nonlactating, nonpregnant women</td>
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Nutritional status is an outcome, reflecting the purposive actions of individuals given preferences and constraints. In behavioral models, an individual’s nutritional status often is treated as an argument in the welfare function of individuals or the households in which they reside (Behrman and Deolalikar 1988; Strauss and Thomas 1995). It is assumed that welfare increases as nutritional status improves, but possibly at a diminishing rate and, as suggested above, increases in certain measures of nutritional
status, such as body mass, may be associated with reductions in welfare beyond a certain point.

In allocating resources, household decision makers take into account the extent to which these investments will make both their children and themselves better-off in the future as well as currently. These allocations are constrained in several ways. There are resource constraints reflecting income (itself an outcome) and time available as well as prices faced by households. There is also a constraint arising from the production process for health outcomes, including nutritional status. This constraint links nutrient intakes – the physical consumption of macronutrients (calories and protein) and micronutrients (minerals and vitamins) – as well as time devoted to the production of health and nutrition, the individual’s genetic make-up and knowledge and skill regarding the combination of these inputs to produce nutritional status. There are interdependencies in the production of nutritional status and other dimensions of health; for example, malaria limits hemoglobin formation.

It is important to recognize that many nutritional outcomes are the consequence of cumulative processes that begin in utero. A number of maternal factors have been shown to be significant determinants of intrauterine growth retardation (IUGR), the characterization of a newborn who does not attain their growth potential. Most important are mother’s stature (reflecting her own poor nutritional status during childhood), her nutritional status prior to conception as measured by her weight and micro-nutrient status,

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3 Implicit in this framework is the assumption that parents or other decision makers are in agreement regarding investments in nutrition and that they are willing to pool their resources in order to undertake these investments. Where there is disagreement on the nature and the allocation of these investments, the ability of individual parents to impose their preferences – their bargaining power – also plays a role (Alderman, et. al.1995; Behrman 1997; Haddad, Hoddinott and Alderman 1997).
and her weight gain during pregnancy. Diarrheal disease, intestinal parasites, and respiratory infections may also lead to IUGR and where endemic (such as sub-Saharan Africa), malaria is a major determinant. In developed countries, smoking is also a significant contributor to IUGR. IUGR is measured as the prevalence of newborns below the 10th percentile for weight given gestational age (ACC/SCN, 2000). Because gestational age is rarely known, IUGR is often proxied by low birthweight (LBW). As of 2000, it is estimated that 11% of newborns, or 11.7 million children have low birthweight (ACC/SCN, 2000).\footnote{Other estimates are higher. For instance, Ceesay, et al. (1997) claim that there are over 22 million LBW children per year. Blanc and Wardlaw (2002) discuss aspects of estimates from micro data.}

In pre-school and school age children, nutritional status is often assessed in terms of anthropometry. “The basic principle of anthropometry is that prolonged or severe nutrient depletion eventually leads to retardation of linear (skeletal) growth in children and to loss of, or failure to accumulate, muscle mass and fat in both children and adults” (Morris, 2001, p.12). A particularly useful measure is height given age as this reflects the cumulative impact of events affecting nutritional status that result in stunting. As of 2000, it is estimated that one child in three under the age of five, 182 million children in all, are stunted (ACC/SCN, 2000).

A number of factors contribute to poor anthropometric status in children. One is low birthweight; a number of studies show a correlation between low birthweight and subsequent stature though, in the absence of any subsequent intervention, not between low birthweight and growth (Ashworth, Morris and Lira 1997; Hoddinott and Kinsey 2001; Li, et. al. 2003; Ruel 2001). In addition, the first two years of life pose numerous
nutritional challenges to the newborn. Growth rates are highest in infancy, thus adverse factors have a greater potential for causing retardation at this time. Younger children have higher nutritional requirements per kilogram of body weight and are also more susceptible to infections. They are also less able to make their needs known and are more vulnerable to the effects of poor care practices such as the failure to introduce safe weaning foods in adequate quantities. Evidence from numerous studies clearly indicates that the immediate causes of growth faltering are poor diets and infection (primarily gastrointestinal) and that these are interactive (Chen 1983; NAS 1989). For these reasons almost all the growth retardation observed in developing countries has its origins in the first two to three years of life (Martorell 1995).

