ABSTRACT

Title of Dissertation: DETERMINANTS OF CHILDHOOD MORBIDITY AND THE ROLE OF MALNUTRITION: EVIDENCE FROM INDONESIA

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Studies that have attempted to examine the impact of early childhood malnutrition on acute illness have failed to adequately establish the causal link from malnutrition to acute illness. The empirical challenge arises because household behavioral decisions that influence investment in a child's nutrition and growth are very likely correlated with other household decisions that affect a child's incidence of illness. These include decisions to invest in hygiene and sanitation or a mother's knowledge and use of appropriate feeding practices. There may also be unobserved risk factors, such as genetic endowments, which introduce correlation between one of the regressors – nutritional status – and the error term in a disease production equation.

In this dissertation, I test two basic hypotheses: (1) chronic undernutrition in early childhood, as measured by stunting in children under five, increases the probability of contemporaneous acute illness; and (2) there is a significant effect of early childhood

malnutrition on the probability of developing acute illness later in childhood. I estimate a model that predicts the incidence of febrile, diarrheal and respiratory disease, diseases which combined account for the greatest total burden of morbidity and mortality in children in developing countries.

I focus my research on contemporaneous and longer-term acute illness outcomes in children under five for three reasons. First, substantial research has shown that children are at greatest risk of malnutrition in the early years of life, particularly before age two (Victora *et al.* 2008; Ruel *et al* 2008). In this period, children are no longer exclusively breastfeed and they have high nutritional requirements because they are growing quickly. Second, the burden of infectious disease is disproportionately borne by children under five due to their relatively immature immune systems and their dependence on caregivers to use appropriate feeding and hygiene practices to avoid infection (Martorell 1999; Martorell and Habicht 1986). Third, since most of the literature on the long-term consequences for human capital formation focuses on conditions in early childhood, by placing this research question in the same context, it can be more clearly seen as contributing to the broader literature on human capital formation.

I employ instrumental variables to allow identification of the impact of early childhood malnutrition on acute illness. I use a panel dataset from three waves of the Indonesian Family Life Survey (IFLS) to address the measurement challenges that arise due to the unobservable household factors that influence both the likelihood of early childhood

malnutrition and acute illness, and the synergistic nature of malnutrition and infection. My results show a strong and statistically significant contemporaneous effect of malnutrition on the likelihood of acute illness. I find that children under five who are stunted are 16 percent more likely than children who are not stunted to report symptoms of acute illness. I find that the impact of malnutrition on the likelihood of acute illness remains positive and significant four years into the future. Children who were stunted in 1993 are still 5 percent more likely than non-stunted children to experience acute illness in 1997. While I find this impact of early childhood stunting on future illness outcomes dissipates seven years later, I present suggestive evidence that this may reflect the fact that many of the children in my sample who were stunted in 1993 are in fact no longer stunted by 2000. Overall, these results suggest that efforts at reducing early childhood malnutrition can lead not only to immediate health benefits in terms of lower rates of infectious disease, but also lead to better health outcomes in the future.

Many international organizations and bilateral donors are prioritizing improvements in early childhood nutrition with the goal of improving long-term human capital outcomes (World Bank 2002; USAID 2008). The most important implication of my results is that improvements in early childhood nutrition and reducing the burden of disease are complementary objectives; improved early childhood nutrition will facilitate meeting the Millennium Development Goal of reducing the burden of disease. Further, to the extent improvements in pre-school nutritional status reduce either the incidence of acute illness, the severity of acute illness episodes, or both, such improvements may have indirect benefits. These include reducing school absenteeism which likely will enhance

the acquisition of knowledge at school and lead to higher school completion rates among children in developing countries.

# DETERMINANTS OF CHILDHOOD MORBIDITY AND THE ROLE OF MALNUTRITION: EVIDENCE FROM INDONESIA

by
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## Dedication

For my girls, who gave me reason to persevere.

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## Chapter 1 Introduction and Statement of the Problem

The goal of this dissertation is to test two hypotheses: chronic malnutrition in early childhood, as measured by stunting in children 0-5 years old, increases the probability of contemporaneous acute illness; and (2) early childhood malnutrition increases the probability of developing acute illness later in childhood. The hypotheses are tested using data from the 1993, 1997 and 2000 waves of the Indonesia Family Life Survey. Specifically, I examine the impact of malnutrition, measured by low height-for-age (stunting) on the probability that a child experiences febrile, diarrheal or respiratory disease. The remainder of this chapter motivates the importance of the topic and discusses the failure of the literature to adequately address it. This is followed by a description of my research strategy and a preview of key results.

### Motivation and Limitations of the Existing Literature

Infectious diseases kill between 14 and 17 million people each year and debilitate another 50 million. They cause 63 percent of all childhood deaths and 48 percent of premature deaths[1] worldwide (WHO 2008a). Children under five years of age are particularly vulnerable to infectious diseases. More than 70 percent of all deaths from infectious disease occur in children under five, most due to largely preventable diarrheal, respiratory and febrile illnesses (WHO 2008; Layne 2004). The overwhelming majority of new cases of infectious disease

[1] Defined as death before attaining average life expectancy, which varies by country, year and gender.

occur in developing countries where poor infrastructure, poor education, poor hygiene practices, crowding in urban areas and lack of universal access to high quality healthcare combine to create a disease-rich environment.

The global burden of malnutrition follows a similar socio-economic, demographic and geographic distribution. One in six people worldwide are malnourished, most in developing countries (Spielman and Pandya-Lorch 2009). Childhood undernutrition, in particular, is highly prevalent in developing countries. One-third of children under five years old in developing countries are *stunted* in growth (de Onis *et al.*2000), meaning that their height-for-age is significantly lower than would be expected in a well-nourished population.

Studies in the medical and the epidemiological literature have established a correlation between infectious disease and secondary malnutrition (malnutrition arising as a result of infection) (Scrimshaw, Taylor and Gordon 1968; Mosley and Chen 1984; Duggan *et al.*1986.). The same literature suggests that malnutrition may negatively impact the immune system and make one more susceptible to infection and/or more severe disease once infected (Scrimshaw, Taylor and Gordon 1968; Moore *et al.* 1997; Moore *et al.* 1999). Experts recently estimated that more than one-third of child deaths and 11 percent of the total disease burden worldwide are attributable to early childhood malnutrition (Bhutta *et al.* 2008). However, these estimates are based on an observed correlation between malnutrition and morbidity and mortality, not an established causation.

This literature suffers from econometric limitations because it fails to adequately account for the potential endogeneity of the health outcomes (Behrman 1996; Glewwe and King 2001; Alderman, Hoddinott and Kinsey 2006). Studies that have attempted to examine the impact of early childhood malnutrition on acute illness have failed to adequately establish the causal link from malnutrition to acute illness. This is primarily because the household behavioral decisions that influence investment in a child's nutrition and growth are very likely correlated with other household decisions that would affect a child's probability of illness, such as decisions to invest in hygiene and sanitation or a mother's knowledge and use of appropriate feeding practices. There may also be hidden risk factors, such as genetic endowment, which introduce correlation between one of the regressors – nutritional status – and the error term in a disease production equation. The complex synergism between malnutrition and infectious disease also creates a measurement challenge which makes it difficult to determine whether the relationship between malnutrition and infectious disease is causal and, if so, the direction of causality as well as the magnitude of the effect. The direction of causality is an empirical question which rests on the nature of the illness and whether malnutrition is measured by a short-term indicator such as *wasting* or a longer-term indicator such as *stunting* (Scrimshaw, Taylor and Gordon 1968).

Within the economics and medical literature, the effects of malnutrition have been shown to cause improvements in cognitive outcomes (Maluccio *et al.* 2006;

Case and Paxson 2008; Glewwe and King 2001), educational attainment (Behrman 1996; Brown and Pollitt 1996), chronic morbidity (Barker 1997; Barker 2002; Lucas 2006; Fisher *et al.* 2006) and premature adult mortality (Fisher *et al.* 2006; Choi *et al.* 2000). However, little is known about the impact of malnutrition on the incidence and severity of acute illness. Recent medical literature cites the lack of rigor applied to studies of the relationship between nutritional status and infectious disease and calls for the urgent need to incorporate techniques used in other disciplines to improve our understanding of the relationship between nutrition and immune function (Prentice *et al.* 2008; Victora *et al.* 2008; Black *et al*. 2008).

Understanding the exact association between malnutrition and acute illness is important from both an epidemiological and socioeconomic standpoint. Indeed, understanding the *causal* relationship between the two, specifically whether improvements in nutritional status *cause* a reduction in infectious disease, is critical for the design of effective preventative health policies. If the two are causally related (i.e., if malnutrition causes an increased likelihood of acute illness), decreasing the disease burden is an additional key benefit to decreasing early childhood malnutrition. This is true even if malnutrition primarily affects duration and severity of illness, rather than simply incidence of acute illness. If reductions in malnutrition lead to reductions in the burden of acute illness, we might also expect indirect benefits associated with other human capital outcomes. In addition to its positive direct effect on cognitive outcomes, we would expect

improvements in nutritional status will also yield positive returns in education and labor markets indirectly through nutrition's effect on acute illness since healthier children are likely to miss fewer days of school, and are more alert and energetic in school.

If the relationship between malnutrition and acute illness is causal, the timing, duration and intensity of investments in early childhood nutrition may also depend on the length of the effect of nutritional status on susceptibility to disease. At present, little is known about the *duration* of the effect of malnutrition on susceptibility to illness due to insufficient research on long-term changes in immune function (Prentice *et al.* 2008). There are a small number of studies which suggest a relationship between maternal and infant undernutrition and poor immune function, which may affect children even into middle adulthood (McDade *et al.* 2001; Moore *et al.* 1997; Moore *et al.* 1999). Experts in medicine and nutrition have recently highlighted the need for additional research on the linkage between nutrition and immune function, both to establish causality and to determine the duration of any effect (Prentice *et al.* 2008; Victora *et al.* 2008). Investigating the duration of any potential effect of malnutrition on the likelihood of acute illness is therefore of independent empirical interest.

**Research Strategy**

In this dissertation, I test two basic hypotheses: (1) chronic undernutrition in early childhood, as measured by stunting in children under five, has a causal effect on

the probability of contemporaneous acute illness; and (2) there is a significant effect of early childhood malnutrition on the probability of developing acute illness later in childhood. I estimate a model that predicts the incidence of febrile, diarrheal or respiratory disease, diseases which combined account for the greatest total burden of morbidity and mortality in children in developing countries, and are among the most common infectious diseases in Indonesian children.

I employ instrumental variables to allow identification of the causal impact of early childhood malnutrition on acute illness. I use a panel dataset from three waves of the Indonesian Family Life Survey (IFLS) to address the measurement challenges that arise due to the unobservable household factors that influence both the likelihood of early childhood malnutrition and acute illness, and the synergistic nature of malnutrition and infection. I study the contemporaneous impact of malnutrition on the likelihood of acute illness in a cohort of children aged 0 to 5 years old in 1993. I exploit the panel nature of my data to establish whether there is any long-term impact of early childhood stunting on illness outcomes into later childhood and early adolescence.

This approach addresses two distinct sources of potential bias. In the contemporaneous context, bias may arise from both omitted variables and reverse causality as both induce a correlation between the error term and the regressors. I expect to avoid problems associated with reverse causality by careful selection of an anthropometric indicator. I use height-for-age, or stunting,

because previous research has shown that it is the best indicator of long-term undernutrition. This allows me to avoid concerns of reverse causality that might arise with the use of short-term indicators of malnutrition such as weight-for-age and weight-for-height. The latter are more likely to suffer from reverse causality, particularly in the case of diarrheal disease where children can lose up to 10 percent of their body weight with a single bout of disease (Layne 2004).

I cannot rule out the possibility of omitted variable bias. Factors that clearly affect the incidence of disease and malnutrition, such as household hygiene and child care practices, but that are often difficult to measure, will lead to bias if not properly accounted for in an estimation of the impact of malnutrition on acute illness outcomes. To avoid both these potential sources of bias, I employ instrumental variables when estimating the probability of contemporaneous illness. In the longer-term context, when establishing whether early childhood nutritional status affects acute illness outcomes four years and seven years later, potential bias may arise due to the same omitted variable problem. I explore this possibility by comparing results from estimations using both standard OLS and instrumental variables.

I use parents' heights and the child's birth quarter as instruments for height-for-age. Parents' heights are highly correlated with a child's height because they are a genetic determinant of the latter, but are presumably not correlated with whether or not a child experiences acute illness in early childhood or

adolescence. Any association between parents' heights and childhood acute illness is presumably due to early childhood stunting. Birth quarters have been found to be potentially important predictors of health status, arguably by picking up the relation between time of birth and harvest (Lokshin and Radakan 2009). Findings from a small number of randomized trials provides evidence of deficits in immunocompetence presumably arising from fetal and infant undernutrition during lean seasons in developing countries (Moore *et al.* 1997; Moore *et al.* 1999), which further supports use of birth quarters as potentially viable instruments.

I focus my research on contemporaneous and longer-term acute illness outcomes in children who are identified as malnourished by age five for three reasons. First, substantial research has shown that children are at greatest risk of malnutrition in the early years of life, particularly before age two (Victora *et al.* 2008; Ruel *et al* 2008). In this period, children are no longer exclusively breastfed, they have high nutritional requirements because they are growing quickly. Second, the burden of infectious disease is disproportionately borne by children under five due to their relatively immature immune systems and their dependence on caregivers to use appropriate feeding and hygiene practices to avoid infection (Martorell 1999; Martorell and Habicht 1986). Third, since most of the literature on the long-term consequences for human capital formation focus on conditions in early childhood, by placing this research question in the

same context, it can be more clearly seen as contributing to the broader literature on human capital formation.

Investigation of this research question in the Indonesian context is extremely relevant. According to IFPRI's Global Hunger Index, Southeast Asia has among the highest child malnutrition rates in the world (IFPRI 2008). Though prevalence of stunting has declined from 46 percent of children under five in 1995 to 33 percent of children under five in 2000 (Strauss *et al.* 2004), stunting prevalence remains comparable to many sub-Saharan African countries with much lower overall development. Indonesia also has a high prevalence of the most common diarrheal, respiratory and febrile diseases in the developing world. These conditions make Indonesia an ideal location in which to study the potential impact of early childhood malnutrition on acute illness.

**Results and Policy Implications**

My results show a strong and statistically significant contemporaneous effect of malnutrition on the likelihood of acute illness. I find that children under five who are stunted are 16 percent more likely than children who are not stunted to report symptoms of acute illness. I find that the impact of malnutrition on the likelihood of acute illness remains positive and significant four years into the future. Children who were stunted in 1993 are still 5 percent more likely than non-stunted children to experience acute illness in 1997. While I find this impact of early childhood stunting on future illness outcomes dissipates seven years later,

this may reflect the fact that many of the children in my sample who were stunted in 1993 are in fact no longer stunted by 2000. Overall, these results suggest that efforts at reducing early childhood malnutrition can lead not only to immediate health benefits in terms of lower rates of infectious disease, but also lead to better health outcomes in the future.

My research underscores the importance of decreasing the prevalence of early childhood malnutrition in order to improve both immediate and longer-term health outcomes. Many international organizations and national governments are targeting resources towards reducing the burden of disease.[2] Many international organizations and bilateral donors are also prioritizing improvements in early childhood nutrition with the goal of improving long-term human capital outcomes (World Bank 2002; USAID 2008). The most important implication of my results is that reducing the prevalence of early childhood malnutrition and reducing the burden of disease are complementary objectives; improved early childhood nutrition will facilitate meeting the Millennium Development Goal of reducing the burden of disease. Further, to the extent improvements in pre-school nutritional status reduce either the incidence of acute illness, the severity of acute illness episodes, or both, such improvements may have indirect benefits. These include reducing school absenteeism which likely will enhance the acquisition of knowledge at school and lead to higher school completion rates among children in developing countries.

[2] These include such initiatives as the Global Fund to Fight HIV/AIDS, Tuberculosis and Malaria, the U.S. President's Emergency Plan for AIDS Relief (PEPFAR), Roll Back Malaria and the Global Alliance for Vaccines and Immunisation (GAVI).

The rest of this dissertation is organized as follows. Chapter 2 presents an overview of the physiological foundations of malnutrition and a synthesis of the relevant literature. Chapter 3 presents the empirical model used to explain acute health outcomes. Chapter 4 introduces the survey data, while Chapter 5 discusses the setting. Chapter 6 details the sample I use for empirical estimations and provides descriptive statistics. Chapter 7 presents my empirical findings. Chapter 8 concludes and offers policy insights.

## Chapter 2 Malnutrition and Illness: Physiological Foundations and Literature Review

This chapter defines malnutrition, describes common indicators of malnutrition, and provides an overview of the physiological foundations of malnutrition. This is followed by a review of three strands of relevant literature: the medical literature on malnutrition, morbidity and mortality; the literature linking malnutrition to non-health outcomes, such as education and earnings; and the literature on the socio-economic determinants of morbidity and mortality. The final section of this chapter summarizes the contribution of my present research to the literature.

### 2.1 The Definition of Malnutrition and Common Indicators

Malnutrition is defined generally as an inappropriate amount of the nutrients needed for appropriate growth and development. Malnutrition can be caused by an unbalanced or insufficient diet, or by medical conditions such as infection that affect the digestion of food or absorption of nutrients from food (Scrimshaw, Taylor and Gordon 1968). Malnutrition may be due to either overnutrition or undernutrition (including inadequate intake of calories and/or micronutrients). When malnutrition is due to undernutrition during childhood, it can manifest itself as failure to attain growth potential. The most common indicator of long-term undernutrition during childhood is a child's failure to reach genetic potential in height.

Health or nutritional status of a child is usually assessed in three ways: measurement of growth and body composition (anthropometric indicators such as height, weight, arm circumference and skin fold thickness); analysis of the biochemical content of blood and urine (biochemical indicators such as iron deficiency anemia or Vitamin A deficiency); or through clinical examination of external physical signs of nutrient deficiencies (clinical indicators such as goiter for iodine deficiency or night-blindness in the case of Vitamin A deficiency*).* Among the three assessment methods, anthropometric measurement is a common and easy way to assess health and nutrition status. The other two methods are less practical because of the logistical difficulties involved and because data collection and analysis is expensive and time-consuming. Biochemical and clinical indicators are more useful when malnutrition is extreme (Martorell and Ho 1984). Due to their relative cost-effectiveness for wide-scale usage and relative sensitivity in detecting more moderate forms of malnutrition, anthropometric measurements are the most widely available in household surveys.

Three common measurements include weight-for-age, height-for-age and weight-for-height (often converted into Body Mass Index, or BMI). Each measurement is generally converted to a *z*-score, which indicates the number of standard deviations an individual is above or below the mean of a reference population. A *z*-score of two standard deviations or more below the mean indicates moderate malnutrition, while a *z*-score of three standard deviations or more below the

mean indicates severe malnutrition. Weight-for-age, routinely collected in growth promotion programs, is the most common assessment of child nutrition status. It is a measure of both short- and long-term insults to health. A child is considered underweight if his/her weight-for-age is two standard deviations or more below the mean for the reference population. Weight-for-height is a measure of acute or short-term nutritional deficiency. Wasting, a term applied when weight-for-height falls two standard deviations or more below the mean for the reference population, is a sensitive indicator often used for short-term program intervention such as providing nutritional supplementation in emergencies. Wasting can be calculated without knowing the age of a child and, therefore, is the best malnutrition indicator when it is difficult to determine the exact ages of the children being measured (such as in complex emergencies like famines). Height-for-age is a measure of linear growth [3] and reflects the long-term, cumulative effects of inadequate nutrition and poor health status. A child is considered stunted if his/her height-for-age is two standard deviations or more below the mean for the reference population, and severely stunted if height-for-age is three standard deviations or more below the mean.[4]

One potential criticism of the use of anthropometric measurements as indicators of malnutrition is that ethnic differences in genetic potential, particularly height-

[3] Linear growth is defined as longitudinal growth of the human skeleton, or growth in stature.

[4] Height-for-age z-scores (HAZ), often reported in the literature, are based on fitting a standard normal distribution to the growth curves of a (healthy) reference population of children. A child with a z-score of zero is exactly at the median in terms of height for age, while children with positive (negative) z-scores are taller (shorter) than average. Approximately 2.3 percent of a healthy population, by definition, will fall at or below 2 standard deviations below the mean, while approximately 0.14 percent will fall at or below 3 standard deviations below the mean.

for-age, may cause some children with genetically-determined short stature but who are otherwise healthy to be incorrectly classified as being malnourished. The selection of an appropriate reference group against which a child's growth should be compared was the subject of much discussion in the 1970s and 1980s (Waterlow 1980; Gueri, Gurney and Jutsum 1981; Johnson, Yamanaka and Formacion 1984). Results from a number of studies confirmed, however, that "children from widely varying ethnic backgrounds tend to develop at the same rate given a similar socioeconomic environment" (Gueri, Gurney and Jutsum 1981). If there are ethnic differences in genetic growth potential, such differences generally do not show until the pre-pubertal growth spurt (Martorell and Habicht 1986; Habicht *et al.* 1974). The new WHO international child growth standards, released in 2006, upon which my results are based, were developed to account for differences in ethnicities and diets (WHO 2006b). I further avoid this problem in the study of contemporaneous illness by restricting my sample to children who are age 0 to 5 years old. When studying the longer-term effects malnutrition on the probability of acute illness, I extend the analysis until the birth cohort of 0 to 5 year olds in 1993 is no older than 12 years old.

### 2.2 Physiological Process of Growth and the Impact of Acute Illness on Nutritional Status

Human growth is an outcome of complex physiological processes involving genetics, nutrition, health care and environmental influences. Estimates suggest that 60-80 percent of the variation among individuals in height can be explained

by differences in genetics; whereas 20-40 percent of adult height is determined by net nutrition (food intake minus demands from disease) (Pierce 2009; Lai 2006).[5] Inadequate health care and nutrition, and poor environmental conditions that contribute to disease, can prevent the attainment of a child's full growth potential. Insults that may prevent children from attaining their genetic growth potential can occur either *in utero,* post-natally or both. The long-term effects of such insults ultimately depend on factors including maternal health status and the timing of the insult itself (Adair and Guilkey 1997; Hall and Peckham 1997; Glewwe and King 2001; Victora *et al.* 2008).

Growth-related insults *in utero* generally arise from persistent, untreated illness during pregnancy. These can reduce placental blood flow, leading to a proportionate reduction in skeletal and soft tissue growth (Villar and Belizan 1982; Kramer 1987a, 1987b). Intrauterine growth retardation in full term infants accounts for the majority of low birth weight infants in less developed countries, due in part to the high prevalence of infectious diseases and conditions known to promote chronic stunting *in utero* such as malaria, anemia and helminth (worm) infections (Kramer 2000; Villar and Belizan 1982). Poor nutritional status of women during pregnancy also plays an important role in determining fetal growth (Victora *et al.* 2008; Grantham-McGregor *et al.* 1999a).

[5] A full review of the genetics of human height is beyond the scope of this work. Here, I focus on the interrelated influences of nutrition, health care and environment (infectious disease specifically) on height.

