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#### Abstract

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#### Keywords

Childhood morbidity, Diseases, Health, INCAP, Guatemala, Cardiovascular, Type 2 diabetes, Metabolic syndrome, Height, Life outcomes

#### Disciplines

Demography, Population, and Ecology | Family, Life Course, and Society | Social and Behavioral Sciences | Sociology

#### Comments

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# The Effects of Early Childhood Diseases on Young Adult Health in Guatemala

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## Abstract

This study examines the relationship between early childhood morbidity and young adult health in a poor developing country with a high prevalence of childhood diseases. I take advantage of the rich observational data collected by the Institute of Nutrition of Central America and Panama (INCAP) Longitudinal Study in Guatemala to estimate the effects of five types of childhood illnesses on metabolic syndrome in young adulthood, a predictor of cardiovascular disease and type 2 diabetes. This analysis supports the hypothesis that poor health in childhood is associated with a higher probability of metabolic syndrome in young adulthood. I also find that adult height, often used as a proxy for childhood conditions, does not capture the effects of childhood morbidity. Thus, studies that include height but no direct measure of childhood morbidity are likely to underestimate the effects of child health on later life outcomes. The results highlight the significance of child health programs that can improve population health over the life course.

#### Introduction

A growing body of literature suggests that childhood circumstances have lasting effects on morbidity later in life (Barker 1995; Peck 1994; Preston, Hill and Drevenstedt 1998; Wadsworth 1986). Prior research has focused on an array of childhood factors such as social and economic deprivation, nutrition, or exposure to environmental toxins and infectious disease. Some work has focused on the "programming" of chronic diseases during gestation or early childhood (Barker 1995) and other research has emphasized the accumulation of insults from exposure to adverse conditions over the life course (Kuh and Ben-Shlomo 1997). While empirical evidence has accumulated, suggesting that we can best understand chronic morbidity with data on circumstances throughout the life course, empirical investigation of *early* life influences on adult health has been limited by the scarcity of rich life course data.

Most research on the effects of childhood disease on adult health outcomes has been based on data from affluent countries. This study extends the literature by utilizing data from a developing country with a high prevalence of childhood diseases. Specifically, I examine the association between childhood illness and young adult health among a sample of Guatemalan adults aged 29-35, who were born in four villages in the district of El Progreso between 1968 and 1973. This cohort participated in a randomized community nutritional supplementation program conducted by the Institute for Nutrition for Central America and Panama (INCAP) between 1969 and 1977, during which data on early life conditions were collected. A follow-up survey was conducted in 2002-2004 that included clinic-reported measures of adult health. I take advantage of the rich

observational data collected in the INCAP study to estimate the effects of five types of childhood illness (diarrhea, anorexia, serious illness, respiratory and infectious diseases) on adult health. To determine whether childhood health has long-term and direct consequences for adult health, we control for a number of confounding factors such as socioeconomic status in childhood, achieved education, as well as adult smoking behavior. Next, I examine the extent to which adult height captures the estimated effects of childhood health, because adult height is commonly used as a proxy for childhood health and deprivation in studies where direct measures are not available. I then explore the extent to which socioeconomic status and health behaviors in adulthood mediate the influence of childhood morbidity on adult health.

#### Background

The majority of research on chronic disease in the social sciences and social epidemiology has focused on adult life circumstances, and specifically the relationship between socioeconomic status and disease prevalence and mortality. In general, researchers agree that socioeconomic status is negatively associated with chronic health conditions throughout the life cycle (Adler et al. 1994). A growing body of literature has begun to address the effects of childhood circumstances on adult socioeconomic status and adult morbidity and mortality (Kuh and Ben-Shlomo 1997). Researchers have found that childhood conditions influence adult health through a variety of pathways such as nutrition (Gunnell et al. 1996), conditions in utero (Osmond and Barker 2000; Hales et al. 1991), behavioral factors (Lundberg 1993, 1997), or exposure to infectious disease,

viruses, or environmental toxins (Zhu et al. 2000; Hall and Peckham 1997; Power and Peckham 1990).