A growing body of evidence indicates that growth lost in early years is, at best, only partially regained during childhood and adolescence, particularly when children remain in poor environments (Martorell, et al. 1994). For example, Martorell (1995, 1999), Martorell, Khan and Schroeder (1994) and Simondon, et. al. (1998) all find that stature at age three is strongly correlated with attained body size at adulthood in Guatemala and Senegal. Hoddinott and Kinsey (2001) find that children who were initially aged 12-24 months in the aftermath of a drought in rural Zimbabwe in 1994/95 had z scores for height-for-age that are about six tenths of a standard deviation below that of comparable children not exposed to this drought when measured at ages 60-72 months. However, older children did not suffer such permanent consequences; this is consistent with evidence that child development has “sensitive” periods where development is more receptive to influence and that during such periods, some shocks may be reversible while others are not (Bornstein 1989; Yaqub 2002). Alderman, Hoddinott and Kinsey (2002)
find that in rural Zimbabwe, children exposed to the civil war or to the 1982-84 droughts were found to have lower stature compared to siblings not affected by these shocks when remeasured in 2000. The magnitudes of these shocks are large; exposure to the 1982-84 drought reduced stature in late adolescence by 2.3 centimeters.

It is also important to recognize that “severe malnutrition in early childhood leads to deficits in cognitive development … if the children return to poor environments” (Grantham-McGregor, Fernald and Sethuraman 1999, p.66. See also Pollitt 1990). Although many studies from developed countries fail to show difference in development levels for children with low birth weights (LBW), there are few longitudinal studies from developing counties from which to generalize (Hack 1998). More recent studies that test the impact of low birth-weight over a wide range indicate that the relationship between birth weight and cognitive function carries into the range of normal weights even in developed countries (Richards, et al. 2001; Matte, et al. 2001). Even if, as Richards, et al. observe, this association between birth weight and cognitive ability partially attenuates over time – they followed a cohort for 43 years – the significant difference in function at age 8 affects educational attainment.

Malnourished children are found to score poorly on tests of cognitive function, have poorer psychomotor development and fine motor skills. They tend to have lower activity levels, interact less frequently in their environments and fail to acquire skills at normal rates (Grantham-McGregor, et. al. 1997, 1999; Johnston, et. al. 1987; Lasky, et. al. 1981). Controlled experiments with animals suggests that this may occur because malnutrition results in irreversible damage to brain development such as that associated with the insulation of neural fibers (Yaqub 2002).
One such deficiency that has been studied both in laboratory and in epidemiological studies is iodine deficiency, which adversely affects the development of the central nervous system. A meta-analysis indicates that individuals with an iodine deficiency had, on average, 13.5 points lower IQs than comparison groups. While interventions have shown that provision of iodine to pregnant women can reduce this gap, the provision of iodine to school aged children does not appear to reverse earlier damage (Grantham-McGregor, Fernald and Sethuraman 1999b). Prevalences of iodine deficiency are not available by age; it is estimated that altogether, globally more than 700 million people are affected.

Adequate iron intake is also necessary for brain development. The studies reviewed by Grantham-McGregor, Fernald and Sethuraman (1999b) do not indicate that subsequent interventions to rectify these deficiencies reverse this damage. Given that more than 40 per cent of children aged 0-4 in developing countries suffer from anemia (ACC/SCN 2000) this could be a major global contributor to poor schooling outcomes. Anemia in school aged children may also affect schooling whether or not there had been earlier impaired brain development. Supplementation trials for school age children consistently indicate improved cognition, although this is less regularly observed with interventions aimed at deficient younger children.

Reduced breastfeeding – an effect of low birth weight as well as a common cause of childhood malnutrition - is also a well-documented influence of cognitive development, even in developed countries (Grantham-McGregor, Fernald and
Sethuraman 1999a). This is in keeping with the prevailing view that very young children are most vulnerable to impaired cognitive development.\(^5\)

### 3. The links between malnutrition and economic productivity

We now turn to the micro evidence about the productivity impact of improved nutrition in developing countries – from conception through infancy and childhood and into adolescence and adulthood. These many channels through which these gains may operate are grouped in the following way: the saving of resources that otherwise would go towards dealing with diseases and other problems related to malnutrition; the direct gains arising from improvements in physical stature and strength as well as improved micronutrient status; and the indirect gains arising from the links between nutritional status and schooling, nutritional status and cognitive development and the subsequent links between schooling, cognitive ability and adult productivity.