Children have especially high nutritional requirements to maintain the growth needed to reach their genetic potential. Growth rates during the first two to three years of life are much higher than in later life, (Martorell 1999; Ruel *et al.* 2008). At the same time, children in this age range are particularly susceptible to disruptions in growth due to illness. This is because their immune systems are still developing and they must rely on parents and caregivers to ensure proper hygiene, health care and nutrition (Pelletier 1994; Pelletier *et al.* 1994). Inappropriate feeding practices, including early weaning from breastfeeding, premature introduction of complementary feeding and culturally-dictated diets during illness, often exacerbate symptoms and/or contribute to delayed recovery (Scrimshaw, Taylor and Gordon 1968; Powers 2004).

All infectious diseases have direct adverse metabolic effects (Scrimshaw, Taylor and Gordon 1968). Respiratory and febrile illnesses increase metabolism and thus caloric requirements (Beisel 1981).[6] Diarrheal illness and worm infestation result in both direct loss of nutrients and malabsorption of micronutrients (Martorell and Ho 1984). The clinical symptoms of malaise, anorexia and vomiting frequently influence the amount and kind of food consumed.[7]

In low- and middle-income settings, a high prevalence of infectious illnesses combines with poor sanitation to facilitate fecal-oral transmission of diarrheal and

[6] In a classic study of appropriate treatment courses for common infectious diseases, Meara (1916) estimated a 25 percent increase in caloric requirement when fever is 102º F or greater.

[7] Some of these effects are believed to act as an evolutionary defense mechanism to deprive infectious agents of nutrients and thereby hasten recovery (Prentice *et al.* 2008).

parasitic illnesses (Grantham-McGregor *et al* 1999b). Both short-term and chronic infections during infancy and early childhood may promote stunting. Combined with poor feeding practices, high disease burden settings promote repeated infections that may prevent a child from completely restoring weight lost during illnesses, thereby resulting in a drop in the growth trajectory over the long term (Martorell and Ho 1984; Martorell *et al* 1975; Rowland and McCollum 1977).

The biological response to repeated infection and interruptions in nutritional intake is one of adaptation. Growth is slowed in favor of preserving vital organs; vital functions like circulation, respiration and maintenance of body temperature receive higher priority (Martorell and Ho 1984). This is particularly true of protein-energy malnutrition, one of the most common forms of malnutrition in developing countries. Scientific evidence suggests that such height-restricting biological responses to early childhood nutritional insults and disease may have short-run survival advantages. A chronically malnourished child is thought to adapt to the condition (unless it is severe) partly by reducing his or her need for nutrients through growth failure. In contrast to Seckler's "small but healthy" hypothesis (Seckler 1980),[8] there is widespread agreement within the scientific community that, though adaptation may have short-run advantages, there are many potential long-run negative consequences, including increased risk of

[8] Seckler (1980) argued that impoverished people living in developing countries have attained an optimum size with respect to their environment – they are simply "Small but Healthy." Seckler's controversial hypothesis has been discredited by anthropologists and nutritional experts as increasing evidence mounts against his "no-cost adaptation" argument.

coronary disease, diabetes and hypertension (Barker 1997; Adair, Kuzawa and Borja 2001; Victora *et al.* 2008).[9]

## 2.3 Review of the Medical Literature on Malnutrition, Morbidity and Mortality

Most knowledge of the interactions of nutrition and infection comes from the clinical and biomedical literature, which outlines the ways in which malnutrition impairs the body's defense mechanisms. These studies typically do not consider any sociodemographic or economic factors that might influence illness outcomes, but focus instead on establishing the physiological link between nutritional status and risk of morbidity or mortality. There are three related strands of literature which focus on the interaction of nutrition and infection. The first is primarily associated with establishing the linkage between nutritional deficiencies and infection. The second smaller body of literature examines the effect of nutritional interventions on disease outcomes. The third explores the long-term impacts of early childhood malnutrition on chronic morbidity and premature adult mortality. Each makes an important contribution to our understanding of the potential importance of malnutrition in determining health outcomes, but should be interpreted with caution since most provide evidence of associations among health outcomes, rather than evidence of causation or magnitude of impact.

[9] The Barker Hypothesis (also known as the "thrifty phenotype") posits that, in response to undernutrition during critical stages in fetal growth, both the structure and functions of organs are "programmed" in ways that predispose people to a number of chronic conditions later in life including coronary heart disease, hypertension, stroke and diabetes (Barker 1997).

### 2.3.1 The Linkage Between Nutritional Deficiencies and Infection

An expansive scientific review conducted under the auspices of the World Health Organization (Scrimshaw, Taylor and Gordon 1968) outlined the specific ways in which nutritional deficiencies reduce the capacity of the host to resist the consequences of infection. They concluded that malnutrition can result in the following negative impacts on immune function:

(a) reduced capacity to form specific antibodies;
(b) reduced activity of the cells which attack foreign agents;
(c) reduced capacity to produce non-specific protective substances (e.g., mucus);
(d) reduced non-specific resistance to bacterial toxins;
(e) alterations in tissue integrity; and
(f) diminished inflammatory response and slower healing of wounds.

More recent evidence suggests that cell-mediated immunity is most affected by undernutrition (Martorell and Ho 1984). Two studies undertaken in The Gambia indicate a relationship between immune function and being born during the hungry season (Moore *et al.* 1999), and increased mortality risk from infections among young adults born during the hungry season (Moore *et al.* 1997). Studies conducted in Pakistan and the Philippines found that antibody response to vaccines were lower in children born at lower birth weights relative to higher birth weight babies (McDade *et al.* 2001; Moore *et al.* 2004).

During the mid-20$^{th}$ century, a wide array of medical studies was conducted designed to link nutritional deficiencies with the incidence and severity of disease, and an increased risk of mortality. A number of these studies used the natural

experiments associated with World Wars I and II. One study found an association between excess mortality among Danish soldiers during World War I and the lack of meat and fish in the diet (Faber 1938), while another (Palmer, Joblon and Edwards 1957) found rates of tuberculosis were four times higher in white Navy recruits who were 15 percent or more underweight for their height.

Many studies establish that acute respiratory and acute diarrheal diseases occur more frequently, are more severe and last longer among malnourished children. In a comparative study of the effect of diarrhea in children, Oropeza and Torres (1963) found morbidity in malnourished children three times higher and mortality seven times higher than in well-nourished children. In a study of the effects of diet on tuberculosis among Norwegian cadets, Leitch (1945) found quality of diet was a more significant predictor than net caloric intake in explaining tuberculosis incidence. Other studies found that malnourishment is correlated with more severe typhus (Gordon 1948), higher fatality rates associated with yellow fever (Kuczynski 1937), and increased susceptibility to clinical malaria and increased severity of malarial disease (Terzia *et al.* 1953).

During the early 1980s, there were a number of studies conducted in developing countries to examine whether or not malnourished children are more likely to become infected and/or suffer more severe illness compared with well-nourished children. The findings were mixed. Few studies found an increased incidence of illness among malnourished children (typically measured by weight-for-age,

though some also consider height-for-age and/or BMI). Wasting tended to be a stronger predictor of diarrheal illness than stunting. Delgado *et al.* (1983), for example, found that weight-for-age and BMI were stronger predictors of the incidence of diarrheal illness than height-for-age. More studies found a correlation between nutritional status and duration of illness (a possible indicator of severity). These studies, however, raise concerns about both omitted variable bias and reverse causality. None controlled for other possible influences on both nutritional status and acute health outcomes.

Many of the studies relate incidence or duration of illness to a child's weight-for-age or BMI measured within weeks of a reported illness (often diarrheal), or at the end of the study period. Using short-term indicators of malnutrition such as wasting (low weight-for-age or BMI), introduces potential endogeneity due to reverse causality. The illness outcome in these studies is usually diarrhea which could quite easily result in significant weight loss in a short enough time period that a weight measurement would not accurately reflect a child's "normal" weight. For example, a single bout of cholera, a common diarrheal disease in many developing countries, can result in a 10-20 percent loss of a child's body weight (mostly fluids) (Layne 2004).

In a longitudinal study of the incidence, duration and clinical severity of diarrheal and respiratory illness in 137 children in urban Costa Rica, James (1972) found no association between weight-for-age at the start of study and incidence of

respiratory illness. Neither height-for-age nor BMI were included as indicators of malnutrition. While James (1972) found no association between weight-for-age at the start of study and incidence of diarrhea among children under 3 years old, he did find twice the incidence among children 3-5 years old who were below the 75$^{th}$ percentile for weight-for-age.[10] Duration of both diarrheal and respiratory illness was found to be significantly correlated with low weight-for-age. The author reported no significant differences were found between normal and underweight children in terms of socioeconomic or environmental conditions, including household size, average weekly income, rent, and quality and quantity of the water source. It is not possible to verify the validity of this claim as no supporting data are presented. Importantly, however, the author noted that he was unable to control for inadequate child care or preventive health care, yet found evidence during home visits of a distinct difference in standards of care between malnourished and well-nourished children (James 1972).

There is some suggestive evidence that malnutrition, as indicated by stunting, increases the severity of illness. Tomkins (1981) investigated the relationship between weight-for-age, height-for-age and BMI, and the incidence of and percent of time spent ill with diarrhea among 343 children aged 6-32 months

[10] James (1972) used the Gomez-Galvan classification of malnutrition by which a child's is considered 2$^{nd}$ degree malnourished if his/her weight-for-age is less than 75 percent of the theoretical average of a reference population (i.e., 75$^{th}$ percent or less of the weight of children in the 50$^{th}$ percentile of weight-for-age). This standard was widely used until it fell into disfavor in the early 1980s (Gueri, Gurney and Jutsum 1981) and was replaced by the NCHS/WHO Child Growth Standards which was based on a U.S. reference population. The NCHS/WHO standards were themselves later replaced in 2006 by the WHO International Child Growth Standards, which are based on a six-country study that more accurately reflects the dietary and ethnic diversity of developing country children (WHO 2006). See WHO (2006a) for technical details about the WHO standards

during a three-month period in rural Nigeria. While he found no increased incidence of diarrhea for children with low height-for-age, he did find the percent of time spent ill was significantly higher among children with low height-for-age compared with children with normal height-for-age.

In another longitudinal study, Trowbridge, Newton and Campbell (1981) measured the increased risk of severity of diarrheal illness (proxied by duration of illness) based on weight-for-age, height-for-age and BMI of 216 children 12-36 months old in El Salvador. They took anthropometric measurements at the beginning and end of the one-year study period. They found that, although initial weight-for-age was not significantly associated with duration of diarrheal illness over the next year, both height-for-age and BMI were significantly associated. The fact BMI was also found to be a significant predictor may reflect the fact that BMI may reflect stunting, wasting or both. The authors noted that their findings of a link between poor nutritional status and acute illness outcomes likely suffered from omitted variable biases since the greater frequency of diarrhea among children with poorer nutritional status may have been "related more to increased exposure to enteric pathogens from poor environmental conditions and personal hygiene than to nutritional factors" (1375).

Martorell and Ho (1984) investigated the contribution of mild and moderate malnutrition to increased morbidity and mortality risk. They found weak evidence of increased incidence of diarrhea and respiratory illness among malnourished

children, but stronger evidence that duration and severity of illness is greater among malnourished children as compared to better nourished children. The authors suggested that determinants of infection (incidence) may be more closely aligned with the environmental conditions which influence disease prevalence rather than a child's nutritional status itself.

There are a small number of studies conducted in developing countries that suggest a relationship between maternal and infant undernutrition and problems in immune function. These may affect children even into middle adulthood. Studies in the Philippines and Pakistan (McDade *et al.* 2001; Moore *et al.* 2004) provide evidence of an association between low birth weight and long-lasting immune deficits[11] in adolescents and adults (specifically, a decreased ability to mount an immune response to vaccinations, which lowers the effectiveness of vaccinations). Two studies undertaken in The Gambia indicate a relationship between immune function and birth during the lean season, (Moore *et al.* 1997; Moore *et al.* 1999). Moore *et al.* (1999) reports a ten-fold increase in infection-related mortality risk among adults born during the lean season, and hypothesizes that impaired development of the thymus[12] during fetal growth may

[11] Adaptive immune responses are those involved in the ability to recognize and remember specific pathogens so as to mount stronger attacks in future encounters with an infectious pathogen. Most immunizations, such as the seasonal flu vaccine, are designed to provide the immune system with a 'kickstart' so that the system can mount a stronger attack if and when faced with a live virus.

[12] Unlike other vital organs, the thymus develops early during the fetal phase, enlarges during childhood, but then begins to atrophy in puberty and eventually begins to shrink with advancing age after puberty (Gray 1918). T-cells, which mature in the thymus, are one of the many important cells involved in adaptive immune responses. One indication of the importance of the thymus to immunocompetence is that patients with the congenital condition DiGeorge Syndrome

result in inadequate immune function once infected. The authors note that season of birth is used as a proxy for early-life exposures to nutritional and infectious stresses since the lean season in The Gambia is both a time of lowest food supplies and increased infections.

As noted above, most knowledge of the interactions of nutrition and infection comes from the clinical and biomedical literature which generally do not consider sociodemographic or economic determinants of nutritional status and risk of morbidity or mortality. There is a small body of literature that incorporates socioeconomic and environmental factors when examining the effect of nutritional interventions on disease outcomes, but it, too, fails to adequately establish the causal link from malnutrition to acute illness. This is primarily because the household behavioral decisions that influence investment in a child's nutrition and growth are very likely correlated with household decisions to invest in hygiene and sanitation, which affect a child's probability of illness. Any inferences drawn from estimation of the impact of malnutrition on acute illness that do not account for this endogeneity will suffer from omitted variable bias.

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lose their thymus at an early age and become severely immunodeficient and thereby develop a high susceptibility to infection (Wilson *et al.* 1993).

### 2.3.2. The Effect of Nutritional Interventions on Disease Outcomes

The INCAP study in Guatemala[13] is perhaps the most famous randomized controlled trial of the effect of a nutritional intervention during pregnancy and early childhood on health and behavioral outcomes. The study was designed primarily to investigate the impact of malnutrition on behavior and cognition, but it also examined the effects of improved nutrition on the risk of adverse health outcomes and provides the most conclusive evidence of the link between nutrition and disease susceptibility. The study found that the frequency, duration and severity of diarrheal illnesses, and of mortality resulting from measles, were significantly lower in children who received a protein-rich, high-calorie supplement (*atole*) compared to children in the control group, who received a non-protein, low-calorie supplement (*fresco*) (Martorell *et al.* 1975; Scrimshaw 2003; Martorell, Habicht and Rivera 1995).

Victora *et al.* (1999) considered the potential effect of improvements in nutritional status on mortality risk due to pneumonia. They conducted a review of nonexperimental studies that examine the association of pneumonia deaths with low birth weight, underweight status (low weight-for-age) and breastfeeding to derive relative risks associated with each contributing factor. Focusing on decreasing prevalence of protein-energy malnutrition, which they proxied by low weight-for-age, they estimated hypothetical reductions in pneumonia for a given

[13] In the mid-1960s, a longitudinal, multidisciplinary nutrition intervention study was undertaken in Guatemala by the Institute of Nutrition of Central America and Panama (INCAP) to investigate the relationship between undernutrition in pregnancy and early childhood, and health and behavioral outcomes in young children. Further details can be found in Martorell (1992), Martorell, Habicht and Rivera (1995) and Maluccio *et al.* (2006).

percentage of low weight-for-age cases prevented under different prevalence scenarios. In countries where the prevalence of low weight-for-age is moderate, they estimated a 10 percent decrease in prevalence would result in a 2.7 percent reduction in pneumonia deaths. In countries with the highest prevalence of underweight young children, they assumed the same 10 percent decrease in prevalence would translate into a 3.3 percent reduction in pneumonia deaths. If nutritional interventions resulted in a 100 percent decrease in the prevalence of underweight in high prevalence countries, they estimated pneumonia deaths among young children would be cut by one-third.

Victora *et al.* (1999) recognized the significant methodological limitations of their study owing to its reliance on nonexperimental studies. None of the studies reviewed appear to account for potentially important factors (such as maternal education, quality of child care or socioeconomic status) that might influence both nutritional status and acute health outcomes. Indeed, while all of these studies highlight the importance of the correlation between malnutrition and various acute illness outcomes, with the exception of the INCAP study, none adequately addresses the identification issues which would establish the causal relationship and allow one to make unbiased inferences regarding the impact of poor nutritional status on morbidity or mortality rates.

### 2.3.3 Malnutrition, Chronic Morbidity and Premature Mortality

Economists have established some of the long-term functional consequences of malnutrition and illnesses during childhood (Deaton 2006; Fogel 2004; Fogel1997; Strauss and Thomas 2007). Using a variety of experimental and non-experimental methods to establish causality, a number of studies in the medical and economics literature examine the effect of early childhood malnutrition on chronic morbidity and premature adult mortality (Barker 1997; Barker 2002; Lucas 2006; Fisher *et al.* 2006; Choi *et al.* 2000). Since this literature focuses on long-term outcomes, it necessarily does not explore the impact of malnutrition on acute illness.

Barker (1992) provided much of the early research on the effects of early childhood malnutrition on chronic morbidity in adulthood. The Barker Hypothesis (also known as the "thrifty phenotype" or "fetal origins hypothesis") posits that, in response to undernutrition during critical stages in fetal growth, both the structure and functions of organs are "programmed" in ways that predispose people to a number of chronic conditions later in life including coronary heart disease, hypertension, stroke and diabetes (Barker 1997).

Following Barker's work, economists have used natural experiments in order to test the fetal origins hypothesis and establish causal links between early childhood malnutrition and chronic morbidity and premature mortality.[14] Almond

[14] Costa (2003) used records from Civil War veterans to examine the impact of exposure to infectious disease during young adulthood on the age-gradient of subsequent mortality.

(2006), for example, found that *in utero* exposure to the 1918 flu pandemic significantly increased men's disability rates,[15] while Chen and Zhou (2007) found exposure to the 1959-1961 famine in China caused significant long-run effects on adult morbidity, but not on adult mortality. Van den Berg, Lindeboom and Portrait (2006) used the natural experiment of fetal and infant exposure to the Dutch Potato Famine to establish a causal link between early childhood malnutrition and premature mortality. While they found that men exposed to famine *in utero* have a significant lower residual life expectancy at age 50 than others, they did not establish the same effect for females, nor for males exposed to the famine when aged 0 to 2 years.

The relationship between malnutrition and child mortality has been the subject of extensive research in public health and medicine (Martorell and Ho 1984; Puffer and Serrano 1973; Pelletier *et al.* 1993; Prentice *et al.* 2008). Though mortality studies examine well-defined outcomes, mortality is a rare event so researchers often utilize large cross-sectional datasets. This creates methodological problems because appropriate instruments are often much more difficult to find in cross-sectional data. I present only a very brief overview of select findings from this literature; specifically, those findings related to the impact of malnutrition on the severity and duration of chronic illness, and its possible contribution to premature mortality.

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[15] In addition to increased rates of physical disability, Almond (2006) also examined other long-term outcomes. He found that adults exposed to the 1918 flu epidemic during fetal development experienced lower educational attainment and lower income compared with other birth cohorts.

Puffer and Serrano (1973) provided the first systematic attempt to estimate the contribution of malnutrition to child mortality. As with much of the early research, the authors implicitly presumed that malnutrition's contribution is additive and, therefore, that one can partition deaths into those due to malnutrition and those due to other causes. In contrast, Pelletier *et al.* (1993) showed that the synergy between malnutrition and infectious disease means that the mortality rate at a given level of disease exposure depends on a potentiating effect of malnutrition, not on an additive effect. Thus 'disease burden' studies, which implicitly assume an additive contribution of malnutrition to mortality, are based on inappropriate assumptions. Though Pelletier *et al.* (1993) and others have attempted to account for the synergistic relationship between malnutrition and mortality/morbidity, they fail to account for other factors, such as greater disease exposure and less effective medical care among the malnourished, which might jointly determine both health outcomes.

### 2.4 Review of the Literature on Malnutrition and Non-Health Outcomes

There is a growing body of literature, primarily within economics, on the long-term consequences of early childhood malnutrition. The focus has largely been on human capital outcomes in education, cognition and income. Because it is focused on long-term outcomes, this literature does not examine acute illness. I present a very brief overview of this literature.

There are numerous cross-sectional studies that document associations between pre-school nutritional status and subsequent human capital attainments, see Pollitt (1990), Behrman (1996) and Grantham-McGregor *et al.* (1999). However, as Behrman (1996) notes, many of these studies document associations rather than casual relationships between pre-school malnutrition and subsequent attainments. Since pre-schooler health and subsequent educational attainments both reflect household decisions regarding investments in children's human capital, associations in cross-sectional data may substantially over- or understate true causal effects.

Victora *et al.* (2008) review the associations between maternal and child undernutrition and risk of disease in adulthood and long-term human capital outcomes. Based on a review of longitudinal studies in five different developing country contexts, they find many indicators of maternal and child malnutrition (including maternal height; birthweight; and height, weight and BMI at 2 years of age) are associated with adult outcomes (including height, educational attainment and income; and chronic health indicators such as glucose concentration and blood pressure).

Relying on primarily natural experiments and non-experimental methods to establish causality, another growing body of work focuses on the specific impact of malnutrition on long-term human capital outcomes, including education outcomes (Maluccio *et al.* 2006; Glewwe and Jacoby 1995; Glewwe and King

2001; Alderman, Hoddinott and Kinsey 2006; Alderman *et al.* 2001; Behrman 1996; Behrman and Lavy 1998; Alderman, Hoogeveen and Rossi 2009; Case and Paxson 2008), cognitive outcomes (Maluccio *et al.* 2006; Case and Paxson 2008; Glewwe and King 2001) and earnings (Hoddinott *et al.* 2008; Thomas and Strauss 1997; Behrman and Deolalikar 1989). A small subset of this work has been done in experimental settings (Maluccio *et al.* 2006; Thomas *et al.* 2004). Using longitudinal data from Guatemala and Zimbabwe, two recent studies have used instrumental variables procedures to establish a causal link between early childhood malnutrition and education outcomes. Alderman, Hoddinott and Kinsey (2006) studied the impact of pre-school malnutrition on subsequent human capital formation in rural Zimbabwe using a maternal fixed effects-instrumental variables (MFE-IV) estimator with a panel dataset. Representations of civil war and drought shocks are used to identify differences in pre-school nutritional status across siblings. Improvements in height-for-age in preschoolers are associated with increased height as a young adult and number of grades of schooling completed. Glewwe and King (2001) used longitudinal data from the Philippines to establish a causal relationship between preschooler's height-for-age (which they instrumented with an older sibling's height-for-age) and subsequent school attainment and scores on standardized tests.

Undernutrition, especially that linked to famine during fetal development *in utero*, has also been linked to a variety of cognitive outcomes. Pitcher *et al.* (2006) established the link between undernutrition and structural damage in the brain,

as well as impaired motor development and exploratory behavior in infants. St. Clair *et al.* (2005) examined effects of a famine in China while Roseboom *et al.* (2001) studied mental health outcomes among survivors of the Dutch Famine.

While this literature highlights the importance of long-term consequences of early childhood malnutrition, another strand of economics literature focuses on acute health outcomes through exploration of the socioeconomic and environmental determinants of morbidity. This literature ignores nutritional status as a potentially important determinant, and often fails to account (explicitly or implicitly) for the infectious nature of acute illness. I turn to a review of this literature next.