Previous research has documented a positive association between observed early life morbidity and subsequent cardiovascular disease and its risk factors. The best documented positive relationship is between infectious diseases in childhood and adult cardiovascular disease (Hall and Peckham 1997). In an ecological analysis in the US, Buck and Simpson (1982) find that the level of infection in childhood was positively related to rates of heart disease later in life. Other studies have used markers of inflammation to understand the effects of earlier infections on the future risk of heart disease. Markers of chronic inflammation are generally positively associated with atherosclerosis or coronary heart disease (Zhu et al. 2000; Espinola-Klein et al. 2002; Roivainen et al. 2000; Danesh et al. 2000). This chronic inflammation over time, plaque accumulation, and development of atherosclerotic lesions are thought to be the mechanisms by which infectious diseases in childhood influence heart disease in adulthood (Buck and Simpson 1982; Crimmins and Finch 2006).

Other studies have specifically focused on the association between diarrhea and anorexia<sup>1</sup> in early childhood and risk factors for cardiovascular disease in adulthood. Although Batty et al. (2007) find no significant relationship, several studies suggest a positive relationship between childhood diarrhea and adult cardiovascular health. Smith et al.

<sup>&</sup>lt;sup>1</sup> Anorexia here refers to a condition where young children have a lack of appetite and therefore cannot ingest necessary calories due to illness, which is correlated with diarrheal disease. This is not to be confused with anorexia nervosa, an eating disorder characterized by low body weight and body image distortion.

(2006) find that British children suffering from dehydration from diarrheal disease had higher blood pressure as adults, even when adjusting for confounding factors. Korczowski et al. (2004) find higher levels of PCT and CRP (indicators of inflammation) in children hospitalized with diarrhea. Martorell and Habicht (1986) argue that of all infectious diseases, those with diarrheal manifestation are probably the most important for later health outcomes because of their negative effects on child growth (Martinez et al. 1990). Reduced growth in early childhood is hypothesized to have negative effects on a variety of adult health outcomes such as coronary heart disease, diabetes, hypertension, (Barker 1995; Osmund and Barker 2000; Hales et al. 1991).

Finally, some researchers have suggested that childhood respiratory disease may also influence adult health outcomes. While much of this research has focused on the association between childhood respiratory disease and lung health in adulthood (Shaheen, Barker and Holgate 1995; Samet, Tager and Speizer 1983), there is some evidence that group A streptococcal upper respiratory tract infections may affect heart health in adulthood when followed by rheumatic heart disease (RHD) (Elo and Preston 1992). However, there is little evidence of any association between respiratory diseases in early childhood and metabolic syndrome or its components in adulthood.

Since childhood conditions are often unobserved, even in longitudinal studies, researchers have commonly employed adult or "achieved" height as a proxy. Studies using achieved height as an indicator of childhood conditions have concluded that shorter individuals generally experience higher morbidity and mortality in adulthood (Floud,

Wachter and Gregory 1990; Brunner et al. 1996; Elo and Preston 1992; Fogel 1993; Allebeck and Bergh 1992; Fogel and Costa 1997; Rich-Edwards et al. 1995; Waaler 1984; Yarnell et al. 1992). If adult height represents a proxy for childhood circumstances and if early childhood health and social environment is associated with health later in life, then we would expect that achieved height would have no independent effect on adult health if we directly control for these factors. For example, Blackwell, Hayward and Crimmins (2001) found no statistically significant relationship between height and adult chronic disease after controlling for child health. However if achieved height captures the more broadly defined childhood socioeconomic status which also affects adult health, then height may influence adult health, independent of child health (Elo and Preston 1992; Peck and Lundberg 1995).

Estimating the strength of the relationship between childhood morbidity and adult health is complex, not just because of the data requirements, but also because of potential confounding and mediating factors. For example, prior studies reveal that child health is associated with educational attainment (Case, Fertig and Paxson 2002; Kuh and Wadsworth 1993) and income in adulthood (Currie and Madrian 1999; Adler et al. 1994). Childhood health may affect adult health indirectly through these factors in adulthood (Wadsworth and Kuh 1997), rather than directly. Some of the inconclusiveness of this literature on the relationship between childhood conditions and adult health may arise because of imprecise measures of childhood conditions or confounding factors throughout the life course. For example, using proxies for childhood conditions such as adult height or retrospective reports of health status may lead to biased estimates of the

effects of both childhood circumstances and adult circumstances on adult health outcomes. If we underestimate childhood morbidity, then we might overestimate the effect of schooling on adult health (Preston and Taubman 1994). Likewise, unsatisfactory controls for socioeconomic status or health behaviors in adulthood may result in the misestimation of the effects of childhood health conditions on adult health. As Palloni (2006) argues, researchers probably underestimate the effects of child health on adult outcomes, since most of our evidence comes from developed countries, rather than in low-income countries where childhood malnutrition and disease are more prevalent.