\(\text{a) The costs of poor nutrition}\)

One significant cost of malnutrition is higher mortality. The probability of infant mortality is estimated to be significantly higher for LBW than for non-LBW infants. Conley, Strully and Bennett (2003) conclude that intra-uterine resources competition – and, by inference, nutrition – explain a substantial portion of excess mortality of LBW children in the United States. In their study, an additional pound at birth led to a 14% decrease in mortality in the period between 28 days and one year for both fraternal and

\(^5\) One exception is provided by Glewwe and King (2001) who find that malnutrition in the second year of life had a larger impact on the IQs of Philippine school children than that in earlier periods.
identical twins. In contrast, the risk of death in the first 28 days was elevated 27% for each pound difference in weight for fraternal twins compared to only 11% for identical twins, implying a large role for genetic factors. Ashworth (1998) reviews 12 data sets including two from India and one from Guatemala, and concludes that the risk of neonatal death for term infants 2000-2499 grams at birth is four times that for infants 2500-2999 grams and 10 times that of infants 3000-3499 grams. Relative risks of post-neonatal mortality for LBW compared to the two respective groups were two and four times as large. These risk ratios translate into fairly large differences in mortality rates given the relatively high mortality rates in many developing countries. Using the data she reports for the Indian and Guatemalan samples, Alderman and Behrman (2003) estimate that the probability of an infant death (either neonatal or post-neonatal) drops by about 0.078 for each birth in the 2500-2999 grams range instead of in the 2000-2499 gram range.

When the impacts of poor pre-schooler nutrition are added to the effects of low birthweight, Pelletier, et al. (1995) venture the widely cited estimate that 56% of child deaths in developing countries at attributable to the potentiating effects of malnutrition (83% of this, due to the more prevalent mild to moderate malnutrition rather then the severe cases most commonly monitored). More recently, WHO (2002) has claimed that malnutrition contributes to 3.4 million child deaths in 2000 (60% of child deaths). Pelletier and Frongillo (2003) have recently employed data on changes in national
malnutrition rates and mortality to get a different perspective on this association, yet one that supports the earlier evidence on the association of mortality and malnutrition.\(^6\)

The availability of experimental evidence on the use of micronutrient supplements provides unambiguous evidence on the relationship of mortality and vitamin intakes in many environments including ones that show few clinical symptoms of deficiencies. The potential to reduce child deaths by distributing vitamin A on a 6 monthly basis is particularly dramatic; meta-analysis of field trials indicate that such provision of vitamin A can reduce overall child mortality by 25-35\% (Beaton, et al. 1993). Amongst adults, anemia is a particular concern for the health of women of child bearing age not only because of elevated risk of adverse birth outcomes but also because the risk of maternal death is substantially elevated for anemic women; over a fifth of maternal deaths are associated with anemia (Ross and Thomas 1996; Brabin, Hakimi and Pelletier 2001).\(^7\)

Beyond the issue of increased mortality, malnutrition increases the risk of illnesses that impair the welfare of survivors. This relationship between nutrition and both infections and chronic diseases can be traced through different parts of the life cycle. Children with low birth weights – reflecting a range of causes, not all of which are due to dietary deficiencies – stay longer in hospitals in circumstances where births occur in such

\(^6\) These associations do not, however, control for changes in infrastructure or income that may both affect mortality directly as well as influence nutrition nor can they indicate a counterfactual of the impact of improved nutrition on expected mortality. Guilkey and Riphahn (1998) use longitudinal data of Filipino children with controls for the endogeneity of nutrition and other health care choices. Their simulations indicate that children with two months without weight gain in the first year of life (about 10 percent of their sample) would have the risk of mortality elevated by 50\%. Similarly, the scenarios show that if a mother is unable or unwilling to adopt standard recommendations on breastfeeding the hazard of child mortality increases markedly. Care has to be taken, however, in interpreting the last association as causal because mothers may be less likely to be able to breastfeed infants who are at high risk.