### 2.5 Review of the Literature on Socio-Economic Determinants of Morbidity and Mortality

A large body of literature focuses on the determinants of individual and household morbidity and mortality in order to inform policies designed to reduce the burden of disease. The majority of these studies examine the socioeconomic determinants of morbidity outcomes (such as maternal education, household income and access to health care) (Strauss and Thomas 1998, Behrman and Deolalikar 1989; Jalan and Ravallion 2003; Frankenberg 1995; Gertler 2004; Glewwe 1999; Rosenzweig and Schultz 1983), or the influence of hygiene and sanitation factors on acute illness outcomes (Harrington *et al.* 1989; Jalan and

Ravallion 2003; Alberini *et al.* 1996). I provide a brief overview of this literature including the main questions addressed and the main findings.

### 2.5.1. Water, Sanitation and Hygiene

Access to improved drinking water is widely advocated as an effective way to reduce diarrhea-related morbidity and mortality, particularly in the poorest areas of the world and in the most vulnerable populations (i.e., children under five) (Huicho *et al.* 2008). Millennium Development Goal (MDG) 7 explicitly recognizes the importance of increasing access to clean water. Investments in water supply and sanitation have been shown to affect health through reductions in diarrhea, reduction in parasitic infections, increased child growth, and reduced morbidity and mortality (Feacham *et al.* 1983, Esrey *et al.* 1991; Hutley *et al* 1997). A number of studies in the economics literature have examined the impact of increased access to clean water and sanitation on the incidence of diarrheal illness (Jalan and Ravallion 2003; Zwane and Kremer 2007; Gasana *et al.* 2002). Studies have generally found that prevalence and duration of diarrhea are significantly less (on average) for families with piped water than for families without it. However, health gains may bypass poor families, particularly for children under five, when the mother is poorly educated (Jalan and Ravallion 2003), or when interruptions in piped water supply decrease the ability to engage in defensive behavior (Alberini *et al.* 1996).

Improvements in the quantity and quality of water supply, as well as improved sanitation, do not, however, automatically result in improved health (Zwane and Kremer 2007; Kremer 2006). As Zwane and Kremer (2007) argue, increasing uptake of personal defensive behaviors such as handwashing and point-of-use water treatment may be more effective interventions to reduce diarrhea than undertaking expensive piped water and sanitation infrastructure projects. In a study of the determinants of diarrhea in the Rwandan context, Gasana *et al.* (2002) found that contamination at the source is a less important risk factor in explaining childhood diarrheal incidence than contamination of unclean utensils to transport and store water, which highlights the importance of improving health education with a focus on increasing defensive behavior. Findings from a randomized evaluation of spring protection in rural Kenya (Kremer 2006) suggest that while spring water protection can lead to large improvements in source water quality as measured by the fecal indicator bacteria *E. coli,* such an investment may not reduce the incidence of childhood diarrhea.

Handwashing and home drinking water disinfection have, however, been shown to be very effective in reducing the incidence of diarrhea in young children. A 2002 WHO report suggested that handwashing with soap at appropriate times could reduce diarrhea incidence by more than 40 percent (WHO 2002). This is supported by recent meta-analyses (Curtis and Cairncross 2003; Fewtrell *et al.* 2005; Rabie and Curtis 2006) which suggested that handwashing can reduce the incidence of both diarrheal and respiratory infections. Results of a cluster-

randomized controlled trial suggest that handwashing promotion has the potential to reduce student absenteeism by nearly half (Bowen *et al.* 2002).[16] The effectiveness of home drinking water disinfection has been studied in several dozen randomized controlled trials. A recent meta-analysis (Clasen *et al.* 2007) suggested that point-of-use treatment can reduce the incidence of diarrhea by 50 percent.

### 2.5.2 Access to Medical Care

Access to medical care has been found to have a positive effect on child health outcomes though there is some debate in the literature as to the mechanism through which the access-health link is mediated. In addition, given that "access" to health services is typically measured as distance to a health provider or the number of health services reportedly available to the household, access may be serving as a proxy for general level of development. This may result in counterintuitive results due to a failure to control for quality of services, or because larger facilities may be disproportionately located in areas where health outcomes are poor (i.e., endogenous program placement). In an urban Brazilian setting, Thomas, Strauss and Henriques (1990) find a higher per capita number of nurses within municipalities is associated with being stunted among children of literate mothers. Increasing numbers of hospital beds per capita are also associated with shorter children. In Cote d'Ivoire, increasing distance to health

[16] In an earlier study in Lombok, Indonesia, Wilson *et al.* (1991) reported even more dramatic results of a hygiene intervention. The authors report an 89 percent reduction in diarrhea episodes in a treatment group of households with children under five, where the children's mothers were given soap and an explanation of the fecal-oral route of diarrhea transmission.

facilities is associated with lower height and weight-for-height, though the coefficients are small and statistically insignificant (Strauss 1990). Using data from the 1973 Colombia census, Rosenzweig and Schultz (1983) found that clinics and hospital beds are associated with lower child mortality ratios particularly in urban areas, but the effects of these variables decline dramatically with the addition of controls for municipality-level average women's schooling attainment.

Other studies provide more definitive evidence of the importance of access to health care for health outcomes. In a study of the effects of access to health care on infant mortality in Indonesia, Frankenberg (1995) found that adding one maternity clinic to a village decreased the odds of infant mortality by almost 15 percent, in comparison to the risk before the clinic was added. Adding one additional doctor reduced the odds of infant mortality within the village by about 1.7 percent. In Bangladesh, Al-Kabir (1984) found similar evidence for the hypothesis that proximity to care decreases mortality. In that context, the author found mortality rates among children living further than ten miles from a hospital were 40 percent higher than those for children living within three miles of a hospital. Mortality rates among neonates were found to be 30 percent higher when the household was located further than ten miles from a maternity clinic. Using data from the 1971 India census, Rosenzweig and Wolpin (1982) simulated the effect of increasing facility availability, and suggested that doubling the number of villages with family planning programs would reduce the child

mortality rate by about 10 percent, while doubling the number of villages with dispensaries would lower the rate by 25 percent in India.

Gertler's (2004) study of the impacts of Mexico's *Progresa* conditional cash transfers on children's health outcomes exploited exogenous variation in utilization of health care services across villages (due to *Progresa*), to estimate the impact of the services on child health. The author found that newborns in treatment villages were 25 percent less likely, and children under 3 years were 22 percent less likely than their counterparts in the control villages to be reported as being ill in the previous month. These results may be confounded by changes in children's nutrition however, as the study also found that children in program areas were less likely to be stunted or anemic relative to children in control villages.

### 2.5.3. Maternal Education

Research has consistently demonstrated a strong correlation between women's education and children's morbidity and mortality outcomes. Results suggest that children of more educated mothers have better health outcomes; however, as with access to care, the exact mechanism through which maternal education is translated into improved child health is not clear.[17] Glewwe (1999) outlined three possible mechanisms. First, exposure to modern science and methods in school may make women more receptive to modern medical treatments. Second,

[17] Here, I am focusing solely on key findings and outstanding research questions. For a more comprehensive review of this literature, see Behrman and Deolalikar (1988) Behrman (1990), and Strauss and Thomas (1995).

literacy and numeracy skills acquired in school may assist future mothers in diagnosing and treating child health problems. Third, formal education may directly teach health knowledge to future mothers. Research in different settings has produced mixed results.

Increased use of preventive and curative services by more educated mothers has been found in multiple cultural and socio-economic contexts (Abbas and Walker 1986; Akin *et al.* 1986, Wong *et al.* 1987; Streatfield, Singarimbun and Diamond 1990). As Barrera (1990) and Caldwell (1990) argue, maternal education increases the use of health-care services and compliance with recommended treatments, presumably because education changes a mother's knowledge and perception of the importance of modern medicine in the care of her children. As Caldwell (1990) argues, increased maternal education may reflect development of mother's cognitive skills, exposure to new ideas and 'modern' institutions which increase the likelihood of use of health care.

Using data from Morocco, Glewwe (1999) found that while health knowledge appears to be the most important skill that mothers gain through education, education contributes to health knowledge only indirectly via use of literacy and numeracy skills acquired in school.

Rosenzweig and Schultz (1983) argued that female schooling and health-care services may in fact act as partial substitutes for information regarding

knowledge of diseases, treatment of illness and child-care practices. Using data from Colombia, the authors found that the effect of education on child health becomes less important as access to public health-care services improves. Their findings suggested that any advantage conferred by schooling on health outcomes is narrowed in areas where health services are readily accessible to women of varying educational backgrounds. These findings are consistent with other studies (Schultz 1996) that have shown that differentials in child mortality by maternal education are less pronounced in countries with strong public-health programs such as Cuba and Costa Rica.

One possible critique of findings that mother's education improves child health is the hypothesis that education simply reflects unobserved maternal characteristics. Behrman and Wolfe (1987) and Wolfe and Behrman (1987) argued that the association between female schooling and health outcomes may simply reflect the mother's childhood background. The authors suggested that maternal education serves as a proxy for human capital more generally, including health-related skills and habits acquired during childhood. They argued that when mother's childhood background is excluded as an explanatory variable, the impact of maternal education on child health outcomes may be overstated. Based on Nicaraguan data on mothers' siblings to control for unobserved family fixed effects, they found that applying these controls leaves no significant effect of mother's education on child anthropometric status. However, using data from Cote d'Ivoire, Strauss (1990) found that mother's education raises child height-

for-age and weight-for-height even after controlling for family unobservables. As discussed in Chapter 7, my findings are consistent with the findings in Behrman and Wolfe (1987) and Wolfe and Behrman (1987).

Evidence from two studies on the determinants of child nutritional status hints at some of the complexities that may explain the mixed results regarding the impact of socio-economic variables on children's health outcomes. In a study of the determinants of child nutritional status in Mozambique, Sahn and Alderman (1997) found that although maternal education was a significant determinant of anthropometric measures for younger children, it was not for older children. They also found that increases in household income affect the nutritional status of children two and older but not younger children. An important policy implication is that education and behavior change interventions designed to improve childcare practices can be targeted at mothers of younger children, but these children are not likely to achieve improved growth from income-support programs.

In a study on the impact of maternal education on children's nutritional outcomes in Lesotho, Ruel *et a*l. (1992) tested whether maternal nutrition knowledge was a mediating factor in the association between maternal schooling and child nutritional status, as well as whether the mechanism involved differed according to socioeconomic status. In that study, maternal schooling and the mechanism by which it affected the child's weight-for-age were contingent on the family's

socioeconomic status. The authors reported that maternal schooling was positively associated with weight-for-age for both wealthier and poorer households, but that the size of the effect was much larger for poorer households. The effect of maternal schooling on weight-for-age was mediated by the mother's nutrition knowledge only among wealthier households. These findings suggest that improving children's growth in poorer households in Lesotho would require increasing access to a minimum level of resources in order to realize the benefits of any nutrition education.

### 2.5.4. Household Income

Studies that examine the impact of household income on morbidity and mortality outcomes have also found mixed results. Assuming health is a normal good, economic theory would suggest that increases in income would lead to increased use of health goods and services, presumably leading to a decrease in morbidity and mortality. Indeed, there is strong micro-level evidence that income affects health. In Brazil, Thomas (1990) estimated large effects of mother's non-earned income on child health. While several studies have shown that greater household income is associated with a lower likelihood of low birth weight or stunting (Martorell and Scrimshaw 1995; Kramer 1987a; Kramer 1987b; Kramer 2000.),[18]

[18] Though the studies are not designed to estimate the impact of income on any anthropometric indicator, such as stunting or underweight, several studies in developing country contexts have examined the role of income in food consumption, an obvious factor in nutrition outcomes. In Sierra Leone, Strauss (1984) finds significant effects of income on nutritional status. Deaton and Subramanian (1996) report similar findings for South India. Behrman and Deolalikar (1989) provide evidence that increases in incomes lead to increases in caloric consumption, especially at low levels of income.

studies of illness suggest some measure of social conditioning[19] may result in a greater likelihood of *reporting* of illness among wealthier households. In a study of illness reporting in Nepal, Pokhrel (2007) found that income seems to have a modest but significant effect on illness reporting, which supports medical anthropologists' observations that people belonging to different economic groups understand illness differently. Helman (2001) supports this hypothesis and suggests that non-poor households are more likely to report their child as ill than poor households because their income makes 'illness management' more affordable to them.

### 2.5.5. Exposure to Pathogens

Many studies implicitly model risk of disease transmission as a function of access to clean water and sanitation, a potential proxy for disease prevalence. Only a handful of studies in the social sciences have attempted to model disease transmission more explicitly, with the majority of these focused on HIV/AIDS (Kremer 1996; Geoffard and Philipson 1997; Geoffard and Philipson 1996; Philipson and Posner 1993). Using data from Cambodia, Deolilikar and Laxminirayan (2000) examined the socioeconomic determinants of disease transmission.[20] They highlighted the importance of avoiding omitted variable bias by including a measure of the prevailing level of infection among the risk factors

[19] Nobel prize winner Amartya Sen developed the concept of adaptation and cultural or social "conditioning" to explain this phenomenon when comparing self-reported measures in developing versus developed countries, and for different socioeconomics groups within developing countries (Sen 1985).

[20] Despite its potential importance as a determinant of acute illness, Deolalikar and Laxminirayan (2000) do not control for nutritional status.

for infectious disease incidence. They argued that an assumption of uniform exposure to infected individuals will lead to biased estimates of the impact of water and sanitation variables on the probability of illness. .

Ideally, one would measure disease prevalence as the number of other individuals ill within a respondent's community at the time an individual respondent's health is measured. However, independent disease prevalence data is difficult to obtain, and rarely is collected at the same point in time as household surveys illicit health information. One option is to explain incidence with a measure of prevalence generated from the same survey data. However, this creates a problem of reverse causality. Recognizing the endogeneity of this variable, Deolalikar and Laminirayan (2000) proxied disease prevalence by including the mean values of three village characteristics (age, education, sex). Such village-level means likely capture many characteristics about the village that may influence prevalence but likely do so weakly. Faced with a similar issue, I nonparametrically control for location differences in disease prevalence by including district fixed effects.[21] Any differences in district-level factors, such as infrastructure, sanitation and general level of development, will be absorbed by these controls as my specification exploits only within-district variation. I also control for seasonal fluctuations in disease prevalence by including month-of-interview dummies, which control for the fact that the IFLS interviews were conducted throughout a calendar year.

[21] I do not have enough variation within villages to be able to include village fixed effects.

## 2.6 Contribution to the Literature

While the economics literature has contributed to an improved understanding of policy-relevant factors that influence child health outcomes, this literature has ignored the importance of nutritional status as a risk factor in the development of acute illness. Although much of this literature recognizes the importance of initial health endowments (e.g., genetics, nutritional status, prior health conditions) in determining health outcomes, no study explicitly accounts for nutritional status as a proximate determinant of morbidity. In so doing, this literature ignores the importance of nutritional status as a risk factor in determining heterogeneous risk of illness. Even if both genetic endowments and exposure were homogenous, variations in nutritional status may result in heterogeneous risk of infection and/or risk of more severe disease.

This dissertation contributes to the literature by testing two hypotheses: (1) chronic undernutrition in early childhood, as measured by stunting in children under five, increases the probability of contemporaneous acute illness; and (2) there is a significant effect of early childhood undernutrition on the probability of developing acute illness later in childhood. I estimate a model that predicts the incidence of febrile, diarrheal and respiratory disease, diseases which combined account for the preponderance of morbidity and mortality in children in developing countries. I control for household socio-economic status and multiple environmental factors that influence disease transmission. I avoid the pitfalls of endogeneity by using instrumental variables to identify the impact of

early childhood malnutrition on acute illness. I measure malnutrition by stunting (low height-for-age) because it reflects only long-term deficiencies. This avoids the potential reverse causality that might occur if one used weight-for-age or BMI as the measure of malnutrition in the context of acute illness. Finally, by focusing on contemporaneous and longer-term acute illness outcomes in children who are identified as malnourished by age five, this research may contribute to the broader literature on human capital formation.

## Chapter 3 Empirical Model

In this chapter, I briefly review the main empirical challenge when attempting to explain health outcomes. I then present a framework to explain acute health outcomes, and outline the empirical strategy I employ to estimate my equations of interest.

### 3.1 The Empirical Challenge

Although studies in the medical and nutrition literature have firmly established a correlation between malnutrition and acute illness, it is very difficult to establish the causal link from malnutrition to acute illness. Linking early childhood nutrition with later childhood outcomes is difficult because few datasets have sufficiently long panels to make such linkages. Much of the evidence linking childhood nutrition and later human capital outcomes is associational (Behrman 1996) and based on cross-sectional data. More importantly, even studies with panel data often suffer from serious identification problems because it is difficult to disentangle the effects of nutritional investments or other determinants of nutritional outcomes (e.g., environmental factors such as exposure to pathogens) from those of other investments that affect child health outcomes. Household behavioral decisions that influence investment in a child's nutrition and growth are very likely correlated with other household decisions that would affect a child's probability of illness, such as decisions to invest in hygiene and sanitation or a mother's knowledge and use of appropriate feeding practices.

### 3.1 Analytical Framework

I am interested in estimating the effect of nutritional status on the likelihood of acute illness. Specifically, I wish to establish whether early childhood malnutrition, as measured by stunting in children ages 0 to 5, affects the likelihood of acute illness and, if so, what the magnitude of the effect is. I also wish to determine whether this effect persists over time, or dissipates if a child experiences sufficient "catch-up" growth such that early childhood stunting is reversed in later years.

Mosley and Chen (1984) provide a useful analytical framework for the study of child health outcomes which integrates socio-economic and cultural variables that influence the risk of disease via behaviors, and medical science, which focuses on disease processes. I borrow from their framework, and emphasize the specific role of nutritional status as a determinant of acute illness outcomes.

The likelihood of an individual developing acute illness depends on the complex interplay of four primary factors:[22]

1. The amount of pathogens present in the household and community. Unlike other factors, the pathogenic agent is a necessary input into the disease production process, without which a specific disease cannot occur.

[22] Incidence is the rate at which new cases occur in a population during a specified time period. Prevalence, which partially explains incidence, is the proportion of population that is infected at any one time.

Agents include bacteria, viruses, parasites (protozoa or helminthes) and fungi;

2. Whether the person has knowledge of how to prevent infection;
3. The physical ability to prevent infection through averting behavior (e.g., whether the individual has access to clean water for drinking, cooking and handwashing, sanitation, proper ventilation, avoidance of vectors especially mosquitoes and ticks); and
4. If infected, factors that influence whether an individual will develop clinical symptoms include immunity (e.g., because of prior illness), age, nutritional status, general health, habits (e.g., hygiene, smoking, exercise), genetics, and size of airway for respiratory illness.

Once ill, factors that influence severity of illness include immunity, nutritional status, general health, genetics, health-related habits (e.g., smoking), size of airway for respiratory illness (generally a function of age), and physical size (also generally a function of age since age, or more specifically body mass, determines blood and gut volume for example).

Nearly all theoretical work on disease transmission[23] builds upon seminal work by Kermack-McKendrick (1927), who developed a basic model which allows us to characterize the process of infection as:

[23] See, for example, Bailey (1975), Becker (1989), Isham and Medley (1996), Collett and Lye (1987), Feng and Velasco-Hernandez (1997), Diallo (1999), Ghosh and Tapaswi (1999). For examples within the economics literature, see Kremer (1996), Kremer and Morcom (1998), Geoffard and Philipson (1997) and Geoffard and Philipson (1996).

$$\dot{I} = \beta . I . (1 - I) - r(n, c) . I \qquad [3.1]$$

where

*I* = the fraction of the population that is infected

$\beta$ = transmission coefficient of illness and a function of the virulence of the infectious agent, and of the individual's and/or household's defensive behavior;

*r* = the rate of recovery from illness (i.e., duration of illness) which is, in turn, a function of nutritional status and curative care.

The function, *r(n,c)*, represents the health production function. The conditional probability of acquiring an acute illness can be written as: P(acute illness) $= f(I, n, c)$.

The basic intuition of this model is that the fraction of population infected is an increasing function of the transmission coefficient, and a decreasing function of the rate of recovery and nutritional status. Moreover, infection is more likely to spread the lower the defensive habits of households, the lower the recovery rate from illness and the poorer the nutritional status of individuals.

Although this model is very intuitive and lends itself as a useful building block for many theoretical models in the literature, unfortunately, estimating the model parameters empirically is a significant challenge as it is not possible to observe the number of people infected in a population. Moreover, simply regressing acute

illness on nutritional status will lead to biased estimates due to endogeneity resulting from reverse causality and possible omitted variables. In the contemporaneous case, we cannot determine whether poor nutritional status increases the likelihood of acute illness or whether acute illness increases the likelihood of poorer nutritional status. Furthermore, in both the contemporaneous and lagged timeframes, there may be some unobservable factors, such as maternal knowledge and use of proper feeding and hygiene practices, that affect both a child's nutritional status and illness outcomes. Omitting variables that capture unobserved heterogeneity from the regression specification will lead to biased estimates.

In order to isolate the potential effect of early childhood malnutrition on acute illness outcomes, and determine the magnitude of the effect, I need to estimate a model where the right-hand side includes nutritional status and other controls. In order to address endogeneity of nutritional status, I employ an instrumental variables strategy. The empirical design and choice of instruments are explained below.

### 3.2 Estimation Strategy

I employ an instrumental variable strategy to overcome the various empirical challenges identified in the previous subsection. Briefly, I need to find instruments that are highly correlated with the variable suspected of being endogenous (being stunted) but that are not correlated with the outcome of

interest (acute illness) other than via stunted growth. The ideal approach is to identify an exogenous event that may have caused an abrupt change in nutritional status, but which cannot have directly caused the acute health outcome. This exogenous event could be a war, famine, natural disaster, relocation, or a new policy that provides or removes assistance. In different contexts, previous literature has used drought shocks, price shocks and disruptions associated with civil war as identifying instruments for early nutritional status on future human capital outcomes.[24] When examining two health outcomes, if the shock affected only past nutritional outcomes and not the health outcome of interest, such an instrument might be appropriate. As Glewwe and King (2001) and Alderman *et al.* (2001) note, in practice finding a suitable instrument is often very challenging.

Absent such shocks in Indonesia for the time period of my data,[25] I rely instead on a more readily-available and arguably more natural set of instruments for child's height: parents' heights and child's birth quarter. Parents' heights are highly correlated with a child's height because they are a genetic determinant of

[24] Alderman, Hoddinott and Kinsey (2006) use both drought shocks and civil war in Zimbabwe as identifying instruments in an analysis of the impact of pre-school nutrition on adult educational outcomes height attainment. Alderman, Behrman, Lavy and Menon (2001) use price shocks to assess the impact of preschooler height on school enrollment. Adelman (2009) uses disruption associated with civil war in Uganda to identify the impact of social networks on child height.

[25] In a recent paper, Maccini and Wang (2009) use historic rainfall data for Indonesia to test for the effect of early-life environmental exposures on later human capital outcomes. I investigated the use of rainfall data from the same source (GHCN), but found that rainfall data from a sufficient number of weather stations was not available through 1993/94, which made it infeasible to instrument stunting status with rainfall for my sample. I also considered using a policy shock as a possible instrument. The Government of Indonesia instituted a national school feeding program in 1994. However, my research focuses on preschool age children whereas this policy shock would only be an appropriate instrument for studying impacts on older school-age children.

the latter, but are presumably not correlated with whether or not the child experiences acute illness in childhood or early adolescence. Presumably, any association between parents' height and childhood acute illness is due entirely to early childhood stunting. (Indeed, as shown in Chapter 6, stunted growth status is well predicted by parents' heights.)[26]

In addition to parent's heights, I use birth quarter dummies as instruments for a child's stunting status. Birth quarters have been found to be potentially important predictors of health status, presumably because they pick up the relation between time of birth and harvest season, which in turn influences the availability of food.[27] Lokshin and Radakan (2009) use data from three waves of India's National Family Health Survey to investigate the relationship between health outcome and month of birth. They find that seasonal variation in environmental conditions at the time of birth is an important determinant of health outcomes of young children. Assuming that these sets of identifying instruments are valid, instrumental variable estimation produces consistent estimates of the coefficient on the stunted growth regressor. Instrumental variable estimation also removes any measurement error surrounding the stunted growth status, which may arise from poorly measured height or incorrectly recorded age. As long as

[26] Interestingly, Glewwe (1999) uses the heights of both parents as explanatory variables to partially control for the researcher's inability to directly observe a child's health endowment.

[27] A large body of research has shown that birth season is associated with health outcomes such as increased risk of premature adult mortality (Moore *et al.* 2004), developing schizophrenia (Tochigi *et al.* 2004; Davies *et al.* 2003; Torrey *et al.* 1997), and life expectancy among the elderly (Costa and Lahey 2005).

the measurement error is classical, it typically results in attenuation bias (i.e., the estimated coefficient is downward biased).

The main regression specifications exploit individual-level variation in stunted status in a cohort of children aged 0 to 5 in 1993 to measure the contemporaneous effects of stunting on acute illness outcomes. I also exploit the panel nature of my dataset to observe disease outcomes for the cohort of children aged 0 to 5 in 1993 when children are four, and then seven, years older (in 1997 and 2000, respectively). Hence, these specifications explore how stunting during a critical period of development affects the contemporaneous health status of children as well as how stunting affects the probability of acute illness as the children age four and seven years into the future.

*Main Regression Specification:*

To measure the contemporaneous impact of stunted growth status on acute illness, I use two-stage least squares (2SLS). In the first stage, I regress stunted growth status on a set of instruments; namely, all of the exogenous variables in the main structural equation, plus my set of identifying instruments. Formally, the first-stage regression is:

$$Stunted_{93} = \alpha + \beta_1.MotherHeight + \beta_2.FatherHeight + \sum_{i=1}^{3} \gamma_i.BirthQuarter_i + \delta.X + \varphi.H + \mu.V + \theta.D + \varepsilon \quad [3.2]$$

where $Stunted_{93}$ is a binary variable equal to 1 if a child is stunted, $\beta_1$ and $\beta_2$ are the coefficients on mother's height and father's height, respectively, and $\gamma_1$, $\gamma_2$ and $\gamma_3$ are the coefficients on the first three birth quarter dummies, respectively. Note that birth quarter 4 is the omitted category. In terms of control variables, $X$ is a vector of individual-level factors such as age and gender, $H$ is a vector of household characteristics, $V$ is a vector of village-level factors such as rural/urban locality, and $D$ is a vector of district-level dummies. $\alpha$ is the constant and $\varepsilon$ is a mean-zero error term.

The predicted values from this first stage regression, $\widehat{Stunted}_{93}$, replace the stunting status used in the second stage regression:

$$Y = \alpha' + \beta'.\widehat{Stunted}_{93} + \delta'.X + \varphi'.H + \mu'.V + \theta'.D + \varepsilon' \qquad [3.3]$$

where $Y$ is a binary variable equal to 1 if a child reports as having an acute illness. $\beta'$ is the main coefficient of interest since it represents the impact of a child being stunted on the likelihood of acute illness. All other controls are the same as in the first stage.

*Later Childhood Results*

The panel nature of my dataset allows me to follow the same individuals over time. Specifically, I can trace illness outcomes for children who were aged 0-5 in 1993 as they age four years, then seven years, into the future. When estimating

whether early childhood nutritional status affects future acute illness outcomes, potential bias may arise due to omitted variable biases. I explore this possibility by comparing results from estimations based on a linear probability model of illness outcomes in 1997 and 2000 on stunting in 1993, and employing instrumental variables in a 2SLS specification. Chapter 7 presents the estimation results. For comparison, I also present results using a probit model and report the marginal effects.

The basic regression for measuring the impact of stunted growth four years into the future is the following:

$$Y_{97} = \tilde{\alpha} + \tilde{\beta}.\widehat{Stunted}_{93} + \tilde{\delta}.X + \tilde{\varphi}.H + \tilde{\mu}.V + \tilde{\theta}.D + \tilde{\varepsilon} \qquad [3.4]$$

where $Y_{97}$ is a binary variable equal to 1 if a child reports as having an acute illness in 1997. $\tilde{\beta}$ is the main coefficient of interest which represents the impact of a child being stunted in 1993 on the likelihood of acute illness in 1997. The basic regression for measuring the impact of stunted growth seven years into the future is the same after replacing 1997 values with 2000 values.

Although stunted growth status can be assumed predetermined in equation [3.4], endogeneity may still be present. For instance, there may be unobservable household characteristics that influence both a child's nutritional status, and hence physical development, and risk of acute illness. Households that have a

great deal of experience with illness may be relatively more knowledgeable about prevention and self-treatment, for example, or may be less inclined to report a specific acute illness episode (i.e., Sen's social conditioning) (Sen 1985; Helman 2001). Importantly, while I control for mother's formal education, it may be a mother's *informa*l education and social practices, which I do not observe and hence cannot control for, that play a greater role in determining risk of malnutrition and disease transmission.

To remove possible endogeneity, I instrument for stunting status. It is also possible to control for unobserved heterogeneity by including household-specific fixed effects. As long as the unobserved factors remain unchanged over the children in the same household, the "within" estimation procedure gives a consistent estimate of the coefficient on stunted status, even if the unobserved factors are correlated with stunted growth. For this estimation approach to work, there must be variation within the household in the health and stunted growth of the children that are part of that household.

Unfortunately, since my analysis is restricted to children aged 0-5 in 1993, I do not have a sufficient number of households that meet these requirements: most of my households are comprised of children who are all healthy, or children who are all stunted. The best I can do, therefore, is to include district fixed effects, which control for all observable and unobservable differences in socio-

demographics, infrastructure, sanitation, environment and climate, across locales. The results are discussed in Chapter 7.

## Chapter 4 The Setting

This chapter provides an overview of the geographic setting from which my data are drawn, with a focus on Indonesia's burden of malnutrition and infectious disease.

### 4.1 Indonesia: Overview

An archipelago in Southeast Asia, Indonesia is made up of over 13,000 islands straddling the equator, of which about 6,000 are inhabited. Independent from the Dutch since 1949, there is considerable variation across the archipelago in terms of culture, ethnicity and level of economic development. With a population of 238 million in 2008, Indonesia is the fourth most populous country after China, India and the United States, and the most populous Muslim country (U.S. Department of State 2009).

Population density is extremely variable across and within provinces. According to the most recent census, overall population density is estimated at 109 people per square kilometer; however, the majority of the people live on the islands of Java and Bali (over two-thirds of the population lives on Java alone). The population density is 12,635 people per square kilometer on Java, 559 people per square kilometer on Bali, and 99 people per square kilometer in West Sumatra (BPS 2009). In 2008, the urban population was estimated at 52 percent (CIA 2009).

The majority of islands consist of rugged volcanic mountains covered by dense tropical forests; however, some islands have swamps and plains. Elevation ranges from sea level along coastlines to over 16,000 feet in the Sudirman Mountain Range on Papua (formerly Irian Jaya). Indonesia has a tropical climate with an average daily temperature range of 70°to 9 0°F, with 80 to 90 percent humidity in the lowlands. Temperatures and humidity are lower as elevation increases, while temperature below freezing can occur at elevations over 9,800 feet. There are two seasons; a wet season which generally runs October/November to March/April, and a dry season which generally runs April/May to September/October. Annual rainfall averages 40-78 inches, (1.02-1.93 meters per year), though there is considerable variability in mean rainfall even within provinces (BPS 2009). This variation in environment makes Indonesia a particularly rich laboratory for studies related to infectious disease.

The nation was divided into 27 provinces, 241 districts and 3625 subdistricts in 1993, the year in which the first wave of the IFLS was fielded. Since 1993, there have been several redrawings of administrative boundaries, and independence for the former province of Papua (now Irian Jaya) so that current administrative divisions divide the country into 33 provinces, 440 districts, 91 municipalities, 5,263 sub-districts and 62,806 villages.

Indonesia is ranked 111th out of 182 countries on the 2007 Human Development Index (UNDP 2009a), which primarily reflects the government's strategic investments in health; average life expectancy at birth is relatively high – 70.5 years -- in spite of a relatively low GDP per capita of US$3,712 (in purchasing power parity (PPP)). Nonetheless, according to the United National Development Programme, more than 15 percent of the population lives below the national poverty line. Using the World Bank's under $2 per day (PPP) measure, nearly 50 percent of the population lived below the poverty line in 2006 (WFP 2006). The percentage share of income held by the poorest quintile has remained virtually unchanged in the last 15 years (9.3 percent in 1990 vs 9.7 percent in 2004). Despite the MDG of improved access to clean water and sanitation, the proportion with access has not increased significantly; more than 30 percent of urban dwellers and 50 percent of rural Indonesians still lack access to piped drinking water (UNDP 2007).

Government efforts to improve access to education, health and other basic services, particularly during the 1970s and 1980s, have resulted in considerable gains. By 1995, Indonesia had already more than halved the proportion of people whose income is less than $1 a day, and reduced prevalence of underweight children under five from 31.6 percent in 1995 to 28 percent in 2006 (UNDP 2007). According to official government statistics, the under five mortality rate (U5MR) declined by 73 percent between 1971 and 1999, from 218 per 1000 live births to 60 per 1000 live births. During that same period, Infant mortality

rates (IMR) declined by 68 percent, from 145 deaths per 1000 live births to 46 deaths per 1000 live births (BPS 2009). According to the World Health Organization, by 2005, the U5MR further declined to 40 per 1000 live births, and the IMR was 32 per 1000 live births (WHO 2006a). This places Indonesia's IMR on par with, among others, the nations of Georgia, Iran, Nicaragua, Kazakhstan, Egypt and Algeria. However, compared to other Southeast Asian nations, the IMR is still high – Indonesia's IMR is 4.6 times higher than Malaysia, 1.3 times higher than the Philippines and 1.8 times higher than Thailand (UNDP 2007).

National figures mask considerable regional and socioeconomics disparities in health indicators. The poor, rural and less educated classes generally suffer much higher mortality rates than their wealthier, urban and more educated counterparts. According to UNDP data for 2005, the infant mortality rate among the poorest groups was 61 per 1000 live births versus 17 per 1000 live births among the richest (UNDP 2007). Especially wide geographical variation exists for infant, under five and maternal mortality. Figure 4.1 shows IMR rates by province over three five-year time periods immediately preceding the DHS survey year. This comparison is useful since the years in which the DHS were conducted correspond roughly to the IFLS survey years of 1993, 1997 and 2000. Based on the 1994 Indonesian DHS, which provides an estimate of the average IMR for the previous 5-year period (1989-1993), the IMR ranged from 30 per 1000 live births in Jakarta to 110 per 1000 live births in West Nusa Tenggara,

while the national rate (reported elsewhere) was 57 per 1000 live births for that same period (Measure DHS STATcompiler 2009).

**Figure 4.1: Infant Mortality Rates by Province per DHS[28]**

| Province (Island in bold) | 1994 DHS | 1997 DHS | 2002-2203 DHS |
|---|---|---|---|
| **Sumatra** | | | |
| North Sumatra | 61 | 45 | 42 |
| West Sumatra | 68 | 66 | 48 |
| Riau | 72 | 60 | 43 |
| Jambi | 60 | 68 | 41 |
| South Sumatera | 60 | 53 | 30 |
| Bengkulu | 74 | 72 | 53 |
| Lampung | 38 | 48 | 55 |
| Bangka Belitung* | n/a | n/a | 43 |
| **Java** | | | |
| DKI Jakarta | 30 | 26 | 35 |
| West Java | 89 | 61 | 44 |
| Central Java | 51 | 45 | 36 |
| DI Yogyakarta | 30 | 23 | 20 |
| East Java | 62 | 36 | 43 |
| Banten* | n/a | n/a | 38 |
| **Bali and Nusa Tenggara** | | | |
| Bali | 58 | 40 | 14 |
| West Nusa Tenggara | 110 | 111 | 74 |
| East Nusa Tenggara | 71 | 60 | 59 |
| **Kalimantan** | | | |
| West Kalimantan | 97 | 70 | 47 |
| Central Kalimantan | 16 | 55 | 40 |
| South Kalimantan | 83 | 17 | 45 |
| East Kalimantan | 61 | 51 | 42 |
| **Sulawesi** | | | |
| North Sulawesi | 66 | 48 | 25 |
| Central Sulawesi | 87 | 95 | 52 |
| South Sulawesi | 64 | 63 | 47 |
| Southeast Sulawesi | 79 | 78 | 67 |
| Gorontalo* | n/a | n/a | 77 |
| ***National Average for Previous 5 Years*** | **57** | **45.7** | **34.7** |

*Denotes provinces that were split off from South Sumatra, West Java, and North Sulawesi provinces, respectively, as a result of redrawing of administrative boundaries in 2001. Province-specific IMRs are therefore not available prior to the 2002-2003 DHS. *Source: UNDP 2007*

[28] The 2002-2003 Indonesian DHS did not include Nanggroe Aceh Darussalam, Maluku, North Maluku, and Papua province. Previous surveys included East Timor, which gained independence in 2002; n/a = not applicable

Indonesia continues to struggle with high maternal mortality. In 1990, nearly 60 percent of deliveries took place at home without a skilled birth attendant (the rates were 40 percent in urban and 76 percent in rural areas). Though government campaigns have increased access to village midwives, the maternal mortality ratio remains among the highest in Southeast Asia, at 307 per 100,000 live births (DHS 2003). The lifetime risk of a mother dying of causes related to childbirth is estimated to be 1 in 65, compared with 1 in 76 for all developing countries, 1 in 1,100 in Thailand and 1 in 8,000 in industrialized countries (WHO 2002; UNICEF 2008). While these figures are derived from the last nationally-representative household survey, more recent UNICEF estimates place the maternal mortality rate at 420 per 100,000 live births. Using 2005 data, the UN estimates that countries with similar MMR include South Africa, Bhutan and Yemen (UN 2009b). By comparison, the average MMR in industrialized countries is 8 per 100,000 live births. Sub-Saharan Africa suffers from the highest MMR at 920 maternal deaths per 100,000 live births, followed by South Asia with an MMR of 500 (UNICEF 2008).

### 4.2 Burden of Malnutrition in Indonesia

Like many of its Southeast Asian counterparts, Indonesia suffers from high rates of malnutrition measured in terms of stunting, underweight and wasting. Despite general improvements in food availability, health and social services in the last 25 years, hunger and malnutrition exist in some form in almost every district in Indonesia. At present, about half the population is iron-deficient and one-third is

at risk of iodine deficiency disorders. Vitamin A deficiency disorders affect nearly 10 million children (Atmarita 2005). Malnutrition is particularly prevalent in rural areas and among the relatively poor in both urban and rural areas. This variation in prevalence of nutritional status makes data from Indonesia particularly well-suited to the task at hand.

One study found prevalence of underweight (low weight-for-age) among Indonesian preschool children declined from 37.5 percent in 1989 to 27.5 percent in 2003, but that prevalence of stunting (low height-for-age) in preschool children changed little during the 1990s, from 44.5 percent in 1990 to 45.6 percent in 2001 (Atmarita 2005). Based on the IFLS, Strauss *et al.* (2004) found that prevalence of stunting among children under five declined from 43 percent to 33 percent for boys from 1997 to 2000, and from 40 percent to 33 percent for girls during the same three-year period, levels which are comparable to many sub-Saharan African countries. Using a pooled cross-section of children under five years of age from four rounds of the Indonesia Socio-Economic Household Survey (SUSENAS), Waters *et al.* (2004), estimated the prevalence of underweight as 37.7 percent in 1992 and 28.5 percent in 1999.[29]

[29] All of these studies used the old WHO/NCHS child growth standards, which used a U.S. reference population. In 2006, the WHO released new international child growth standards which use as a reference population a six-country cohort intended to be more representative of the diversity of growth experiences across both developing and developed countries. See WHO (2006a) for a general description of the new standards, and how the old NCHS/WHO and new standards compare with one another.

### 4.3 Burden of Infectious Disease in Indonesia

Communicable diseases continue to be the major cause of morbidity and mortality in Indonesia. Among all age groups, in terms of clinic caseload, the top ten infectious diseases across the archipelago are tuberculosis, malaria, dengue and dengue hemorrhagic fever, acute respiratory infections, dysentery and shigellosis and vaccine-preventable diseases such as neonatal tetanus, pertussis, measles, hepatitis B and polio (MOH 2007).[30] Among all ages, four of the top ten causes of death in 2002 were attributable to infectious disease: tuberculosis, lower respiratory infections, perinatal conditions (of which nearly 50 percent are due to infectious diseases), and diarrheal disease (WHO 2006a).

Although a reduction in under-five and infant mortality rates[31] reflects significant progress towards the MDGs, persistent high rates of death among Indonesian children within the first year of life (one-third of which occur within one month after birth), are a reflection of the quality of health care during prenatal, delivery and postnatal periods. Among the young, morbidity and mortality are dominated by preventable or treatable infections. The top three causes of infant mortality--acute respiratory infections, prenatal complications and diarrhea – account for 75 percent of all infant deaths (UNDP 2007). Between 2000-2003, the three major causes of under five mortality were diarrheal diseases (18 percent of all deaths), pneumonia (14 percent) and premature birth (12 percent)

[30] This reflects both inpatient and outpatient caseloads. Among inpatients, the greatest caseloads were due to diarrhea, typhoid and dengue hemorraghic fever (MOH 2007).

[31] The infant mortality rate is the fraction of children (expressed as a rate per 1,000 live births) who die before their first birthday.

(WHO 2005). More than 600,000 children under five presented with lower respiratory infections to public hospitals in 2005 alone (MOH 2007).

Indonesia has the third largest number of tuberculosis cases in the world. In 2005, the national prevalence rate of tuberculosis was 262 per 100,000 people (UNDP 2007). An estimated 250 people died of tuberculosis each day during 2006. Over half a million new cases are estimated to occur every year (WHO 2008b). Malaria is widespread throughout large parts of Indonesia. Over 50 percent of the population lives in malaria endemic areas, with the highest burden in the eastern provinces. There are an estimated 30 million cases of malaria each year, only 10 percent of which are treated at health facilities (WFP 2006).

Other infectious diseases are also common. Dengue is widespread and follows a similar seasonal pattern as malaria, usually increasing during the rainy season of October to March when mosquito vectors become more abundant (WFP 2006). Large-scale outbreaks of dengue haemorrhagic fever are reported every year. The HIV/AIDS rate is 2.65 per 100,000 but is accelerating sharply across the country, particularly among young women age 15-24. The threat of avian influenza remains a grave concern both within the country and for international public health. Ten years after ‘eradication’, poliomyelitis recently reappeared in several provinces. Long-ago eradicated in most other regions of the world, leprosy remains a threat in certain provinces, particularly in the eastern provinces.

Indonesia ranks third in terms of the global burden of leprosy, with case fatality rates near 75 percent (WHO 2006a).

Lack of access to clean water and proper sanitation contributes substantially to high levels of morbidity and mortality in Indonesia, especially in children under five and particularly in rural areas (WHO/UNICEF 2008). According to a household survey carried out by the Ministry of Health in 1998, many households that lack access to regular water supply dig their own wells, which are unprotected and thus easily contaminated. Between 1990 and 2006, coverage of improved drinking water sources expanded from 72 percent to 80 percent of the total population, but coverage in rural areas still lags behind urban areas (71 percent of rural residents have access to an improved drinking water source, compared to 89 percent of urban residents). This compares with averages in Southeast Asia of 86 percent for the entire population (92 percent in urban areas and 81 percent in rural areas) (WHO/UNICEF 2008).

People often rely on unhygienic practices for defecation. According to a 2006 estimate, nearly 29 percent of Indonesia's population still practices open defecation (39 percent in rural areas and 18 percent in urban areas). The global average is 18 percent (WHO/UNICEF 2008). Diarrheal diseases, worms and hepatitis are all easily spread through this practice. Although there has been progress towards MDG 7[32] of increasing access to safe drinking water, progress

[32] Target 3 of MDG 7 is to halve, by 2015, the proportion of the population without sustainable access to safe drinking water and basic sanitation (UN 2009b).

towards improvements in basic sanitation has been stagnant for nearly 20 years. Unsurprisingly, water-borne diseases are a major cause of morbidity and mortality in Indonesia, particularly among small children.

A recent study under the auspices of Water and Sanitation Program of the World Bank estimated the burden of disease related to poor sanitation in Cambodia, Indonesia, the Philippines and Vietnam (Napitupulu and Hutton 2008). It attributes an estimated 89.4 million diarrhea cases, over one million helminthes cases and 28 million scabies cases (a skin disorder related to hygiene) to poor sanitation in Indonesia each year. Of the 50,000 deaths annually attributable to poor sanitation in Indonesia, at least 45,000 are estimated to be in the under-five age group. The study authors estimate poor sanitation results in an annual economic loss of US $6.3 billion. Estimated total annual morbidity attributable to poor sanitation and hygiene is 120 million.

As discussed in more detail in Chapter 6, the symptoms reported by children in my IFLS sample are suggestive of the most common respiratory, diarrheal and febrile diseases in Indonesia, and reflect the high burden of disease across the archipelago.

## Chapter 5 The Indonesian Family Life Survey

This chapter provides an overview of the survey from which my data are drawn. I start with a description of the survey waves, follow with a short discussion of the sampling frame and then outline the questionnaires.

### 5.1 Indonesian Family Life Survey

My data are drawn from three waves of the Indonesian Family Life Survey (IFLS), an ongoing, multipurpose longitudinal survey which collects data on individual respondents, their families, their households and the communities in which they live. The IFLS is based on a sample of households representing about 83 percent of the Indonesian population living in 13 of the nation's 27 provinces in 1993. See Figure 5.1 for a map of the IFLS provinces.

**Figure 5.1: Map of IFLS Provinces**


*Source: IFLS documentation, RAND Corporation.*

The IFLS collects socio-demographic information including age, sex, marital status, education and household composition. In addition, data are collected on individual-level information on schooling, multiple indicators of health status including anthropometric measurements, and community-level information on prices and the availability of services. In addition, the IFLS provides detailed information about the communities in which IFLS households are located and their health and school facilities. These data cover aspects of the physical and social environment, infrastructure, employment opportunities, food prices, access to health and educational facilities, and the quality and prices of services available at those facilities.

I use data from the first three full waves of the survey: IFLS1 in 1993/94, IFLS2 in 1997 and IFLS3 in 2000. IFLS1 included a sample of 7,224 households (22,000+ adults) in 13 of 27 provinces.[33] The majority of interviews in each wave of the survey was conducted between the end of the dry season and the first part of the wet season. IFLS1 was fielded between October 1993 and January 1994. IFLS2 was fielded between August 1997 and January 1998. IFLS3 was fielded between July 2000 and December 2000.[34]

[33] A subsample of 25 percent of households was surveyed in 1998 to assess the impacts of the 1998 financial crisis (IFLS2+), data which I do not include in this study. A fourth wave, fielded in 2007/2008, covers the full sample but had not yet been publicly released as work was being finalized for this research.