\(^7\) As with associations of child mortality and nutrition, it is difficult to prove causality with these associations. Randomized controlled trials to substantiate the observational data would need to be large given the risk of maternal mortality and are generally considered unethical.
settings and have higher risks of subsequent hospitalization (Vitoria, et al. 1999). In addition, they use outpatient services more frequently than do normal children. For young children, in general, malnutrition leads to a vicious cycle with impaired immunity leading to infection with attendant loss of appetite and catabolism and, thus, an increased likelihood of additional malnutrition.

Increased morbidity has direct resource costs in terms of health care services as well as lost employment or schooling for the caregivers. The magnitudes of these costs differ according to the medical system, markets, and policies of a country. In developed countries the additional costs for the survivors can be substantial. Lightwood, Phibbs, and Glanz (1999) calculate the excess direct medical costs due to low birth weight in the United States attributed to one cause - maternal smoking - to be $263 M in 1995. For example, 75% of the $5.5-6 billion of excess costs due to LBW in the United States estimated by Lewit, et al. (1995) is due to the costs of health care in infancy. A further 10% of these costs are attributed to higher requirements for special education as well as increased grade repetition. Such requirements for special education or for social services are substantial in developed countries (Petrou, et al. 2001). While these costs may be far less in low-income countries where, for example, the majority of births occur outside a clinical setting, these lower medical costs associated with LBW come at the expense of higher mortality. In the absence of an educational system that can recognize and accommodate the individual needs of students, however, these costs are not incurred during childhood but rather in the form of reduced productivity in adulthood.

Undernutrition, particularly fetal undernutrition at critical periods, may result in permanent changes in body structure and metabolism. Even if there are not subsequent
nutritional insults, these changes can lead to increased probabilities of chronic non-infectious diseases later in life. The hypothesis that fetal malnutrition has far ranging consequences for adult health is bolstered by studies that track low birth weight infants into their adult years and document increased susceptibility to coronary heart disease, non-insulin dependent diabetes, high blood pressure, obstructive lung disease, high blood cholesterol and renal damage (Barker 1998). For example, while the various studies on the impact of the Dutch famine indicate few long-term consequences on young adults, more recent evidence shows that children whose mothers were starved in early pregnancy have higher rates of obesity and of heart disease as adults (Roseboom, et al. 2001). In contrast, children of mothers deprived in later pregnancy – the group most likely to be of low birth weight – had a greater risk of diabetes (Ravelli 1998).

The evidence for the fetal origins hypothesis is still being assessed. The fact that some consequences may not be observed until the affected individuals reached middle age is an important consideration for interpreting the range of evidence being assembled. There are few panels that follow cohorts this far and extrapolation from shorter panels or from less affluent cohorts with different life histories is currently necessary, albeit uncertain. In addition, there are at least two other explanations for the association between LBW and adult diseases. LBW may be an indicator of poor socioeconomic status. Low SES may have a causal impact on adult disease probabilities via other variables such as poor nutrition later in life or higher rates of smoking. If so, LBW may only be a correlate and not a causal variable. A different possibility is that LBW may be due to a genetic predisposition to insulin resistance. This would tend to account for a higher pre-disposition for adult diabetes and coronary heart diseases that reflects genetics
rather than aspects of the uterine environment that may be influenced by medical and nutritional interventions.\textsuperscript{8} Finally, even if there are the effects proposed in the fetal origins hypothesis, due to there long lag, the present discounted value of improvements due to prenatal interventions to offset them is not likely to be very large (Alderman and Behrman 2003; also see the end of Section 3).

In addition to the consequences of undernutrition, it is well known that poor diets can contribute heart disease, stroke and diabetes. Evidence that obesity as well as diseases commonly associated with over consumption are occurring among low income population, often side by side with under-nutrition has added to the challenge of addressing nutritional deficiencies (Doak, et al. 2000).

Lastly, malnutrition may have long-term consequences through the intergenerational transmission of poor nutrition and anthropometric status. Recall that there is considerable epidemiological evidence that stature by age three is strongly correlated with attained body size at adulthood. Taller women experience fewer complications during childbirth, typically have children with higher birthweights and experience lower risks of child and maternal mortality (Ramakrishnan, et al. 1999; World Bank 1993). However Behrman and Rosenzweig (2003) find that intergenerational birth weight effects are primarily genetic, not due to better nutrition in the womb.

\textit{b) Direct links between nutrition and productivity}

\textsuperscript{8} There is an additional aspect of the hypothesis of subsequent costs stemming from biological adaptation to deprivation \textit{in utero} that has a bearing on the estimation of the consequences of LBW. The implications of the hypothesis will be different if the consequences are a direct result of the deprivation compared to the possibility that they only manifest themselves if the deprivation is followed by relative abundance (Lucas, Fewtrell and Cole 1999, Cameron 2001). High rates of diabetes among Native Americans or Ethiopian immigrants to Israel, for example, seem to be an indirect effect of removal from an environment for which certain genes may once have been adaptive.
There is considerable evidence of a direct link between nutrition and productivity. Behrman (1993), Behrman and Deolalikar (1989), Deolalikar (1988), Foster and Rosenzweig (1993), Glick and Sahn (1997), Haddad and Bouis (1991), Schultz (1996), Strauss and Thomas (1998) and Thomas and Strauss (1997) all find that after controlling for a variety of characteristics, that lower adult height – a consequence, in part, of poor nutrition in childhood, is associated with reduced earnings as an adult. For example, Thomas and Strauss (1997) estimate the direct impact of adult height on wages for urban Brazil. While the elasticity varies somewhat according to gender and specification, for both men and women who work in the market sector a 1% increase in height leads to a 2-2.4% increase in wages or earnings.\(^9\) While their study is particularly sophisticated in the methodology used to account for labor selectivity and joint determination of health, this result is similar to others reported in the literature. Indeed, height is even a significant explanatory variable for wages in the United States (Strauss and Thomas 1998).

Nevertheless, the direct impact of height on wages is likely less than the impact of schooling on wages over plausible ranges for each,\(^{10}\) even if the indirect effect of height on wages mediated by the relationship between height and schooling in included.

Micronutrient status also has important productivity effects. In particular, anemia is associated with reduced productivity both in cross-sectional data and in randomized interventions (Li, et al. 1994; Basta, Karyadi and Scrimshaw 1979). The magnitude may depend on the nature of the task. For example, piece work may have greater incentives

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\(^9\) In related, rather grim work, Margo and Steckel (1982) found that the value of an American slave fell by roughly 1.5 percent for every reduction in height of one inch.

\(^{10}\) Strauss and Thomas (1998) point out that an illiterate man would need to be 30 cm taller than his literate coworker to have the same expected wage.
for effort while heavy physical labor may show greater increases in productivity, though anemia is nevertheless a factor in productive with relatively light work (Horton and Ross 2003).

c) Indirect links: nutrition, cognitive development, schooling and productivity

Poorly nourished children, as evidenced for example by low height-for-age, tend to start school later, progress through school less rapidly, have lower schooling attainment, and perform less well on cognitive achievement tests when older. These associations appear to reflect significant and substantial effects in poor populations even when statistical methods such as instrumental variables are used to control for the behavioral determinants of pre-school malnutrition.

There are three broad means by which nutrition can affect schooling. First, malnourished children may receive less schooling, because their caregivers seek to invest less in their education, because schools use physical size as a rough indicator of school readiness or because malnourished children may have higher rates of morbidity and thus greater rates of absenteeism from school. While delayed entry, the second way by which nutrition may influence schooling, does not necessarily lead to less completed education – although under a prevailing model of the returns to education this would be an expected consequence of delayed enrollment if the opportunity cost of a year of schooling increases with age – late enrollment leads to lower expected lifetime earnings. In order to maintain total years of schooling with delayed entry, an individual would have to enter the work force later. As Glewwe and Jacoby (1995) illustrate, for each year of delay in entry to primary school in Ghana a child in their study loses 3 percent of lifetime wealth. The third pathway from malnutrition to educational outcomes is via the capacity to learn,
a direct consequence of the consequences of poor nutrition for cognitive development described in Section 2. Additionally, a hungry child may be less likely to pay attention in school and, thus, learn less even if he or she has no long-term impairment of intellectual ability.\(^\text{11}\) These three pathways clearly interact; a child with reduced ability to learn will likely spend less time in school as well as learn less while in class.