[34] A small portion of interviews were conducted just before or after these dates (generally in the month immediately preceding or following the majority of interviews), usually as a result of tracking members of origin households that had joined 'split-off' households in non-ILFS enumeration areas.

## 5.2 Sampling Frame

The IFLS is a longitudinal survey so the sampling scheme for the first wave is the primary determinant of the sample in subsequent waves. The IFLS1 sampling scheme stratified on provinces and urban/rural location, then randomly sampled within these strata. Provinces were selected to maximize representation of the population, capture the cultural and socioeconomic diversity of Indonesia, and be cost-effective given the size and terrain of the country. The sample included 13 of Indonesia's 27 provinces, designed to be representative of 83 percent of the population.

Within each of the 13 provinces, 321 enumeration areas (EAs) were randomly chosen from a nationally-representative sample frame used in a socioeconomic survey conducted in 1993 by Indonesia's Central Bureau of Statistics. Urban EAs and EAs in smaller provinces were oversampled to facilitate urban-rural and Javanese—non-Javanese comparisons.[35] Within a selected EA, households were chosen randomly.

Within IFLS1 households, members were randomly selected to provide detailed individual information. In practice, depending on household status, this within-household sampling scheme resulted in high percentages (>95 percent) of household members being interviewed. Figure 5.2 provides an overview of the within-household sampling.

[35] A proportional sample would have been dominated by Javanese who make up approximately 50 percent of population.

**Figure 5.2: IFLS Within-Household Sampling Scheme**

IFLS 1 within-household sampling scheme:

- the household head and his/her spouse
- 2 randomly-selected children of the head and spouse, age 0-14
- an individual age 50 or older and his/her spouse, randomly selected from remaining members
- for a randomly selected 25 percent of the households, an individual age 15-49 and his/her spouse, randomly selected from remaining members

Notes: All adults age 15 yrs+ had a nonzero probability of selection.
Based on selection rules, the number of adults eligible for interview was as high as 6 per household, but was restricted to 4 adults for budgetary reasons. Note that the mean household size in rural areas is 5.8 (with a standard error around the mean of 0.09) and 6.3 (with a standard error around the mean of 0.09) in urban areas. For households with more than 4 adults, a selection rule was applied, which was a binding constraint in fewer than 1 percent of households

*Source: IFLS documentation, RAND Corporation*

In IFLS1, the practice of sampling within the household yielded lower interview rates for certain groups, such as never-married adults and children born to someone other than the household head or spouse. For ILFS2 and subsequent waves, a protocol change dictated interviewing every household member 12 years of age and older in an origin household, and a subset of members in split-off households. Due to attrition and addition of split-off households, the number of households per urban/rural EA diverged from the original sampling frame, and the geographic distribution changed slightly.

### 5.3 Tracking of Panel Respondents and Response Rates

The first wave of the survey (IFLS1) was administered in 1993 to over 24,000 individuals living in 7,224 households. All IFLS1 household members who provided detailed individual-level data in 1993 (*panel respondents)* were

prioritized for tracking if by 1997 they had moved out of the 1993 household. In IFLS2, 94 percent of IFLS1 households and 91 percent of IFLS1 *target* individuals were re-interviewed. High re-interview rates were obtained in part because the survey team was committed to tracking and interviewing individuals who had moved or split off from the origin IFLS1 households. The next wave IFLS3, was fielded in 2000 and included nearly 39,000 individuals in 10,400 households. The re-contact rate was 95.3 percent of IFLS1 households. Nearly 91 percent of IFLS1 households are complete panel households in that they were interviewed in all three waves. Such high re-interview rates lessen the risk of bias due to nonrandom attrition in studies using the data. Table 5.1 outlines the interview targets and sample sizes for each wave.

**Table 5.1: IFLS Panel**

| | | |
|---|---|---|
| IFLS1 - 1993 | 7,224 households<br>24,704 individuals<br>(3,630 children 0-5 years old) | In-home, face-to-face interview with household head, spouse and sample of their children and sample of other adult household members. |
| IFLS2 - 1997 | 7,586 households<br>31,901 individuals<br>(3,722 children 0-5 years old) | Follow-up all households, all 1993 "main" respondents and all 1993 household members born before 1967. |
| IFLS3 - 2000 | 10,400 households<br>39,000 individuals<br>(3,941 children 0-5 years old) | Follow-up all households, all 1993 "main" respondents, all 1993 household members born before 1967, sample of other 1993 household |

*Source: IFLS documentation, RAND Corporation*

Household weights are designed to correct for oversampling of urban EAs and EAs in smaller provinces, as well as differential sampling rates in urban and rural EAs (30 households in urban areas versus 20 households in rural areas). When household weights are applied to the IFLS1 household sample, the resulting weighted distribution should reflect the 1993 distribution of households by urban

and rural status within each of the 13 IFLS provinces. The sample is re-weighted every wave.

### 5.4. Survey Instrument

The Household Survey questionnaire was divided into books, usually addressed to different respondents, and subdivided into topical modules. Three books collected information at the household level, generally from the household head or spouse. Four books collected individual-level data from adult respondents, ever-married female respondents and children younger than 15. Individual physical measures of health status were recorded for each household member in an anthropometry book.

Household composition and information about each member's age, sex, marital status, and education, health status and insurance coverage was collected. Household members 15 years and older were queried about their educational, marital, work and migration histories. Ever-married women age 15-49 were interviewed about retrospective life histories on marriage, children ever born, pregnancy outcomes and health-related behavior during pregnancy and childbirth, infant feeding practice and contraceptive use.

For both adults and children, the IFLS collected extensive measures of health status including self-reported measures of general health status (e.g., excellent, poor, etc.), morbidity including specific symptoms, inpatient and outpatient visits,

use and cost of self-treatment in past 4 weeks (including over-the-counter medicines and traditional herbs and medicines), and vaccinations (children only). Beginning with IFLS2, surveyors also collected the number of days of missed activities in previous 4 weeks due to poor health, and the number of days in bed in previous 4 weeks due to poor health.

Children's health and education status was collected for children younger than 15. For children younger than 11, the child's mother, female guardian, or caretaker answered the questions. Children between the ages of 11 and 14 were allowed to respond for themselves if they felt comfortable doing so.

In IFLS1, a nurse recorded height and weight for each household member. Starting in IFLS2, physical assessments were expanded from anthropometry alone to include blood pressure and pulse (respondents 15 and older), lung capacity (respondents 9 and older), and hemoglobin (respondents 1 and older). In addition, respondents 15 and older were timed while they rose from a sitting to a standing position five times (a physical assessment devised by the WHO team). Starting in IFLS2, the nurse also evaluated the individuals' health status on a 9-point scale (relative to the health status of other people of the same age and sex) and recorded comments about the individual's health. As an indication of household health, the iodine content of the household's salt was also tested starting in IFLS2. Physical characteristics of the house, interviewer-observations

of environmental conditions in and around house, availability of electricity and sources of drinking water were recorded.

The Community-Facility Survey collected community-level information from a community representative, usually the village head or similar representative, including detailed data on environmental conditions such as average rainfall, altitude, availability of electricity, water sources, sanitation, land allocation and land use. Both from the community representative and enumerator observations, the IFLS also collected data on village demographics and welfare conditions including village population, number of female-headed households, number of families living in various welfare categories, whether there were any slums in the village, types of employment villagers are engaged in, predominant sources of income, and community participation.

A Service Availability Roster collected data on health facilities available to community residents in each of the IFLS communities. These include community health centers (puskesmas), private practitioners, community health posts (posyandu), traditional practitioners and hospitals, as well as distances, travel time and travel costs to each of the facilities typically used by community members.

The variables used in this paper are drawn primarily from the children's acute morbidity module, anthropometric measurements and pregnancy history modules

within the Household Survey, along with select village sanitation, facility and environmental variables from the Community-Facility Survey.

### 5.6 Potential Selectivity Biases

When using longitudinal data, one important concern is attrition of respondents over time. If subjects drop out of the study for reasons that are correlated with the outcome variable of interest, coefficient estimates may be affected by selectivity bias.

Briefly, I note here that the IFLS had very high re-contact rates of panel respondents.[36] As noted above, 94 percent of IFLS1 households were re-contacted in IFLS2 and 95.3 percent of IFLS1 households in IFLS3. Nearly 91 percent of IFLS1 households are complete panel households in that they were interviewed in all three waves. Such high re-interview rates lessen the risk of bias due to nonrandom attrition in studies using the data. Nonetheless, the risk of possible bias remains.

To better understand whether attrition is correlated with the outcome variables of this study, I compare the descriptive statistics of the sample of children aged 0-5 in 1993 that did and did not complete the three waves of surveys. The results of this analysis are presented in Table 5.2. The total number of sample respondents aged 0-5 for whom I have nutrition and illness data in 1993 is 2,493. Out of these,

[36] For a detailed discussion of attrition within the IFLS, see Thomas, Frankenberg and Smith (2000).

I am able to follow 2,197 children through the next two waves of the IFLS, resulting in an attrition rate of 11.9 percent.

The comparison of means between the attrited and non-attrited respondents in Table 5.2 shows no significant differences for the most important variables in my specifications, namely stunted status, illness outcome and parents' heights. There are some differences in socio-economic characteristics, namely children whose parent(s) did not respond to all of the questions were slightly younger, had less educated mothers, and came from poorer households as compared to children for whom we have all responses. The attrited children were also more likely to live in districts at higher elevation. However, as discussed more fully in Chapter 7, all results continue to hold using the unbalanced panel.

In the next chapter, I describe in greater detail the sample I use to estimate models explaining acute illness.

## Chapter 6 The Data

In this chapter, I describe the sample I use to estimate my equations of interest. I provide a brief summary of the data requirements. I conclude with details on the main covariates used in my regression analysis.

### 6.1 Summary of Data Requirements

Given my interest in estimating the impact of early childhood malnutrition on the probability of reporting acute illness, and the problems associated with such estimation, data requirements are high. First, I need data on an indicator of young children's long-term nutritional status (height-for-age specifically) during the critical period of development before age five. Second, I need data on contemporaneous acute illness outcomes. To assess whether or not there are any long-term impacts, I also need data on acute illness outcomes sufficiently long enough after nutritional status is measured to determine long-term impacts. Third, I need data on key community characteristics that influence prevalence of infectious disease. Fourth, I need appropriate instruments for children's nutritional status. The remainder of this Chapter describes the data I use to estimate the empirical models.

### 6.2 Description of the Sample

To estimate the contemporaneous impact of malnutrition on acute illness, I use the 1993 cohort of children 0-5 years old for whom age and height

measurements are recorded. As discussed in Chapter 2, I use both parents' heights and the child's birth quarter as instruments. In terms of data, this type of econometric model therefore not only requires information on heights of the children, but also for both their parents. Information on birth quarter is derived from the child's date of birth.

To estimate the long-term impact of malnutrition on acute illness, I start with the sample of children 0-5 years old in 1993 for whom age and height measurements are recorded. I track these children through the next two IFLS waves in 1997 and 2000. This sample of 2,197 children age 0-5 years in 1993 becomes my main sample for estimating the medium- and long-term impact of early childhood malnutrition on acute illness outcomes. I track this group of children through 1997 when they have reached the age of 4-9 years, and again 3 years later in 2000, when they are between 7-12 years old.

### 6.3 Symptoms of Acute Illness

The child acute morbidity module collected information on general health status (e.g., very healthy, somewhat healthy, somewhat unhealthy, unhealthy), the number of days in previous 4 weeks the child missed activities due to poor health, the number of days in previous 4 weeks the child spent in bed due to poor health, and individual symptoms experienced in the previous 4 weeks. For young children, the informant was typically the child's mother though it could also be a knowledgeable female guardian or caretaker.

When working with morbidity data in which individual symptoms are reported, standard practice is to classify symptoms as indicative of one or more types of illness (febrile illness, respiratory or diarrheal illness, for example) , or as individual symptoms indicative of some underlying infectious process (DHS 2007; Das and Sánchez-Páramo 2003; Deolalikar and Laxminarayan 2000). This is the practice I employ here.

With each successive wave, the list of possible symptoms reported was revised slightly. In IFLS1, the set of symptoms was less exhaustive than in IFLS2 and IFLS3. Though it asked about general symptoms of respiratory and diarrheal illness, IFSL1 lacked some detail about the exact nature of respiratory and diarrheal symptoms. Table 6.1 outlines the list of possible symptoms included in each wave, and whether or not each symptom was considered indicative of acute illness for the purposes of this research. I address these discrepancies among waves below.

**Table 6.1: List of Reported Symptoms**

| Symptom | Included in IFLS1 | Included in IFLS2 | Included in IFLS3 | Considered Indicative of Infection (yes/no) |
|---|---|---|---|---|
| Headache | ● | ● | ● | Yes, if combined with fever |
| Runny nose | | ● | ● | Yes, if combined with fever |
| Cough:<br>Dry cough<br>Cough with phlegm<br>Bloody cough | ● | ●<br>●<br>●<br>● | ●<br>●<br>●<br>● | Yes |
| Difficulty breathing:<br>Wheezing<br>Short rapid breath | ● | ●<br>●<br>● | ●<br>●<br>● | Yes |
| Fever | ● | ● | ● | Yes |
| Stomachache | | ● | ● | No (except in IFLS1 when combined with nausea/vomiting) |
| Heartburn | ● | ● | | No |
| Nausea/vomiting | ● | ● | ● | Yes |
| Diarrhea minimum 3 times per day:<br>Mixed with blood<br>Mixed with mucous<br>Pale liquid | ● | ●<br>●<br>●<br>● | ●<br>●<br>●<br>● | Yes |
| Skin infection (boil, abscess, itching) | ● | ● | ● | Yes, if combined with fever |
| Eye infection | ● | ● | ● | No |
| Toothache | ● | ● | ● | No |
| Ear infection | ● | ● | | Yes |
| Rash with red spots | | ● | | Yes, if combined with fever |
| Rash with pus-filled spots | | ● | | Yes, if combined with fever |
| Worms | ● | ● | | Yes |
| Convulsions | | ● | | Yes, if combined with fever |
| Wound/injury | | ● | | No |
| Infections | ● | | | Yes |
| Measles | ● | | | Yes |
| Rheumatism | ● | | | No |
| Other | | ● | ● | No |

IFLS1 recorded certain sets of symptoms as a single symptom, including cough/cold/influenza, nausea/vomiting/heartburn, and respiratory problems/asthma. Diarrhea was recorded as a single symptom, without details as to whether it was bloody, mucousy or watery.

Starting in IFLS2, if a child was reported to have experienced a cough, difficulty breathing or diarrhea, the interviewer prompted the respondent for details. If a cough was reported, the interviewer attempted to ascertain the nature of the cough (whether it was a dry cough, cough with phlegm or bloody cough, which would be indicative of tuberculosis), If difficulty breathing was reported, the interviewer asked whether the child had been wheezing or experiencing short rapid breath. If diarrhea was reported, respondents were asked whether it was bloody (indicative of dysentery), mucousy (indicative of shigellosis) or watery (indicative of cholera).

I construct a simple binary measure of "acute illness" that takes on a value of 1 if an individual reports any of the following symptoms, 0 otherwise:[37]

1. Fever
2. Headache, if combined with fever
3. Skin infection, if combined with fever
4. Convulsions, if combined with fever
5. Diarrhea minimal of 3x per day (whether mixed with blood, mucous or pale liquid)
6. Cough (whether dry cough, cough with phlegm or bloody cough)
7. Difficulty breathing (whether wheezing or short, rapid breath )
8. Rash with red spots (indicative of measles, possibly typhoid fever or typhus)
9. Rash with pus-filled spots (indicative of, among others, Staphylococcus infection or hand, foot & mouth disease)
10. Infections
11. Measles

[37] I arrived at this set of symptoms based on coursework in infectious disease epidemiology, a review of the *Control of Communicable Disease Manual 18th Edition* (Heymann 2004), and discussions with a physician-epidemiologist.

Many respondents reported more than one symptom. I construct a single dichotomous variable if a respondent reports symptoms strongly suggestive of the most common infectious illnesses among young children. I include certain symptoms (like headache) as indicative of acute illness only if they were combined with one or two other symptoms (such as fever). As a conservative measure, I only consider rashes and runny nose indicative of acute illness if combined with fever, since these symptoms could also be associated with allergies. I exclude eye infection and toothache since, although these symptoms may be infectious in nature, it is reasonable to assume they are not related to the major respiratory, diarrheal and febrile diseases I am especially interested in. I exclude heartburn, wound/injury and rheumatism since these are very unlikely to be related to any infectious disease. I exclude 'other' because I lack information in the data as to what this symptom might be.

In Indonesia, the most common infectious diseases associated with fever are malaria, respiratory and intestinal infections, measles and typhoid. Prevalence of acute respiratory infections, primarily pneumonia in children under five, is estimated at 27 percent (DHS 1997). Measles and typhus are common infections with symptoms of rash. Tuberculosis is endemic in much of Indonesia.

Tables 6.2 reports the percentage of children under age five in 1993 reporting individual symptoms in the four weeks preceding the survey in IFLS1, IFLS2 and IFLS3, respectively.

**Table 6.2: Proportion of Three Cohorts of Children 0-5 Years Old Reporting Symptoms of Acute Illness in Three Waves of the IFLS**

| Symptom | Illness in 1993 *1993 cohort of 0-5 year olds* | Illness in 1997 *1997 cohort of 0-5 year olds* | Illness in 2000 *2000 cohort of 0-5 year olds* |
|---|---|---|---|
| Headache | 0.17 | 0.23 | 0.22 |
| Runny nose | | 0.58 | 0.61 |
| Cough | 0.46 | 0.40 | 0.46 |
| Difficulty breathing | 0.03 | 0.04 | 0.05 |
| Fever | 0.36 | 0.43 | 0.45 |
| Stomachache | | 0.13 | 0.14 |
| Nausea/vomiting | 0.07 | 0.09 | 0.11 |
| Diarrhea minimum 3 times per day | 0.13 | 0.16 | 0.15 |
| Skin infection (boil, abscess, itching) | 0.11 | 0.13 | 0.12 |
| Ear infection with draining water | 0.03 | 0.02 | |
| Eye infection | 0.05 | 0.04 | 0.04 |
| Rash with red spots | | 0.08 | |
| Rash with pus-filled spots | | 0.02 | |
| Worms | 0.04 | 0.05 | |
| Convulsions | | 0.006 | |
| Infections | 0.01 | | |
| Measles | 0.02 | | |
| **Sick in previous 4 weeks** | **0.47** | **0.61** | **0.64** |

*Note: Cells are shaded in grey for individual symptoms that were not catalogued in that IFLS wave.*

Notably, symptoms among 0-5 year olds – in particular, the prevalence of cough, fever and diarrhea -- are fairly stable across time periods. Given the ages of this cohort, prevalence of these specific symptoms is in line with expectations.

These results are a common feature of most developing-country populations, and reflect the high prevalence of diarrheal, respiratory and febrile illnesses among young children. The proportion of respondents reporting an illness episode in the four-week reference period is comparable to other countries. Based on various sample surveys in Indonesia, Behrman *et al.* (1987) reports an annual prevalence rate of 2.5 illness episodes per person per year among all age groups. Using the 1997 DHS data from Cambodia, Deolalikar and Laxminirayan (2000)

estimate 1.8 illness episodes per person per year. In the Indian context, Das and Sánchez-Páramo (2003) find illness episodes among adults are reported in one out of every five weeks.

The prevalence rate of diarrhea of 13-16 percent among 0-5 year olds is in line with findings from other studies. A 2004 study in of residents of North Jakarta reported prevalence of diarrhea of 13 percent among children under five, and 4 percent for adults, during the previous four-week period. Elsewhere, estimates of diarrheal prevalence among under fives ranges from 5 percent (Karachi, Pakistan) to 12 percent in the West Bank, Palestine (Abu-Alrub *et al.* 2008). [38]

Table 6.3 illustrates how the prevalence of certain symptoms changes as the panel ages, from 0-5 in 1993 until they reach 4-9 years of age and finally 7-12 years of age in 2000. Overall, the prevalence of illness remains fairly stable, though specific symptoms change. In line with expectations, the prevalence of cough, fever and diarrhea all decrease, though the reporting of headaches increases.

[38] Abu-Alrub et al. (2008) report the following prevalence rates for diarrhea due to one particular bacterium (Cryptosporidium *spp*): 8.8% in Iraq; 16.6%, 11.6%, 27.9% in Egypt for the years 1986, 1987 and 1996 respectively; and 7.3% in India.

**Table 6.3: Proportion of 1993 Cohort of Children 0-5 Years Old Reporting Symptoms of Acute Illness in Three Waves of the IFLS**

| Symptom | 0-5 yo Illness in 1993 | 4-9 yo Illness in 1997 | 7-12 yo Illness in 2000 |
|---|---|---|---|
| Headache | 0.17 | 0.27 | 0.30 |
| Runny nose | | 0.44 | 0.41 |
| Cough | 0.46 | 0.31 | 0.30 |
| Difficulty breathing | 0.03 | 0.04 | 0.03 |
| Fever | 0.36 | 0.32 | 0.26 |
| Nausea/vomiting | 0.07 | 0.06 | 0.05 |
| Diarrhea minimum 3 times per day | 0.13 | 0.08 | 0.06 |
| Skin infection (boil, abscess, itching) | 0.11 | 0.11 | 0.10 |
| Ear infection with draining water | 0.03 | 0.01 | |
| **Sick in previous 4 weeks** | **0.47** | **0.46** | **0.43** |

*Note: Cells are shaded in grey for individual symptoms that were not catalogued in that IFLS wave.*

## 6.4 Description of Main Covariates

*Age*

From the IFLS Household Survey, I construct variables on the age, gender and residence of children. I utilized the data provider's 'best guess' of date of birth to construct age in months for all children, which I need to compare each child's height-for-age to the international child growth standards.

*Height-for-Age as a Proxy Indicator of Nutritional Status*

In IFLS1 and IFLS3, a nurse recorded height in centimeters for each household member.[39] Using data on a child's height and age in months, it is possible to

[39] Anthropometric measurements were also taken in 1997 but those data have not been publicly released.

construct continuous or discrete measures of height-for-age.[40] I discuss each in turn below.

First, I note that the data providers point out that some implausible height/weight/age combinations exist in the IFLS-1 anthropometry data, likely because of transcription errors. Prior to public release, they found cases where height and weight were recorded in the reverse order and cases where weight had been recorded in pounds and not kilograms. Those cases were corrected before public release. In addition, the data providers suspect there were cases where the anthropometrist wrote the height/weight values for person N on the line for person N+1 or N-1. They note that "such an interviewer error would lead to oddities such as 80-kilogram five-year olds," but that such errors are only a few percent of the total number of people with anthropometry measurements (RAND 1995).

*Height-for-Age Z-scores*

I construct height-for-age z-scores for children aged 0-5 in my sample based on the new WHO Child Growth Standards. *Z*-scores are constructed for each individual *i* using the formula:

[40] In either case, in order to determine whether or not a child is stunted for his or her age, I must first convert age in years to age in months and combine this with the child's gender and reported height. I do this by subtracting the month of birth (obtained from the tracking dataset) from the month in which the child was measured, adjusting for each year of age. I use the new WHO international child growth standards (released in 2006) to compare the height-for-age of each child with the relevant cut-offs.

$$Z_i = \frac{Height_i - Height_{WHO}}{SD_{WHO}}$$

where $Height_{WHO}$ represents the mean height for a well-nourished child of the same age and gender, and $SD_{WHO}$ is the corresponding standard deviation.