Though intuitively plausible, it is difficult to ascertain or quantify the causal pathway between nutrition and learning. Many of the observable factors that affect nutrition, such as family assets and parental education, are also ones that affect education. Similarly, unobservable attitudes about investment in children and in intra-family equity influence health provision and schooling decisions in a complex manner. Thus, while there are many studies that document associations between nutrition and schooling (see Pollitt 1990 and Behrman 1996 for reviews), there are far fewer studies that accurately portray the causal impact of child health and nutrition on school performance.

Four recent studies represent the most complete efforts at distinguishing the distinct contributory role of nutrition on education from associations. Glewwe and Jacoby (1995) found delayed enrollments among the malnourished in their cross-sectional study, but not difference in the total years completed. By contrast, Alderman, Hoddinott, and Kinsey (2002) track a cohort of Zimbabweans over two decades finding that both delayed school initiations and fewer grades completed for those children

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\(^{11}\) A few studies have attempted to investigate the tie between hunger and classroom performance using experimental design. Available results, however, are not conclusive regarding long term consequences, perhaps, in part, because controlled studies are hampered by difficulties in running experiments for an appreciable duration as well as the difficulty of encouraging parents to conform to the protocols of research design and the inability to use a placebo. Moreover, as shown in Grantham-McGregor, Chang and Walker (1997), while feeding children may improve attention, its impact on learning depends on the classroom organization. On this point, also see Powell, et al. (1998).
malnourished as children. Extrapolating beyond the drought shocks used for identification, the study concludes that had the median pre-school child in the sample achieved the stature of a median child in a developed country, by adolescence she would be 4.6 centimeters taller, had completed an additional 0.7 grades of schooling as well as started school seven months earlier.

Glewwe, Jacoby, and King (2001) track children from birth through primary school and find that better nourished children both start school earlier and repeat fewer grades. A 0.6 standard deviation increase in the stature of malnourished children would increase completed schooling by nearly 12 months. The Filipino setting of this study is one in which most children initiate school. Using longitudinal data from rural Pakistan where school initiation is much lower, Alderman et al. (2001) find that malnutrition decreases the probability of ever attending school, particularly for girls. An improvement of 0.5 standard deviations in nutrition would increase school initiation by 4% for boys but 19% for girls. As the average girl (boy) in the villages studied who begins school competes 6.3 (7.6) years of schooling, improvements in nutrition would have a significant effect on schooling attainment.

It is relatively straightforward to infer from the impact of nutrition on years of schooling to the productivity lost using the substantial literature on wages and schooling. There are hundreds of studies on the impact of grades of schooling completed on wages - many of which are surveyed in Psacharopoulos (1994) and Rosenzweig (1995). Wages, however, are also directly influenced by cognitive ability, as well as by the appreciable influence of cognitive ability on schooling achieved. Poor cognitive function as a child is associated with poorer cognitive achievement as an adult, see Martorell (1995),

It is possible to use these studies to estimate the magnitude of the productivity costs of poor nutrition. For example, Alderman, Hoddinott and Kinsey (2002) use the values for the returns to education and age/job experience in the Zimbabwean manufacturing sector provided by Bigsten \textit{et al.} (2000, Table 5) to infer the costs associated with poor nutrition in Zimbabwe. The loss of 0.7 grades of schooling and the 7 month delay in starting school there translates into a 12 per cent reduction in lifetime earnings. Such estimates are likely to be lower bounds given Fogel’s (1994) evidence that links short stature amongst males to the early onset of chronic diseases and to premature mortality.