The *z*-score density plot of children 0-5 in my sample is presented in Figure 6.1 below. Even though the data providers detected and fixed a number of implausible height/weight/age combinations, I still find evidence of measurement error in the recording of height in my sample which translates into some unrealistically large z-scores (in absolute terms). To prevent these outliers from affecting my results, I employ the standard practice of dropping observations with *z*-scores equal to or greater than +/-6 SD from the mean. In Figure 6.2, I restrict the sample to *z*-scores in the range [-6,6]. I lose 200 observations when I do so.

**Figure 6.1 *Z*-score Kernel Density, Unrestricted Sample**

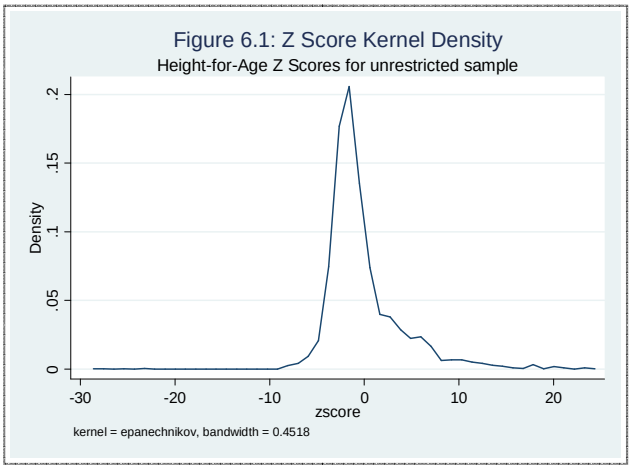
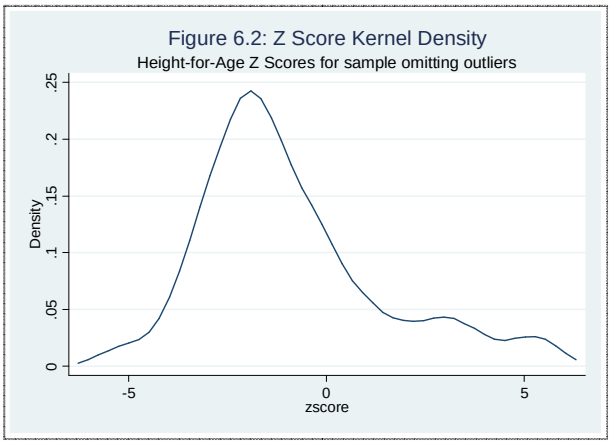


**Figure 6.2 *Z*-score Kernel Density, Sample Omitting Outliers**




Even with this correction, there remains reason to believe that z-scores are quite imperfect measures since there may be substantial measurement error in reported heights in the IFLS data.

*Discrete Measure of Stunting Status*

I create a dummy variable, stunted, which is equal to 1 if an individual child's height-for-age is at least two standard deviations below the WHO mean for that child's age and gender. In line with standard practice using anthropometric data, a child is considered stunted (low height-for-age) if his/her height-for-age is two or more standards deviations below the mean for the reference population. The choice of two standard deviations or more below the mean to define stunting (which will capture both moderate and severe stunting) is the norm in the medical

and economics literature, and allows comparability with findings from other literature.[41]

*Per Capita Expenditure*

I use household per capita expenditure (PCE) as constructed by the data provider. Household expenditure include expenditures on food, non-food (including frequently purchased goods/services and durables), and education and housing expenditures. All values are monthly figures. PCE is expressed in nominal terms for IFLS1, and in real terms for IFLS2 and IFLS3. To obtain the real values, both temporal and spatial deflators were used, using prices in December 2000 in Jakarta as the base. Deflators were only available from 1997 onwards, so for IFLS1, household expenditure is in nominal values only. When expenditure values were missing in the original data sets due to "Don't Know" or "Missing", they were replaced by the data provider, when possible, by imputed values. The values used for imputation are the median values of the variable taken at either the community, sub-district or district level, whichever was available with first preference given to the lowest administrative level available.

Household per capita expenditure was constructed by dividing household expenditure by the number of household members.

[41] I lose precision in measuring the slope by relying on a discrete rather than continuous measure. As a robustness check, in addition to using the discrete stunted variable, I run my regression analysis using the continuous *z*-score variable. I discuss these results in Chapter 7.

*Maternal Education*

Though there are conflicting findings regarding the exact mechanism, there is evidence suggesting that parental education, particularly a mother's education, matters to child health outcomes (Strauss and Thomas 1998; Ruel *et al. 1992;* Glewwe 1999*;* Rosenzweig and Schultz 1983). I link children to their birth mothers to obtain mother's education. I assume the education of the child's mother is the most important relevant indicator of formal education for the child. I categorize mother's education as either no education, at least some primary education or at least some secondary education.

*Mother's Height / Father's Height*

I link children to their birth mothers and fathers to obtain the height of the child's mother and father. I use parents' heights as one set of instruments. Parental height is a natural instrument for child's height because genetics explain 60-80 percent of the variation in a child's height, with the remaining 20-40 percent determined by net nutrition (food intake minus demands from disease) (Lai 2006).

The same caveat regarding height measurements applies to mother's height and father's height in that some implausible height/weight/age combinations were detected prior to public release of the data. To address this issue, and prevent extreme outliers from affecting my results, I replace a parent's height for those

reporting heights below the lowest 1 percent and above the highest 1 percent with the value for the 1st percentile and 99th percentile, respectively.

*Household Size*

Household size may affect both a child's nutrition and acute illness outcomes through several pathways. A larger number of household members increases competition for food, increases disease transmission, may decrease mother's available time and care, and may increase availability of substitute caregivers for young children. The overall effect of household size on a child's health outcomes is likely complex and, therefore, it is difficult to determine *a priori* what the sign of the coefficient might be. Nonetheless, I expect household size to be a potentially important determinant of illness.

*Disease Prevalence*

I wish to determine whether disease prevalence impacts the likelihood of any one individual falling ill, controlling for certain individual, household and village characteristics (age, education of self or mother, income, hygiene and sanitation conditions, rural/urban residence, province, etc.).

The IFLS recorded average village altitude, rainfall and whether the village is located on a river. Unfortunately, my sample of children ages 0 to 5 in1993 lacks sufficient observations to use these as controls without substantial loss of data. I have less than 1400 observations, which would result in the loss of nearly 40

percent of my sample. Instead, I control for seasonality of disease prevalence by including month of interview dummies, and further control for location differences in disease prevalence by including district fixed effects.

*District Characteristics*

In addition to the environmental characteristics such as rainfall and altitude that may influence disease prevalence, IFLS2 and ILFS3 included data on the availability of health care facilities (including private health care providers and hospitals) and elementary schools per 1000 residents in each district. These data were not collected in IFLS1 and so are not available for the 1993 cohort. As with the environmental characteristics, my sample of children ages 0 to 5 in1993 lacks sufficient observations to use these as controls without substantial loss of data. Estimation of district fixed effects will absorb all the differences in district-level factors, including availability of health care and general level of development.[42]

[42] Village fixed effects would have been preferred to district fixed effects. However, there was insufficient varation in stunting status among children 0-5 years old sampled within IFLS villages.

## Chapter 7 Results

This chapter presents results from my analysis of the impact of early childhood malnutrition, as measured by stunting (low height-for-age) on acute illness. I first contrast children 0-5 years old in 1993 (IFLS1) by comparing means and checking for significant differences between those who were and were not stunted in 1993. Next, I present the results of empirical specifications designed to determine whether stunting can explain acute illness outcomes. I discuss the main instrumental variable specification for contemporaneous illness and my findings. Next, I present findings from specifications that measure the long-term impact of early childhood stunting. I briefly discuss how nutritional status changes over time for my sample of children, and whether these changes are likely driving any of the long-term results on acute illness.

### 7.1 Summary Statistics

I start by comparing summary statistics on stunting for the cohort of children ages 0-5 in 1993. The summary statistics are presented in Table 7.1. Roughly half of the sample is female (49 percent) and slightly more than half live in rural areas (59 percent). The mean age is 2.65 years and birth weight is 3.16 kg.[43] Mean mother's height is 150.06 cm and mean father's height is 160.92 cm.

[43] 1 kg = approximately 2.2 lbs. A birthweight of 3.16 kg is equivalent to 6.97 lbs.

In terms of socio-economic characteristics, the average monthly household per capita expenditure, reported in nominal terms, is 51,032 Rupiah[44] (standard deviation of 37,169). Just under one-fifth of mothers have no education in 1993, compared with 32 percent reporting at least secondary education. I control for mothers' formal education in all my regression specifications.

The variables at the heart of this dissertation are, of course, acute illness and stunting. As shown in Table 6.2 and Table 7.1, respectively, about 47 percent of the sample reports an acute illness in 1993, and the stunting prevalence among children in my sample is 35 percent. Although medical science and anthropological studies have largely discredited the 'short but healthy' hypothesis (Victora *et al.* 2008), it is nevertheless interesting to note simple correlations between malnutrition indicators in my data. The correlation between stunting (low height-for-age) and underweight (low weight-for-age) is high (0.48) and statistically significant at the 1 percent level. Thus, children who are short for their age tend to be underweight for their age as well. Since weight is partially a function of height, this by itself cannot discount the possibility that stunted children are simply short but healthy. However, the correlation between stunting (low height-for-age) and wasted (low weight-for-height) is also positive. The correlation is weak (the coefficient is 0.05) but statistically significant at the 1 percent level.

[44] Equivalent to approximately US $24.30 (standard deviation of US $17.70) at the prevailing exchange rate in 1993.

*Comparison of Means*

While the above are raw means as reported in the IFLS1 sample, it is important to compare the means across stunted and non-stunted children within the 1993 wave. Table 7.2 presents this mean comparison and reports t-tests to verify whether the differences in sub-sample means are statistically significant.

As shown in the table, children who are stunted are significantly more likely than non-stunted children to have been born at a lower birth weight,[45] have shorter parents (confirming that parents' height matters), live in poorer households (as measured by per capita expenditure), slightly larger households (0.3 members more), and have mothers with lower levels of education. At a more aggregate level, stunted children are more likely to be living in rural areas, and in communities that receive lower average rainfall and are located at higher altitudes. Given that villages located at higher altitudes in Indonesia are likely in more remote areas, it is plausible that households have less access to heath, sanitation and other facilities in such areas.[46] In the regression framework that follows in the next subsection, I fully control for all observable and unobservable geographic characteristics by including a full set of location dummies.

[45] The difference is only 60 grams (approximately 2 ounces), which represents less than 2 percent of the mean birthweight for children in my sample.

[46] Babies born at higher altitudes have systematically lower birthweights (Jensen and Moore 1997; Hartinger *et al.* 2006; Yip 1987). In a study of Colorado infants which controlled for other risk factors for low birthweight (including maternal age, gestation, smoking, prenatal care), Jensen and Moore (1997) found that birthweight declined by 102 g for every 1000 m increase in elevation. Whether differences in birthweights due to altitude result in differential health risk is unclear (Unger et al. 1988)

## 7.2 Does Early Childhood Stunting Explain Likelihood of Acute Illness?

In this subsection, I present analysis for the contemporaneous effects of stunting on acute illness. I wish to determine whether stunting can explain acute illness outcomes. Running an OLS regression on contemporaneous data, however, is problematic since the main regressor (stunted) may be endogenous. That is, while stunting in 1993 may in fact explain acute illness in 1993, the causality could easily be attributed in the opposite direction (i.e., acute illness leads to stunting). This may be particularly true during critical periods of growth, such as before age 2, when growth velocity is especially high (Martorell 1999; Ruel *et al.* 2008). It is possible that severe or prolonged 'acute illness' (e.g., severe diarrheal episode) could affect gains in height (Black *et al.* 2008). An additional concern is that there are likely unobserved variables that affect illness outcomes and their omission would lead to biased regression estimates. For instance, parental hygiene practice may affect illness outcomes, yet such measures remain unquantified. As explained in detail in Chapter 3, I employ an instrumental variables strategy to account for these empirical challenges.

*First Stage Instruments – Parents' Height and Birth Quarter*

Parents' heights are highly correlated with child's height for obvious genetic reasons, but are presumably not directly associated with whether or not the child experiences acute illness. I include both mother's height and father's height as instruments for a child's stunting in 1993.

As an additional identifying instrument, I use birth quarter dummies. As detailed in Chapter 2, previous research has found an association between birth quarter and health outcomes which has been attributed to rainfall and weather patterns, and therefore abundance of food (see for example Lokshin and Radyakin (2009); Moore *et al.* (2004)). As literature in several disciplines has argued (Loskhin and Radyakin 2009; Moore *et al.* 1997; Moore *et al.* 1999), birth quarter effects on health outcomes may be a result of both higher prevalence of infectious disease and lower food availability during lean seasons. In the first stage, I also control for gender, age, village location (urban/rural), mother's education and district dummies.

Table 7.3 presents the first stage results for my IV estimation where I regress stunting status on both parents' heights, birth quarters and other controls. The coefficients on both parents' heights are very strongly significant at the 1 percent level. The negative sign on the coefficient is as we would expect -- children of taller parents are less likely to exhibit low height-for-age. An increase of 1 cm in mother's height leads to a 1.2 percent lower likelihood of a child being stunted, while the same increase in father's height leads to a 0.8 percent lower likelihood of a child being stunted.

Birth quarter dummies enter very significantly individually and jointly. Note that birth quarter 4 is the omitted category. The joint significance of the first stage variables is very strong, with an F-statistic varying between 34.40 and 39.96,

depending on the specification. Importantly, adding birth quarters does not diminish the magnitude or significance of the coefficients on parents' heights. In terms of coefficients, birth quarter dummies show very strong correlation with the probability of being stunted (statistical significance at the 1 percent level). In sum, my first stage regression is well behaved and the instruments are strongly associated with stunting.

Compared to children born in the last quarter of the year, children born in the first three quarters are substantially more likely to be stunted. Children born earlier in the year are at particularly higher risk of stunting relative to children born later in the year. The Indonesian rice harvest cycle is the likely explanation for this finding. Rice is the main staple crop throughout the country, and is harvested in three seasons with some regional variation. Just over half of total rice production is harvested between January and April (51 percent), with relatively smaller harvests between May-August (31 percent) and September-December (18 percent of production) (WFP 2006). Children born in the fourth quarter likely have better nutritional outcomes for three reasons. First, conception and pregnancy coincide with periods of relative food abundance. Second, children born in lowest harvest season may be more likely to breastfed since mothers will be required to contribute less work for a smaller harvest. Finally, the early months of life for children born in the fourth quarter also correspond with periods of relative food abundance.

Turning to another control that is potentially associated with both stunting and acute illness outcomes, household size does not appear to have an important effect on the likelihood of stunting.  the illness outcomes.  The coefficient on household size is roughly -0.02 in the contemporaneous context, and -0.01 when explaining illness in both 1997 and 2000.

The table also presents results from specifications that include district fixed effects. District fixed effects account for differences across districts in basic environmental and sanitation conditions. Controlling for these differences is important as disease production is likely affected by these variables. The F-statistic with district fixed effects remains very high.[47]

*Second Stage Results*

The second stage of the instrumental variables approach regresses acute illness outcomes in 1993 on the predicted value from the first stage regressions, along with other exogenous controls. These results are reported in Table 7.4.

I present two different second-stage specifications, one with only parents' heights as instruments, and the second where I include birth quarters as additional instruments.  Both these specifications are presented with and without district

[47] Note that using parametric values of district-level disease prevalence (proxied by district-average rainfall and altitude, and proximity to a river) instead of district fixed effects does not affect the first or second stage results.

fixed effects. Since I have multiple instruments in all specifications, I report Hansen J statistics and Chi-square p-values from overidentification tests.[48] When using both parents' heights and birth quarters as instruments, the second stage results show that the coefficient on stunted growth is positive and significant at the 5 percent level, and equal to 0.16. That is, children who are stunted are 16 percent more likely to report acute illness as compared to non-stunted children. The results with only parents' heights as instruments, also significant at the 5 percent level, indicate that stunting increases the likelihood of acute illness by 23 percent. These results show that being stunted has a strong effect on the likelihood of reporting contemporaneous acute illness.

Looking at the coefficients on the additional controls, I find that children from poorer households, as measured by household per capita expenditure, are less likely to report acute illness episodes. At first blush, this seems to contradict *a priori* expectations. However, the fact that household per capita expenditure has a negative effect on the likelihood of stunting suggests some measure of social conditioning[49] may be behind these results. Recall that height is an objective measure, whereas acute illness is a subjective measure since it is self-reported (or reported by the child's mother as here). If more well-to-do households are

[48] An overidentification test examines the validity of the particular combination of instruments used by contrasting that combination with all other possible convex combinations of instruments. When there are more instruments than endogenous regressors, a chi-square test can be used to test the overidentifying restrictions. The test statistic is called the Hansen J Statistic.

[49] Nobel prize winner Amartya Sen developed the concept of adaptation and cultural or social "conditioning" to explain just this type of surprising phenomenon when comparing self-reported measures in developing versus developed countries, and for different socioeconomics groups within developing countries (Sen 1985).

more sensitive to acute illness, perhaps because they have a lower overall burden of disease, such households may be more likely to report illness episodes despite experiencing the same or even fewer illness episodes relative to poorer households. In a study of illness reporting in Nepal, Pokhrel (2007) finds that income seems to have a modest but significant effect on illness reporting, which supports medical anthropologists' observations that people belonging to different economic groups understand illness differently. Helman (2001) suggests that non-poor households are more likely to report their child as ill than the poor households because their income makes 'illness management' more affordable to them.

Household size does not appear to have an important effect on the likelihood of illness outcomes. Interestingly, in the contemporaneous context, the coefficient is negative in sign and statistically significant at the 1 percent level; the magnitude suggests that for each additional household member, the likelihood of reporting acute illness decreases 2 percent. Given the multiple pathways through which household size may affect health outcomes, it is difficult to interpret these findings. When explaining future illness, the effect of household size continues to be negative, is similar in magnitude (-0.02, and -0.01 in 1997 and 2000, respectively), though is no longer statistically significant.

### 7.3 Does Early Childhood Stunting Influence Future Illness Outcomes?

Results indicate that early childhood stunting substantially increases the likelihood of developing acute illness contemporaneously. An important empirical question is whether early childhood malnutrition remains an important determinant of future illness outcomes and, if so, for how long. As discussed in Chapter 2, the medical literature finds long-lasting effects of malnutrition on immune function. However, the literature speaks only generically to the possible duration of the effect of malnutrition on susceptibility to disease.

I exploit the panel nature of the IFLS to address these questions. I follow the children who were 0-5 years old in 1993 and observe illness outcomes for them in 1997, and then again in 2000. Hence, I am able to study whether illness outcomes are affected by early childhood stunting four and seven years into the future, when this cohort of children are between 4-12 years old.

The main regressions I present in Tables 7.5 and 7.6 explain the likelihood of acute illness in 1997 and 2000, respectively, based on stunting status in 1993. Since future illness cannot cause past stunting, I report results from both OLS and probit models. The stability of the coefficients when moving from OLS to a probit model suggests that imposing linearity despite the dichotomous nature of the dependent variable is not unreasonable. The results from both models look very similar. Although endogeneity concerns are allayed by using a lagged main regressor, there may be unobserved factors that affect both the likelihood of

current illness and past stunting status. To account for such omitted variables, I once again instrument for nutritional status using parents' heights and birth quarter dummies.[50]

As I argued previously, IV is also valuable when the key regressor is mis-measured. Because a mis-measured regressor means that its OLS coefficient will be downward-biased, when I use IV estimation I would expect the coefficient on this regression to be larger than its OLS counterpart (Angrist and Krueger 2001; Stock *et al.* 2002). Further, measurement error will likely lead to inflated standard errors of the IV coefficients because the instruments are only valid for the correctly measured portion of the sample.

*IFLS2 Results*

Table 7.5 presents results for 1997. Column (1) estimates the determinants of acute illness using a linear probability model whereas column (2) displays the marginal effects from a probit model. Columns (3) and (4) repeat this exercise with month of interview dummies as additional controls. The IFLS interviews were conducted over 4-6 months, and at slightly different times of the year each wave. Given that disease prevalence will vary over time depending on weather, rainfall and other environmental conditions, controlling for seasonality will capture

[50] As a test of endogeneity, I use OLS and IV estimations to conduct a Hausman specification test. While the p-values obtained from the Hausman test in the contemporaneous context were just barely high enough to prevent me from being able to reject the null hypothesis of exogeneity (p-values were 0.13 in 1993), here the p-values are very high, ranging from 0.3 (2000 illness) to 0.9 (1997 illness).

any seasonal spikes in the incidence of acute illness outcomes in my sample. The results from the two models are almost identical. Importantly the coefficient on stunted growth is positive and significant, though the magnitude is smaller than in the contemporaneous case. Specifically, children who were stunted in 1993 are 5 percent more likely to report acute illnesses in 1997 as compared to children who were not stunted in 1993.

Column (5) adds district fixed effects. The results are robust to these additional controls. Finally, column (6) presents results from the second stage of the 2SLS procedure using both parents' heights and birth quarters as instruments. The magnitude and sign of the coefficient remains comparable to their counterparts in the previous specifications, but, as expected, with IV, the coefficient on being stunted in 1993 is estimated less efficiently and is no longer statistically significant at the conventional levels.

Overall, the sign and magnitude of the coefficient on stunting suggests that the effect of stunted growth in 1993 is substantially less in 1997 than it was in 1993, dropping from 16 percent to 5 percent, and in the case of IV is no longer significant. These results suggest that the effects of stunting do persist four years into the future, but are substantially less pronounced than in the contemporaneous case.

*IFLS3 Results*

Next, I explore whether the relationship between acute illness and stunted growth holds seven years into the future. By using IFLS3 data from 2000, I run the same regression specification as above on the same sample of children who were 0-5 in 1993, replacing 1997 variables with 2000 variables. Results are presented in Table 7.6.

Surprisingly, unlike the 1997 results, being stunted in 1993 no longer explains the likelihood of acute illness in 2000. The coefficients on stunted in 1993 in all specifications are very close to zero, and none are statistically significant. The IV coefficient is also not significant, though the coefficient is a bit larger. As I will explore in detail below, the magnitude of the coefficient on stunting in the IV specification may be affected by the change in stunting status. Many children who were stunted in 1993 are in fact no longer stunted by 2000. Conversely, some who were not stunted in 1993 are stunted by 2000. Since parents' heights and birth quarters only instrument for 1993 stunting status, the actual sample over which the instrument is valid is further reduced. These changes in stunting status may explain why the IV coefficient on stunting in 1993 is higher in 2000 than in 1997 since the instrument is only capturing the effect on children who were stunted in 1993 and remain stunted in 2000. Unfortunately, the lack of anthropometric measurements from the IFLS2 wave in 1997 precludes me from conducting the same analysis for that wave.

*Changes in Stunting Status*

Table 7.7 presents frequencies of changes in nutritional status between 1993 and 2000 and indicates that, indeed, there is a great deal of movement in both directions, both declines and improvements in nutritional status, as measured by low height-for-age. While 72 percent of children experience no change (they are either never stunted or remain stunted throughout the seven-year period), nearly 28 percent of children experienced either a decline or an improvement in nutritional status, as measured by low height-for-age.

In Table 7.8, I investigate the determinants of changes in stunting status between 1993 and 2000 for this cohort of children. The first four columns of this table restrict the sample to children who were stunted in 1993. The left-hand side variable, “Improvement in Stunting Status”, is a dummy that equals 1 if a child’s nutritional status improves by 2000 and s/he is no longer stunted. This variable is then regressed on socio-economic and demographic variables available in the dataset.

I find that improvements in nutritional status are positively associated with household wealth, as measured by per-capita expenditure, though the coefficient loses significance after I add more controls. Further, younger children are more likely to improve their nutritional status, as depicted by the negative and strongly significant coefficient on age. Also appearing very strongly are genetic factors: mother’s height and father’s height are both strongly and positively associated

with improvements in nutritional status, as measured by height-for-age. The quarter dummies are also significant, and suggest that children born in the fourth quarter were significantly more likely to experience improvements than children born in the first three quarters of the year.

Columns (5) – (8) study the determinants of deterioration in stunting status. That is, these regressions investigate the likely reasons why children who are not stunted in 1993 later falter in growth by 2000. Interestingly, boys are more likely to experience a deterioration in nutritional status, as are younger children. Children belonging to poorer households and with mothers with less education are also more likely to experience a deterioration. Finally, genetic factors also appear strongly with both parents' heights strongly and negatively correlated with deterioration in stunting status. The correlation between parents' heights and whether a child experiences an improvement or deterioration in stunting status seven years hence may in fact reflect genetic and ethnic differences that only become apparent during the pre-pubertal growth spurt (WHO 2006b).

While the specifications presented in Table 7.8 likely have omitted variables, the reported correlations are nonetheless interesting to investigate and importantly support findings from the medical literature. While conventional wisdom holds there is little catch up growth (Martorell *et al.* 1994), other nutrition studies challenge this view. Adair (1999) uses a longitudinal dataset from the Philippines to examine the possibility of catch-up growth and finds large potential for catch-

up growth in children into the pre-adolescent years. In particular, children with taller mothers, who were first born, longer at birth, less severely stunted in early infancy and those with fewer siblings were more likely to experience catch-up growth between 2 years and 12 years of age.[51] My results are consistent with the latter study.

### 7.4 Robustness Checks

*Potential Endogeneity of Parent's Heights*

I have argued the use of parents' heights as instruments on the basis that parents' heights account for the genetic component of a child's height. I use a child's birth quarter, which affects a child's nutritional status via availability of food during critical periods of prenatal and early postnatal growth (Lokshin and Radakan 2009; Moore *et al.* 1997; Moore *et al.* 1999), to capture any shocks to a child's early nutritional outcomes. The combination of these instruments allows me to capture the deviation in genetic potential in height for that individual due to nutritional shocks.[52]

[51] Cameron *et al.* (2005) provides provocative evidence that whether or not evidence for catch-up growth can be detected is highly sensitive to the choice of height measurement. Specifically, the authors argue that height *z* scores rather than height should be used. Their theoretical exercise supports the potential for catch-up growth.

[52] Given the non-experimental nature of my data, and the lack of an exogenous shock for the time period of my data, estimating a model with household fixed effects, thereby fixing the mother, would be the ideal way to test the independent effect of stunting on illness outcomes. Unfortunately, there is insufficient variation within households in my sample. Nearly all of my households are comprised of children who are all healthy, or children who are all stunted. Instead, I do the next best thing, which is to include district fixed effects, which soak up all observable and unobservable differences in sociodemographics, infrastructure, sanitation, environment and climate, across locales.

Recall that parents' heights are a very strong predictor of a child's stunting status, as evidenced by a F-statistic from the test of joint significance of 34.4 in the first stage of my IV results (in which district fixed effects are included). Recall also that when using both parents' heights and birth quarters as instruments for a child's stunting status, I find that children who are stunted are 16 percent more likely to report contemporaneous acute illness compared to children who are not stunted.

When explaining a child's acute illness outcomes, one possible concern with the use of parent's heights as an instrument for a child's stunting status is that there may be unobserved variables that influence both a parent's height and the probability of a child's acute illness. This might be the case because a parent's height is a function of genetics, parent's nutrition during childhood, along with any insults (e.g., disease) that influenced parent's attained height. In other words, a parent's attained height reflects the parent's human capital more generally, rather than solely the parent's genetics. If a parent was malnourished as a child, that malnourishment may have been a result of investment choices the grandparents made in the parent's nutrition and hygiene which affected nutrition and disease outcomes of the parents, which then create an intergenerational transmission of malnutrition. Parents may also have suffered insults due to famine, war or other shocks that affected their attained height.

Evidence from a number of studies suggests that smaller mothers are more likely to have smaller babies (Victora *et al.* 2008; Grantham-McGregor *et al.* 1999a;

Martorell 1999; Martorell and Habicht 1986), which may be explained by genetics, a parent's own malnutrition as an infant, or some combination of both. The relationship between a mother's birth weight and birth length, in particular, has been found to be a strong predictor of a child's birth size, more so than the relationship between father's birth size and offspring (Victora *et al.* 2008).

A mother's height, in particular, may be correlated with illness through the mother's (household's) poverty status and human capital generally. A simple correlation between mother's height and household per capita expenditure does reveal a positive relationship, with a correlation coefficient of 0.14. (The same correlation coefficient for father's height is 0.10.) Though the effect is weak, this positive relationship is statistically significant and in line with findings from the literature on the relationship between attained height and adult earnings and marriage market outcomes (Hoddinott *et al.* 2008; Thomas and Strauss 1997; Behrman and Deolalikar 1989; Maluccio *et al.* 2006).

Although it is true that, on average, on a population basis, shorter parents have been found to have lower earnings and poorer education attainment, both of which might affect a child's illness outcomes, I control for household income, maternal education and whether the household lives in a rural or urban area, outcomes which are all correlated with poverty. I also control for observable and unobservable locational differences by including district fixed effects.

In addition, I report the results of overidentification tests. I do not reject the null hypothesis that the instruments are valid (uncorrelated with the error term) when using either parents' heights alone, or parents' heights and birth quarters as my set of instruments.

For the sake of completeness, I also estimate a model of acute illness in which I include parents' heights as additional controls[53] and instrument stunting with birth quarters only. Results for each of the three waves are presented in Table 7.4 (Column 5), Table 7.5 (Column 7) and Table 7.6 (Column 7).

As results in all three time periods show, parents' heights have *no* independent effect on the acute illness outcomes after controlling for the demographic and socioeconomic variables discussed above. Further, even with parents' heights as controls and only birth quarter as instruments, I find that children who are stunted are still 11 percent more likely to report contemporaneous acute illness compared to children who are not stunted. While this result diminishes in the longer-term regressions, the fact that parents' heights as controls show no independent effect on the probability of acute illness and the results of my tests of overidentifying restrictions lend support to my decision to use them as instruments rather than controls.

[53] Glewwe (1999) uses the heights of both parents as explanatory variables to partially control for the researcher's inability to directly observe a child's health endowment. However, he does not describe any explicit rationale, though he hints that parents' heights proxy for unobservable genetic factors.

*Choice of Dependent Variable*

In this dissertation, I have deployed a binary indicator of stunting status based on a continuous measure, namely the height-for-age z score. In line with standard practice in the literature, I have classified children as stunted if they are 2 or more standard deviations below the age-and gender-specific mean height. Here, I check whether the results would be have been similar, had I used directly the height-for-age z score.

As shown in Table 7.9, in the first stage, all instruments should and do have the opposite signs with respect to their counterparts in the regressions where the dependent variable is the stunted dummy. Thus, we expect taller parents to have taller children (i.e., larger z-scores). The relative disadvantage of being born earlier in the year remains apparent by both the sign and relative magnitudes of the birth quarter dummies. The most important instruments (parents' height and birth quarters) remain strong predictors of a child's z score.

As shown in Table 7.10, the second stage results when using the continuous *z*-score variable are similar to the results when using the discrete variable to capture stunting status. There is an association between the continuous z score (instrumented) and contemporaneous illness in 1993. The association between low height-for-age and acute illness is not as strong, however, as one would infer from using the binary indicator of stunting status. The magnitude of the coefficient on the continuous height-for-age z score in 1993, -0.042, implies that

for a child with a borderline classification of stunting (z-score of -2.0), the likelihood of acute illness increases by 8.4 percent. With a z-score of -2.5, i.e., between moderately and severely stunted), the likelihood of acute illness increases by 10.5 percent. Finally, a severely stunted child (z-score of -3), the likelihood of acute illness increases by 12.6 percent over children who are not stunted. For comparison, using the same set of instruments the coefficient on stunted when using the stunted dummy is 0.16. This effect dissipates by 1997, as well as by 2000 (as was the case with the discrete variable).

In summary, the results from using the continuous z-score indicate a somewhat smaller impact of stunting on acute illness than when I use the binary indicator of stunting status. The magnitude of the effects are, however, well within the range of those seen with the binary indicator of stunting, and generally confirm the association between stunting and acute illness.

*Potential Selectivity Bias*

In Chapter 5, I briefly noted that my results are essentially unchanged when using the unbalanced panel. Here, I discuss these findings in more detail. As Tables A1 in Appendix A shows, the first stage results are nearly identical. The coefficient on mother's height is only slightly lower using the unbalanced panel (-0.011) than the balanced panel (-0.012). The magnitude of the coefficient on father's height in the first stage is also slightly lower (-.006 in balanced panel vs -.008 in unbalanced). As Tables A2 in Appendix A indicates, in the second stage,

the magnitude of the coefficient on stunting when using only birth quarters as instruments is slightly lower than in the balanced panel (0.082 vs 0.09), while the magnitude is slightly higher when including parents heights as either the sole set of instruments (0.233 vs 0.288) or when both parents' heights and birth quarters are used (0.157 vs 0.163). Despite slight differences in the magnitudes of some of the coefficients, the level of statistical significance remains unchanged.

# Tables and Figures

**TABLE 5.2: ATTRITION IN THE PANEL**
**COMPARISON OF MEANS FOR ATTRITED AND NON-ATTRITED SAMPLES**

| | | Non-Attrited Sample | Attrited Sample | Difference | |
|---|---|---|---|---|---|
| **Stunted in 1993** | | | | | |
| | (Mean) | 0.35 | 0.30 | 0.05 | |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Any Illness in 1993** | | | | | |
| | (Mean) | 0.49 | 0.48 | 0.01 | |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Female** | | | | | |
| | (Mean) | 0.49 | 0.51 | -0.02 | |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Age in 1993** | | | | | |
| | (Mean) | 2.65 | 2.44 | 0.21 | ** |
| | (Std. Err.) | 0.04 | 0.10 | 0.11 | |
| | (Number of obs) | 2197 | 296 | | |
| **Birthweight** | | | | | |
| | (Mean) | 3.16 | 3.15 | 0.02 | |
| | (Std. Err.) | 0.02 | 0.05 | 0.05 | |
| | (Number of obs) | 1083 | 143 | | |
| **Mother's Height (cm)** | | | | | |
| | (Mean) | 150.06 | 150.56 | -0.50 | |
| | (Std. Err.) | 0.11 | 0.29 | 0.31 | |
| | (Number of obs) | 2197 | 296 | | |
| **Father's Height (cm)** | | | | | |
| | (Mean) | 160.92 | 161.13 | -0.21 | |
| | (Std. Err.) | 0.12 | 0.38 | 0.40 | |
| | (Number of obs) | 2197 | 296 | | |
| **Per Capita Expenditure in 1993 (nominal)** | | | | | |
| | (Mean) | 0.51 | 0.56 | -0.05 | * |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Living in Rural Area in 1993** | | | | | |
| | (Mean) | 0.59 | 0.48 | 0.10 | ** |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Mother Has No Education** | | | | | |
| | (Mean) | 0.19 | 0.16 | 0.04 | |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 2197 | 296 | | |
| **Mother Has Primary Education** | | | | | |
| | (Mean) | 0.65 | 0.57 | 0.08 | ** |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **Mother Has Secondary Education** | | | | | |
| | (Mean) | 0.32 | 0.32 | 0.00 | |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2197 | 296 | | |
| **District Average Altitude, m above sea level** | | | | | |
| | (Mean) | 1.59 | 1.82 | -0.23 | ** |
| | (Std. Err.) | 0.03 | 0.07 | 0.07 | |
| | (Number of obs) | 1410 | 193 | | |
| **District Average Rainfall in 1991/92** | | | | | |
| | (Mean) | 0.33 | 0.29 | 0.03 | |
| | (Std. Err.) | 0.01 | 0.03 | 0.03 | |
| | (Number of obs) | 2143 | 283 | | |

* significant at 10 percent
** significant at 5 percent

**TABLE 7.1: SUMMARY STATISTICS FOR CHILDREN 0-5 YEARS OLD IN 1993**

| | Obs | Mean | Std. Dev. | Min | Max |
|---|---|---|---|---|---|
| Stunted in 1993 (dummy = 1 if HFA <- - 2 SD) | 2197 | 0.35 | 0.48 | 0 | 1 |
| Female | 2197 | 0.49 | 0.50 | 0 | 1 |
| Age in 1993 (years) | 2197 | 2.65 | 1.64 | 0 | 5 |
| Birthweight (kg) | 1083 | 3.16 | 0.51 | 2.1 | 4.1 |
| Mother's Height (cm) | 2197 | 150.06 | 5.19 | 137.80 | 162.80 |
| Father's Height (cm) | 2197 | 160.92 | 5.63 | 144.30 | 174.10 |
| HH Per Capita Expenditure in 1993 (nominal) | 2197 | 0.51 | 0.37 | 0.14 | 1.75 |
| Living in Rural Area in 1993 | 2197 | 0.59 | 0.49 | 0 | 1 |
| Mother Has No Education | 2197 | 0.19 | 0.39 | 0 | 1 |
| Mother Has Primary Education | 2197 | 0.65 | 0.48 | 0 | 1 |
| Mother Has Secondary Education | 2197 | 0.32 | 0.47 | 0 | 1 |
| Household Size | 2197 | 5.39 | 1.81 | 3 | 14 |
| District Average Rainfall in 1991/92 (m/yr) | 1410 | 1.59 | 0.95 | 0.00 | 4.08 |
| District Average Altitude* | 2143 | | | 0 | 2 |
| *% <500 meters* | | *0.74* | | | |
| *% 500-700 meters* | | *0.19* | | | |
| *% >700 meters* | | *0.07* | | | |

* IFLS 1 classifies village altitude above sea level as:

0. less than 500 meters

1. 500 - 700 meters

2. more than 700 meters

**TABLE 7.2: COMPARISON OF MEANS FOR CHILDREN 0-5 YEARS OLD IN 1993**

| | | Not Stunted in 1993 | Stunted in 1993 | Difference | |
|---|---|---|---|---|---|
| **Female** | | | | | |
| | (Mean) | 0.49 | 0.48 | 0.02 | |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Age in 1993** | | | | | |
| | (Mean) | 2.50 | 2.92 | -0.42 | ** |
| | (Std. Err.) | 0.04 | 0.06 | 0.07 | |
| | (Number of obs) | 1435 | 762 | | |
| **Birthweight** | | | | | |
| | (Mean) | 3.18 | 3.12 | 0.07 | * |
| | (Std. Err.) | 0.02 | 0.03 | 0.03 | |
| | (Number of obs) | 758 | 325 | | |
| **Born During 1st Quarter** | | | | | |
| | (Mean) | 0.19 | 0.31 | -0.12 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Born During 2nd Quarter** | | | | | |
| | (Mean) | 0.25 | 0.29 | -0.04 | * |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Born During 3rd Quarter** | | | | | |
| | (Mean) | 0.30 | 0.29 | 0.01 | |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Born During 4th Quarter** | | | | | |
| | (Mean) | 0.27 | 0.12 | 0.15 | ** |
| | (Std. Err.) | 0.01 | 0.01 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Mother's Height (cm)** | | | | | |
| | (Mean) | 150.62 | 149.00 | 1.62 | ** |
| | (Std. Err.) | 0.13 | 0.19 | 0.23 | |
| | (Number of obs) | 1435 | 762 | | |
| **Father's Height (cm)** | | | | | |
| | (Mean) | 161.44 | 159.94 | 1.51 | ** |
| | (Std. Err.) | 0.15 | 0.20 | 0.25 | |
| | (Number of obs) | 1435 | 762 | | |
| **Per Capita Expenditure in 1993 (nominal)** | | | | | |
| | (Mean) | 0.54 | 0.45 | 0.09 | ** |
| | (Std. Err.) | 0.01 | 0.01 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Household Size** | | | | | |
| | (Mean) | 5.285 | 5.592 | -0.3068 | ** |
| | (Std. Err.) | 0.046 | 0.068 | 0.08256 | |
| | (Number of obs) | 1435 | 762 | | |
| **Living in Rural Area in 1993** | | | | | |
| | (Mean) | 0.54 | 0.68 | -0.14 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Mother Has No Education** | | | | | |
| | (Mean) | 0.18 | 0.22 | -0.05 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Mother Has Primary Education** | | | | | |
| | (Mean) | 0.63 | 0.69 | -0.06 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **Mother Has Secondary Education** | | | | | |
| | (Mean) | 0.36 | 0.26 | 0.10 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1435 | 762 | | |
| **District Average Altitude, m above sea level** | | | | | |
| | (Mean) | 0.31 | 0.37 | -0.06 | ** |
| | (Std. Err.) | 0.01 | 0.02 | 0.02 | |
| | (Number of obs) | 1402 | 741 | | |
| **District Average Rainfall in 1991/92** | | | | | |
| | (Mean) | 1.67 | 1.43 | 0.24 | ** |
| | (Std. Err.) | 0.03 | 0.04 | 0.05 | |
| | (Number of obs) | 937 | 473 | | |

* significant at 10 percent
** significant at 5 percent

**TABLE 7.3: INSTRUMENTAL VARIABLES FIRST STAGE RESULTS**
**First Stage Results for Children Aged 0-5 in 1993**

| | [1] | [2] | [3] | [4] |
|---|---|---|---|---|
| | *Dependent Binary Variable: Stunted in 1993* | | | |
| ***Instruments:*** | | | | |
| Mother's height (cm) | -0.012 | -0.012 | -0.012 | -0.012 |
| | [0.002]*** | [0.002]*** | [0.002]*** | [0.002]*** |
| Father's height (cm) | -0.008 | -0.008 | -0.008 | -0.007 |
| | [0.002]*** | [0.002]*** | [0.002]*** | [0.002]*** |
| Born in First Quarter of Year | | | 0.276 | 0.273 |
| | | | [0.027]*** | [0.027]*** |
| Born in Second Quarter of Year | | | 0.205 | 0.2 |
| | | | [0.026]*** | [0.027]*** |
| Born in Third Quarter of Year | | | 0.153 | 0.154 |
| | | | [0.025]*** | [0.026]*** |
| ***Additional Controls:*** | | | | |
| Female | -0.024 | -0.027 | -0.028 | -0.032 |
| | [0.019] | [0.020] | [0.019] | [0.019]* |
| Age | 0.036 | 0.037 | 0.036 | 0.037 |
| | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** |
| Living in Rural Area | 0.102 | 0.053 | 0.104 | 0.056 |
| | [0.022]*** | [0.026]** | [0.021]*** | [0.025]** |
| Per Capita Expenditure | -0.028 | -0.016 | -0.029 | -0.019 |
| | [0.030] | [0.031] | [0.030] | [0.031] |
| Mother Has Primary Education | -0.028 | -0.035 | -0.034 | -0.042 |
| | [0.028] | [0.029] | [0.028] | [0.028] |
| Mother Has Secondary Education | -0.029 | -0.029 | -0.034 | -0.035 |
| | [0.031] | [0.031] | [0.030] | [0.031] |
| Household Size in 1993 | 0.009 | 0.009 | 0.008 | 0.008 |
| | [0.006] | [0.006] | [0.006] | [0.006] |
| Month of Interview Dummies | YES | YES | YES | YES |
| District Fixed Effects | | YES | | YES |
| Constant | 3.344 | | 3.113 | |
| | [0.383]*** | | [0.379]*** | |
| Observations | 2197 | 2197 | 2197 | 2197 |
| R-squared | 0.102 | 0.125 | 0.142 | 0.163 |
| Test of joint significance of excluded instruments: | | | | |
| **F-statistic** | **35.02** | **33.95** | **39.82** | **37.55** |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

**TABLE 7.4. INSTRUMENTAL VARIABLES ESTIMATION OF ACUTE ILLNESS FOR CHILDREN AGED 0-5 IN 1993**
**Second Stage Results**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] |
|---|---|---|---|---|---|---|---|
| *Dependent Binary Variable: Acute Illness in 1993* | OLS | Instruments: Parents' Heights | Instruments: Parents' Heights | Instruments: Birth Quarters | Instruments: Birth Quarters | Instruments: Parents' Heights and Birth Quarters | Instruments: Parents' Heights and Birth Quarters |
| Stunted in 1993 | 0.038 | 0.251 | 0.246 | 0.104 | 0.106 | 0.166 | 0.163 |
| | [0.020]* | [0.116]** | [0.117]** | [0.096] | [0.097] | [0.074]** | [0.074]** |
| Mother's height (cm) | | | | -0.003 | -0.003 | | |
| | | | | [0.002] | [0.002] | | |
| Father's height (cm) | | | | 0 | 0 | | |
| | | | | [0.002] | [0.002] | | |
| Female | 0.028 | 0.029 | 0.034 | 0.021 | 0.03 | 0.022 | 0.032 |
| | [0.018] | [0.019] | [0.019]* | [0.019] | [0.018] | [0.019] | [0.018]* |
| Age in 1993 | -0.028 | -0.034 | -0.036 | -0.029 | -0.03 | -0.031 | -0.033 |
| | [0.006]*** | [0.007]*** | [0.007]*** | [0.007]*** | [0.007]*** | [0.006]*** | [0.006]*** |
| Living in Rural Area in 1993 | -0.026 | -0.053 | -0.039 | -0.052 | -0.031 | -0.059 | -0.034 |
| | [0.025] | [0.024]** | [0.026] | [0.023]** | [0.025] | [0.022]*** | [0.025] |
| Per Capita Expenditure in 1993 | 0.044 | 0.063 | 0.053 | 0.064 | 0.05 | 0.064 | 0.049 |
| | [0.030] | [0.030]** | [0.030]* | [0.030]** | [0.030]* | [0.030]** | [0.030]* |
| Mother Has Primary Education | -0.008 | -0.003 | -0.003 | -0.007 | -0.008 | -0.004 | -0.005 |
| | [0.026] | [0.026] | [0.026] | [0.026] | [0.026] | [0.026] | [0.026] |
| Mother Has Secondary Education | -0.033 | -0.023 | -0.024 | -0.032 | -0.029 | -0.031 | -0.028 |
| | [0.029] | [0.029] | [0.030] | [0.028] | [0.029] | [0.029] | [0.029] |
| Household Size in 1993 | -0.019 | -0.019 | -0.022 | -0.022 | -0.02 | -0.023 | -0.021 |
| | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** |
| Month of Interview Dummies | YES | YES | YES | YES | YES | YES | YES |
| District Fixed Effects | YES | | YES | | YES | | YES |
| Observations | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 |
| R-squared | 0.29 | | | | | | |
| Overidentification test of all instruments: | | | | | | | |
| **Hansen J Statistic / Chi-square P-value** | | **0.73 / 0.39** | **0.97 / 0.33** | **1.17 / 0.56** | **1.05 / 0.59** | **2.51 / 0.64** | **2.86 / 0.58** |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

Note: The joint null hypothesis for the overidentification test is:
- The instruments are valid instruments, i.e., uncorrelated with the error term
- The excluded instruments are correctly excluded from the estimated equation