Behrman and Rosenzweig (2003) take a more direct approach. They study a sample of adult monozygotic (identical) twins in the United States and determine that with controls for genetic and other endowments shared by such twins (which would not be affected by programs to increase birth weight), the impact of low birth weight on schooling or wages is far larger than it appeared without such controls (e.g., the impact on schooling attainment is estimated to be twice as large, with a pound increase in birth

\(^{12}\) In addition, studies such as Behrman and Rosenzweig (2003) and Strauss (2000) that show the net impact of LBW on earnings capture both the indirect schooling effect and the direct impact of ability as well as any influence of stature.
weight increasing schooling attainment by about a third of a year). This may reflect post-
natal choices on investments (with fewer investments for children with greater birth
weight) or a negative correlation of health and ability endowments.\textsuperscript{13}

Alderman and Behrman (2003) consider the net present value of \textit{all} benefits with
reducing a particular dimension of malnutrition. They consider seven benefits (reduced
infant mortality, reduced neonatal care, reduced childhood morbidity, lost productivity
due to reduced cognitive ability and learning, lost productivity due to smaller stature,
reduction of the costs of chronic illness, and inter-generational benefits due to better birth
outcomes) associated with reducing low birthweights. Given the net present value
approach, the absolute and relative benefits of these seven categories are affected by the
discount rate. Lower discount rates allow benefits that occur later in life to add more to
the total. Conversely, when discount rates are high benefits that occur in infancy
contribute a greater share of the total than they do under lower discount rates.
Nevertheless, over a wide range of assumptions about both discount rates and the
nominal value of benefits, the productivity gains from cognitive ability and schooling
contribute most to the total, despite the fact that these are assumed not to begin to accrue
until age 15. For example, at a discount rate of 5\% and using the core assumption in this
study, the productivity gains from increased cognitive ability represent 41\% of the total
gains ($580) per low birth weight prevented. The reduction in chronic disease was only
4\%. At a 1\% discount rate these two categories contribute 42 and 12 percent
respectively.

\textsuperscript{13} In a similar vein, Conley and Bennett (2000) find that family fixed effects models using siblings (not
necessarily twins) indicate a much larger negative relation between low birth weight and the probability of
completing high school than found in cross-sectional estimates.
Horton and Ross (2003) use a different approach to construct illustrative estimates of the costs of iron deficiency for 10 countries. Rather than reporting in terms of the benefits per case of anemia prevented they report the per capita costs of anemia. Their estimate of the physical loss of productivity comes to 0.57% of GNP. Considering the impact of anemia on schooling and cognitive development, they estimated the median for the total costs to these economies at 4% of GNP or $16.78 per capita.

4. Potential policy interventions

On a very general level, policy interventions are warranted that increase social welfare. Often it is convenient to consider instead two related basic policy motives – increasing efficiency and improving distribution. The distributional goal that is most emphasized is reducing poverty.

Poverty and malnutrition are strongly linked empirically. Based on the available aggregate data, Behrman and Rosenzweig (2003) report that cross-country variation in GDP per capital in PPP terms is inversely related to the percentage of low birth weights among all births and is consistent with almost half of the variation in the percentage of births that are low birth weight (below 2500 grams) across countries. Haddad et al. (2003) estimate that the cross-country elasticity of child underweight rates (for children under five years of age) with respect to per capita income for 1980-96 is –0.5. This is virtually the same as the mean for the elasticity from 12 household data sets (though estimates in both approaches decline somewhat in absolute value with the inclusion of

14 Their estimates suggest, however, that only a small part of this association between LBW and GDP per capita is due to the causal effects of LBW on productivity.
Therefore successful efforts to reduce most forms of malnutrition are likely to have incidences of benefits concentrated relatively among the poor. Thus policies that are focused on alleviating malnutrition are likely to be pro-poor, though explicit efforts at targeting such policies towards the poor is likely to make them more effectively attain the poverty alleviation form of the distributional policy motive.

There also may be important efficiency reasons for policies pertaining to relieving malnutrition. From a social perspective the private incentives to invest in nutrition may be inadequate because there may be positive spillovers from better nutrition (e.g., better nourished individuals may be less susceptible to contagious diseases and less likely therefore to spread them to others), because of imperfect information about the benefits of better nutrition, and because of capital and insurance markets imperfections that lead to less than socially desired levels of investments in nutrition and in other forms of human capital. Therefore there are likely to be efficiency reasons – i.e., differentials between private and social rates of return – for using public resources to alleviate malnutrition. Moreover generally these efficiency reasons are stronger for poorer than for better-off members of society – the poorer are more likely to be in environments in which disease contagion is greater, have more imperfect information and have less access to capital and insurance markets and less possibility of self-financing and self-insuring investments. Thus there may be a number of “win-win” policy options that are pro-poor and pro-efficiency.

To what extent are there actual possible pro-poor distributional gains and efficiency gains from policies that reduce malnutrition is, of course, an empirical
question. The review of studies in Section 3, together with the higher prevalence of malnutrition among poorer members of society noted above, suggests that there are a number of policies to alleviate malnutrition that might be warranted as part of anti-poverty efforts. Malnutrition alleviation policies that improve education and improve economic productivity are likely to have positive long-run benefit for individuals from poor families.

The review of studies in Section 3, however, does not suggest much that is very concrete about the efficiency policy motive. Though there are a priori reasons, as noted, to think that nutrition may be inadequate from a social perspective due to inefficiencies, the available literature provides virtually no empirical evidence on the extent of such inefficiencies or on whether nutritional programs are likely to be very high in policy hierarchies to address existing inefficiencies. Much of the empirical evidence focuses on what arguably are private impacts of improved nutrition and does not address the possibilities of differentials between the private and the social rates of returns to investing in nutrition. However, this distinction between public and private returns is a bit blurred in ‘second best’ situations in which there exist a number of departures from allocative efficiency. In such situations, efficiency gains may come from reducing pre-existing inefficiencies. For example, even if improved health is mainly a private benefit (and this is arguable, as discussed above), if the state already invests in curative health care, investments in nutrition that reduce these health expenditures are second best efficiency gains. Similarly, given the resources most governments devote to the provision of education, there is a public finance argument for investments in nutrition that improve the efficiency of these schooling investments.
5. Conclusions

Malnutrition is widespread in many developing countries, with close to a billion people estimated to be malnourished. Recent work has emphasized the importance of health and nutrition for productivity and economic growth. This chapter adopts such an approach, with focus on the microeconomic evidence of the direct and indirect links between nutritional status and representations of productivity as they accumulate over the life cycle.

We present a brief explanation of the causes and measurement of malnutrition and of estimates of the extent of malnutrition across developing countries as preludes to our survey of micro evidence about the productivity impact in developing countries of improved nutrition on individuals throughout their life cycles – from conception through infancy and childhood and into adolescence and adulthood. The available studies suggest that some of these gains may be considerable and that may operate through many channels – increasing cognitive development, increasing physical stature and strength, inducing earlier school enrollment and more regular school attendance, inducing greater schooling and learning, and increasing adult productivity – as well as saving resources that otherwise would go towards dealing with diseases and other problems related to malnutrition over the lifecycle.

We then examine the possible gains from different nutritional policy strategies and the policy bases for adopting such strategies. The empirical evidence supports the possible use of nutritional policy to attain better pro-poor distributional goals by helping to make individuals from poor families more productive over their life cycles. The
available empirical evidence, however, is not very informative about the efficiency motive for nutritional policies. There are a priori reasons to suspect that there are possible efficiency gains from nutritional policies due to health spillovers and failures in information, capital and insurance markets. But the available empirical evidence is almost silent on the existence of such possibilities, whether there are important differences between private and social rates of returns to investing in nutrition, whether nutritional policies would be effective means of addressing any related efficiencies, and to what extent some types of nutritional policies are likely to be “win-win” pro-poor and pro-efficiency. Therefore there is an important research gap considering possible efficiency reasons for particular nutritional policies.
References


Martorell, Reynaldo. 1995. “Results and Implications of the INCAP Follow-Up Study.” *Journal of Nutrition* 125 (suppl.): 1127S-1138S.


Roseboom, Tessa, H. P van n der Meulen; Ravelli Anita, C Osmond, D. Barker, O. Bleker. 2001. “Effects of Prenatal Exposure to the Dutch Famine on Adult Disease in Later Life; an Overview.” *Molecular and Cellular Endocrinology* 185: 93-98.