Under the null, the Hansen J statistic is distributed as chi-squared in the number of (L-K) overidentifying restrictions

**TABLE 7.5: DETERMINANTS OF ACUTE ILLNESS IN 1997 FOR CHILDREN AGED 0-5 IN 1993**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| *Dependent Binary Variable: Acute Illness in 1997* | OLS | Marginal Effects Probit | OLS | Marginal Effects Probit | Fixed Effects OLS | IV | IV | IV |
| Stunted in 1993 | 0.053 | 0.054 | 0.054 | 0.055 | 0.051 | 0.086 | -0.004 | 0.032 |
| | [0.023]** | [0.023]** | [0.023]** | [0.023]** | [0.023]** | [0.139] | [0.114] | [0.089] |
| Mother's height (cm) | | | | | | | -0.003 | |
| | | | | | | | [0.002] | |
| Father's height (cm) | | | | | | | 0.001 | |
| | | | | | | | [0.002] | |
| Female | 0.022 | 0.023 | 0.023 | 0.023 | 0.026 | 0.023 | 0.021 | 0.022 |
| | [0.021] | [0.021] | [0.021] | [0.022] | [0.021] | [0.021] | [0.021] | [0.021] |
| Age in 1997 | -0.027 | -0.028 | -0.027 | -0.027 | -0.026 | -0.028 | -0.025 | -0.026 |
| | [0.006]*** | [0.007]*** | [0.006]*** | [0.007]*** | [0.007]*** | [0.008]*** | [0.007]*** | [0.007]*** |
| Living in Rural Area in 1997 | -0.067 | -0.068 | -0.067 | -0.068 | -0.063 | -0.071 | -0.061 | -0.065 |
| | [0.023]*** | [0.023]*** | [0.024]*** | [0.024]*** | [0.028]** | [0.029]** | [0.027]** | [0.026]** |
| HH Per Capita Expenditure in 1997 | 0.026 | 0.026 | 0.026 | 0.027 | 0.021 | 0.027 | 0.025 | 0.025 |
| | [0.013]* | [0.013]* | [0.013]** | [0.013]** | [0.014] | [0.014]** | [0.014]* | [0.013]* |
| Mother Has Primary Education | -0.064 | -0.065 | -0.064 | -0.065 | -0.062 | -0.063 | -0.065 | -0.064 |
| | [0.030]** | [0.031]** | [0.030]** | [0.031]** | [0.031]** | [0.030]** | [0.031]** | [0.030]** |
| Mother Has Secondary Education | -0.035 | -0.035 | -0.03 | -0.031 | -0.015 | -0.029 | -0.033 | -0.031 |
| | [0.033] | [0.033] | [0.033] | [0.034] | [0.034] | [0.033] | [0.033] | [0.033] |
| Household Size in 1997 | -0.01 | -0.01 | -0.01 | -0.01 | -0.011 | -0.011 | -0.009 | -0.01 |
| | [0.007] | [0.007] | [0.006] | [0.007] | [0.007]* | [0.007] | [0.007] | [0.007] |
| Month of Interview Dummies | | | YES | YES | YES | YES | YES | YES |
| District Fixed Effects | | | | | YES | YES | YES | YES |
| Instruments | | | | | | Parents' Heights | Birth Quarters | Parents' Heights and Birth Quarters |
| Constant | 0.746 | | 0.672 | | | | | |
| | [0.066]*** | | [0.133]*** | | | | | |
| Observations | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 |
| R-squared | 0.019 | | 0.022 | | 0.05 | | | |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

**TABLE 7.6: DETERMINANTS OF ACUTE ILLNESS IN 2000 FOR CHILDREN AGED 0-5 IN 1993**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| *Dependent Binary Variable: Acute Illness in 2000* | OLS | Marginal Effects Probit | OLS | Marginal Effects Probit | Fixed Effects OLS | IV | IV | IV |
| Stunted in 1993 | 0.002 | 0.002 | -0.003 | -0.002 | 0.003 | 0.195 | 0.032 | 0.097 |
| | [0.023] | [0.023] | [0.023] | [0.023] | [0.023] | [0.144] | [0.116] | [0.090] |
| Mother's height (cm) | | | | | | | -0.003 | |
| | | | | | | | [0.002] | |
| Father's height (cm) | | | | | | | 0 | |
| | | | | | | | [0.002] | |
| Female | 0.006 | 0.006 | 0.002 | 0.002 | 0.007 | 0.007 | 0.003 | 0.005 |
| | [0.021] | [0.021] | [0.021] | [0.021] | [0.021] | [0.022] | [0.021] | [0.021] |
| Age in 2000 | -0.007 | -0.007 | -0.006 | -0.006 | -0.007 | -0.011 | -0.007 | -0.008 |
| | [0.006] | [0.006] | [0.006] | [0.006] | [0.006] | [0.007] | [0.007] | [0.006] |
| Living in Rural Area in 2000 | -0.042 | -0.042 | -0.042 | -0.043 | -0.008 | -0.064 | -0.047 | -0.053 |
| | [0.023]* | [0.023]* | [0.023]* | [0.023]* | [0.031] | [0.028]** | [0.026]* | [0.025]** |
| HH Per Capita Expenditure in 2000 | 0.065 | 0.066 | 0.063 | 0.064 | 0.057 | 0.071 | 0.066 | 0.067 |
| | [0.014]*** | [0.014]*** | [0.014]*** | [0.014]*** | [0.014]*** | [0.015]*** | [0.015]*** | [0.015]*** |
| Mother Has Primary Education | 0.015 | 0.015 | 0.017 | 0.018 | 0.023 | 0.02 | 0.016 | 0.019 |
| | [0.031] | [0.031] | [0.031] | [0.031] | [0.031] | [0.031] | [0.031] | [0.030] |
| Mother Has Secondary Education | 0.006 | 0.006 | 0.009 | 0.009 | 0.02 | 0.018 | 0.012 | 0.014 |
| | [0.033] | [0.034] | [0.034] | [0.034] | [0.035] | [0.034] | [0.034] | [0.034] |
| Household Size in 2000 | -0.009 | -0.009 | -0.009 | -0.009 | -0.007 | -0.014 | -0.011 | -0.012 |
| | [0.006] | [0.007] | [0.006] | [0.007] | [0.007] | [0.008]* | [0.007] | [0.007]* |
| Month of Interview Dummies | | | YES | YES | YES | YES | YES | YES |
| District Fixed Effects | | | | | YES | YES | YES | YES |
| Instruments | | | | | | Parents' Heights | Birth Quarters | Parents' Heights and Birth Quarters |
| Constant | 0.5 | | 0.625 | | | | | |
| | [0.079]*** | | [0.096]*** | | | | | |
| Observations | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 | 2197 |
| R-squared | 0.015 | | 0.022 | | 0.045 | | | |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

**TABLE 7.7: CHANGES IN NUTRITIONAL STATUS BETWEEN 1993 AND 2000**

| Nutritional Status | Percent |
|---|---|
| Not Stunted in either 1993 or 2000 | 54.5% |
| Stunted in both 1993 and 2000 | 18.6% |
| Stunted in 1993, but not in 2000 | 13.8% |
| Not Stunted in 1993, but Stunted in 2000 | 14.1% |

**TABLE 7.8: DETERMINANTS OF CHANGES IN STUNTING STATUS BETWEEEN 1993 AND 2000**

| | [1] | [2] | [3] | [4] | [5] | [6] | [7] | [8] |
|---|---|---|---|---|---|---|---|---|
| | *Dependent Binary Variabile: Improvement in Stunting Status* | | | | *Dependent Binary Variabile: Deterioration in Stunting Status* | | | |
| Female | 0.011 | 0.012 | 0.023 | 0.023 | -0.039 | -0.037 | -0.039 | -0.042 |
| | [0.035] | [0.034] | [0.034] | [0.035] | [0.020]* | [0.020]* | [0.020]** | [0.020]** |
| Age in 2000 | -0.046 | -0.044 | -0.038 | -0.037 | -0.012 | -0.01 | -0.011 | -0.008 |
| | [0.011]*** | [0.010]*** | [0.010]*** | [0.011]*** | [0.006]** | [0.006]* | [0.006]* | [0.006] |
| Per Capita Expenditure in 2000 | 0.056 | 0.04 | 0.035 | 0.026 | -0.054 | -0.045 | -0.047 | -0.043 |
| | [0.025]** | [0.025] | [0.025] | [0.026] | [0.012]*** | [0.012]*** | [0.012]*** | [0.012]*** |
| Mother has Secondary Education | 0.011 | -0.011 | -0.003 | 0.016 | -0.034 | -0.01 | -0.016 | -0.016 |
| | [0.044] | [0.043] | [0.043] | [0.045] | [0.024] | [0.023] | [0.023] | [0.024] |
| Household Size in 2000 | -0.004 | -0.002 | -0.005 | -0.005 | 0.012 | 0.008 | 0.01 | 0.009 |
| | [0.010] | [0.010] | [0.009] | [0.010] | [0.007]* | [0.006] | [0.006] | [0.007] |
| Living in Rural Area in 2000 | -0.06 | -0.044 | -0.047 | -0.031 | 0.034 | 0.029 | 0.033 | 0.003 |
| | [0.040] | [0.039] | [0.038] | [0.054] | [0.022] | [0.021] | [0.021] | [0.027] |
| Mother's height (cm) in 1993 | | 0.016 | 0.016 | 0.016 | | -0.014 | -0.014 | -0.015 |
| | | [0.003]*** | [0.003]*** | [0.003]*** | | [0.002]*** | [0.002]*** | [0.002]*** |
| Father's height (cm) in 1993 | | 0.015 | 0.015 | 0.015 | | -0.008 | -0.007 | -0.008 |
| | | [0.003]*** | [0.003]*** | [0.003]*** | | [0.002]*** | [0.002]*** | [0.002]*** |
| Born in First Quarter of Year | | | -0.238 | -0.231 | | | 0.17 | 0.171 |
| | | | [0.058]*** | [0.061]*** | | | [0.029]*** | [0.030]*** |
| Born in Second Quarter of Year | | | -0.267 | -0.251 | | | 0.15 | 0.154 |
| | | | [0.058]*** | [0.062]*** | | | [0.027]*** | [0.028]*** |
| Born in Third Quarter of Year | | | -0.065 | -0.059 | | | 0.058 | 0.067 |
| | | | [0.060] | [0.062] | | | [0.024]** | [0.025]*** |
| District Fixed Effects | | | | YES | | | | YES |
| Constant | 0.876 | -3.989 | -3.796 | | 0.333 | 3.648 | 3.568 | |
| | [0.133]*** | [0.687]*** | [0.672]*** | [0.717]*** | [0.072]*** | [0.402]*** | [0.401]*** | [0.407]*** |
| Observations | 762 | 762 | 762 | 762 | 1435 | 1435 | 1435 | 1435 |
| R-squared | 0.035 | 0.093 | 0.137 | 0.171 | 0.031 | 0.077 | 0.107 | 0.131 |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

The regressions in columns 1-4 only look at the cohort of children who were stunted in 1993:
Improvement in Stunting Status is a dummy equal to 1 if a child was stunted in 1993 but no longer stunted in 2000; it is equal to 0 for children who were stunted in 1993 and remain stunted in 2000.

The regressions in columns 5-8 only look at the cohort of children who were not stunted in 1993:
Deterioration in Stunting Status is a dummy equal to 1 if a child was not stunted in 1993 but is stunted in 2000; it is equal to 0 for children who were not stunted in 1993 and remain not stunted in 2000.

**Table 7.9: First Stage Results, Using Height-for-Age *z*-scores**

| **First Stage Results** | |
|---|---|
| *Dependent Variable: Height-for-Age Z-score* | |
| Mother's height (cm) in 1993 | 0.049 |
| | [0.010]*** |
| Father's height (cm) in 1993 | 0.024 |
| | [0.009]*** |
| Birth Quarter 1 | -1.076 |
| | [0.151]*** |
| Birth Quarter 2 | -0.927 |
| | [0.147]*** |
| Birth Quarter 3 | -0.63 |
| | [0.145]*** |
| Age in 1993 | -0.213 |
| | [0.029]*** |
| Living in Rural Area in 1993 | -0.309 |
| | [0.132]** |
| Female | 0.179 |
| | [0.099]* |
| Per Capita Expenditure in 1993 (Nominal) | 0.329 |
| | [0.165]** |
| Mother has Primary Education | 0.085 |
| | [0.143] |
| Mother has Secondary Education | -0.077 |
| | [0.154] |
| Household Size | -0.055 |
| | [0.032]* |
| Month of Interview Dummies | YES |
| District Fixed Effects | YES |
| Observations | 1997 |
| R-squared | 0.133 |
| Robust standard errors in brackets | |
| * significant at 10%; ** significant at 5%; *** significant at 1% | |

## Table 7.10: Second Stage Results, Using Height-for-Age *z*-scores

| **Second Stage Results** | | | |
|---|---|---|---|
| | -1 | -2 | -3 |
| | Any Illness in 1993 | Any Illness in 1997 | Any Illness in 2000 |
| Height-for-Age Z-Score | -0.042 | -0.006 | -0.022 |
| | [0.020]** | [0.025] | [0.025] |
| Age | -0.04 | -0.023 | -0.004 |
| | [0.007]*** | [0.008]*** | [0.007] |
| Living in Rural Area | -0.021 | -0.056 | -0.002 |
| | [0.027] | [0.031]* | [0.033] |
| Female | 0.033 | 0.03 | 0.012 |
| | [0.020]* | [0.022] | [0.022] |
| Per Capita Expenditure | 0.069 | 0.02 | 0.067 |
| | [0.033]** | [0.015] | [0.016]*** |
| Mother has Primary Education | 0.003 | -0.071 | 0.027 |
| | [0.027] | [0.032]** | [0.032] |
| Mother has Secondary Education | -0.024 | -0.021 | 0.011 |
| | [0.030] | [0.035] | [0.036] |
| Household Size | -0.02 | -0.011 | -0.008 |
| | [0.006]*** | [0.007] | [0.007] |
| Month of Interview Dummies | YES | YES | YES |
| District Fixed Effects | YES | YES | YES |
| Observations | 1997 | 1997 | 1997 |
| Robust standard errors in brackets | | | |
| * significant at 10%; ** significant at 5%; *** significant at 1% | | | |

## Chapter 8: Conclusion and Policy Implications

In this dissertation, I investigate the determinants of early childhood morbidity using household level panel data from Indonesia. Specifically, I use three waves of the Indonesia Family Life Survey in 1993, 1997 and 2000 to test whether early childhood nutritional status is an important cause of acute illness both in the immediate and long term.

While there is a large medical literature suggesting that malnutrition may negatively impact the immune system and make one more susceptible to infection or more severe disease once infected, there has been little rigorous empirical evidence in the economics literature on the causal link between malnutrition and acute illness. The identification challenge, which most studies have failed to overcome, arises from that fact that household behavioral decisions that influence investment in a child's nutrition and growth are very likely correlated with other household decisions that affect a child's incidence of illness. These include decisions to invest in hygiene and sanitation or a mother's knowledge and use of appropriate feeding practices. There may also be unobserved risk factors, such as genetic endowments, which introduce correlation between one of the regressors – nutritional status – and the error term in a disease production equation.

I use modern econometric methods to avoid omitted variable bias and identify a causal relationship between nutritional status and illness outcomes. Specifically, I

instrument for a child's nutritional status with parents' heights and the child's birth quarter, which plausibly affect illness outcomes only through their impact on nutritional status. There is recent empirical evidence suggesting that both parents' heights and a child's birth quarter are important indicators of nutritional deficiency in young children. The first stage of my instrumental variables estimator is consistent with these results. My second stage findings suggest that early childhood malnutrition substantially affects susceptibility to infectious disease. I find that preschool children aged 0-5 who are stunted in 1993 have a 16 percent greater probability of reporting symptoms of contemporaneous acute illness than children who are not stunted. Further, I find that early childhood stunting continues to affect the likelihood of developing acute illness even four years later, when the children in my sample are 4-9 years old. By the time the same children are 7-12 years of age, however, the effects of early childhood stunting seem to dissipate. Importantly, I find evidence of changes in stunting status over this 7-year period in which 13.8 percent of the children improve their nutritional status and 14.1 percent deteriorate. While I do not have instruments to identify these changes, I present some suggestive evidence that these changes in nutritional status may be the result of "catch-up" growth in my sample of children. I find that such improvements in nutritional status are strongly correlated with younger age at initial stunting, taller parents, being born late in the year and higher household income. Deteriorations in nutritional status are correlated with having shorter parents, lower household income, and a mother with less education.

My results on the effect of nutritional status on contemporaneous and future acute illness have several policy implications. A direct implication is that resources targeted towards improving nutritional status in young children are likely not only to remove nutritional deficiencies, but also to lead to better health outcomes and a decreased burden of future disease. Further, the finding that improvements in stunting are correlated with household income and maternal education suggests that programs aimed at increasing household incomes and female education can have beneficial side-effects by improving young children's nutritional status. An important issue is whether malnutrition influences incidence of disease or severity of disease once infected. While I cannot ascertain from my study whether children are more likely to become infected, or simply more likely to develop symptoms serious enough to report, there are clear benefits to improving their nutrition in either case.

If stunting increases susceptibility to infection, then malnutrition indirectly increases the pool of infected children. This implies that there may be positive externalities associated with reducing malnutrition due to its impact on infectious disease (Miguel and Kremer 2006; Gersovitz and Hammer 2001; Gersovitz 2000). Miguel and Kremer (2006) estimate the program effects of a school-based de-worming project in Kenya and find that infection control substantially improved health and school participation among both treated and untreated children, essentially doubling the private benefits of the de-worming treatment. Hence, decreasing the prevalence of stunting in children under five not only results in

immediate health benefits but may have long-term benefits in terms of human capital outcomes for the targeted children and external benefits in terms of lower prevalence of infectious disease.

Overall, my results suggest that improving preschoolers' nutritional status can reduce short-term and long-term costs associated with poor health outcomes. In addition, improvements in nutritional status can also yield positive human capital returns since healthier children are likely to miss fewer days of school and are likely more alert and energetic in the classroom.

# Appendix A

**TABLE A2: INSTRUMENTAL VARIABLES ESTIMATION OF ACUTE ILLNESS FOR CHILDREN AGED 0-5 IN 1993**
**Second Stage Results**

| **Unbalanced Panel** | [1] | [2] | [3] | [4] | [5] | [6] |
|---|---|---|---|---|---|---|
| *Dependent Binary Variable: Acute Illness in 1993* | Instruments: Parents' Heights | Instruments: Parents' Heights | Instruments: Birth Quarters | Instruments: Birth Quarters | Instruments: Parents' Heights and Birth Quarters | Instruments: Parents' Heights and Birth Quarters |
| Stunted in 1993 | 0.292 | 0.288 | 0.077 | 0.082 | 0.163 | 0.163 |
| | [0.118]** | [0.121]** | [0.097] | [0.096] | [0.075]** | [0.074]** |
| Mother's height (cm) | | | -0.003 | -0.003 | | |
| | | | [0.002] | [0.002] | | |
| Father's height (cm) | | | -0.001 | 0 | | |
| | | | [0.002] | [0.002] | | |
| Female | 0.038 | 0.042 | 0.027 | 0.034 | 0.03 | 0.037 |
| | [0.018]** | [0.018]** | [0.018] | [0.017]** | [0.018]* | [0.017]** |
| Age in 1993 | -0.036 | -0.038 | -0.029 | -0.03 | -0.032 | -0.033 |
| | [0.007]*** | [0.007]*** | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** |
| Living in Rural Area in 1993 | -0.051 | -0.039 | -0.045 | -0.028 | -0.053 | -0.032 |
| | [0.023]** | [0.025] | [0.022]** | [0.024] | [0.021]** | [0.024] |
| Per Capita Expenditure in 1993 | 0.062 | 0.058 | 0.069 | 0.053 | 0.068 | 0.052 |
| | [0.028]** | [0.028]** | [0.028]** | [0.028]* | [0.028]** | [0.028]* |
| Mother Has Primary Education | 0.013 | 0.016 | 0.011 | 0.01 | 0.013 | 0.013 |
| | [0.025] | [0.025] | [0.024] | [0.024] | [0.024] | [0.025] |
| Mother Has Secondary Education | 0.007 | 0.008 | -0.003 | -0.002 | -0.001 | 0.001 |
| | [0.028] | [0.028] | [0.027] | [0.027] | [0.027] | [0.027] |
| Month of Interview Dummies | YES | YES | YES | YES | YES | YES |
| District Fixed Effects | | YES | | YES | | YES |
| Observations | 2493 | 2493 | 2493 | 2493 | 2493 | 2493 |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

**TABLE A2: INSTRUMENTAL VARIABLES ESTIMATION OF ACUTE ILLNESS FOR CHILDREN AGED 0-5 IN 1993**
**Second Stage Results**

| **Unbalanced Panel** | [1] | [2] | [3] | [4] | [5] | [6] |
|---|---|---|---|---|---|---|
| *Dependent Binary Variable: Acute Illness in 1993* | Instruments: Parents' Heights | Instruments: Parents' Heights | Instruments: Birth Quarters | Instruments: Birth Quarters | Instruments: Parents' Heights and Birth Quarters | Instruments: Parents' Heights and Birth Quarters |
| Stunted in 1993 | 0.292 | 0.288 | 0.077 | 0.082 | 0.163 | 0.163 |
| | [0.118]** | [0.121]** | [0.097] | [0.096] | [0.075]** | [0.074]** |
| Mother's height (cm) | | | -0.003 | -0.003 | | |
| | | | [0.002] | [0.002] | | |
| Father's height (cm) | | | -0.001 | 0 | | |
| | | | [0.002] | [0.002] | | |
| Female | 0.038 | 0.042 | 0.027 | 0.034 | 0.03 | 0.037 |
| | [0.018]** | [0.018]** | [0.018] | [0.017]** | [0.018]* | [0.017]** |
| Age in 1993 | -0.036 | -0.038 | -0.029 | -0.03 | -0.032 | -0.033 |
| | [0.007]*** | [0.007]*** | [0.006]*** | [0.006]*** | [0.006]*** | [0.006]*** |
| Living in Rural Area in 1993 | -0.051 | -0.039 | -0.045 | -0.028 | -0.053 | -0.032 |
| | [0.023]** | [0.025] | [0.022]** | [0.024] | [0.021]** | [0.024] |
| Per Capita Expenditure in 1993 | 0.062 | 0.058 | 0.069 | 0.053 | 0.068 | 0.052 |
| | [0.028]** | [0.028]** | [0.028]** | [0.028]* | [0.028]** | [0.028]* |
| Mother Has Primary Education | 0.013 | 0.016 | 0.011 | 0.01 | 0.013 | 0.013 |
| | [0.025] | [0.025] | [0.024] | [0.024] | [0.024] | [0.025] |
| Mother Has Secondary Education | 0.007 | 0.008 | -0.003 | -0.002 | -0.001 | 0.001 |
| | [0.028] | [0.028] | [0.027] | [0.027] | [0.027] | [0.027] |
| Month of Interview Dummies | YES | YES | YES | YES | YES | YES |
| District Fixed Effects | | YES | | YES | | YES |
| Observations | 2493 | 2493 | 2493 | 2493 | 2493 | 2493 |

Robust standard errors in brackets
* significant at 10%; ** significant at 5%; *** significant at 1%

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