In this paper I take advantage of the INCAP Longitudinal Study to examine the extent to which the effect of childhood morbidity on adult health is mediated by factors in adulthood. Our paper tests the following hypotheses derived from prior research. First, I expect that the prevalence of childhood infectious diseases, diarrhea and anorexia will be positively associated with metabolic syndrome and two of its components in adulthood, high blood pressure, and high fasting glucose. I further hypothesize that this relationship will be partially explained by adult socioeconomic position and health-related behaviors. Finally, while I hypothesize that the effects of childhood diseases will be associated with adult health, independent of factors in adulthood, I also expect that adult health behaviors and educational attainment will influence adult health outcomes.

#### **Data and Methods**

This analysis is based on a cohort of young Guatemalan men and women who participated in the Institute of Nutrition of Central America and Panama (INCAP) Longitudinal Study. This cohort took part in a randomized community trial of nutrition supplementation carried out in 1969-1977 and a study led by the International Food Policy Research Institute (IFPRI) in 2002-2004 designed to trace the effects of improved early childhood nutrition on adult function. The data from the original study include rich information on childhood health, nutrition, growth, and the family environment. The follow-up study conducted between 2002 and 2004 includes clinic-measured adult health information as well as data on lifestyle characteristics, education, marriage formation, income and wealth. Spanning 35 years, the INCAP Longitudinal Study is the longest evaluation of a nutrition intervention in developing countries and provides data on childhood conditions rarely available to researchers examining adult health outcomes.

For this analysis, we use 425 respondents that were born in the first six years of the nutritional intervention and whose mothers reported the disease prevalence of their young children. We have the most information on early life conditions for these cohorts. In the 2002-2004 follow-up, there was relatively low attrition<sup>2</sup>. The respondents in the analytic sample used in this analysis are more likely to be female, reside in the study villages, and have slightly less education than the original sample.

#### Dependent Variables

To measure young adult health, I use metabolic syndrome and its five components. Because this sample is composed of young adults, aged 29-35, we cannot study

<sup>&</sup>lt;sup>2</sup> For details on the attrition of this study, see Grajeda et al. 2005.

cardiovascular disease per se, however we can study its risk factors. Metabolic syndrome integrates clinical risk factors associated with increased risk of cardiovascular morbidity and type 2 diabetes (Aguilar Salinas 2004). The five risk factors included are central obesity (waist circumference  $\geq 102$  cm or 40 inches (male),  $\geq 88$  cm or 36 inches(female), dislipidaemia: TG $\geq$  150 mg/dl, dyslipidaemia: HDL-C < 40 mg/dL (male), < 50 mg/dL (female), blood pressure  $\ge 130/85 \text{ mmHg}$ , and fasting plasma glucose  $\geq$  100 mg/dl or drug treatment for elevated glucose (Grundy et al. 2005). Metabolic syndrome is defined as having at least 3 of 5 risk factors and is associated with significantly higher relative risks for coronary heart disease and type 2 diabetes (Aguilar-Salinas et al 2004). The components of the syndrome have synergistic effects on adult health (Groop and Orho-Melander 2001; Ford, Giles and Dietz 2002; Meigs 2002). We measure metabolic syndrome as defined by the Grundy et al (2005). Using this standard, each risk factor is coded as "1" and absence of a risk factor coded "0". These measures of adult health were taken between 2002 and 2004 when respondents were between 29 and 35 years of age. Since collection of all data did not occur at the same time, and response rates vary by the outcome, the sample for each outcome differs  $slightly^3$ . Table 2 reports the prevalence of metabolic syndrome and its components for the analytic sample. 35% of the sample has 3 or more of 5 risk factors, and thus is categorized has having metabolic syndrome. Dislipidaemia is very common among young Guatemalan adults, with about four fifths of the sample having low HDL levels and more than half with high triglycerides. High fasting plasma glucose is less prevalent, with 17% of the

<sup>&</sup>lt;sup>3</sup> In this preliminary draft, the sample size for various regressions varies slightly and the sample sizes are listed at the bottom of each table.

sample with this risk factor. Approximately one third of the sample has high abdominal obesity and an eighth has high blood pressure.

#### Key Independent Variables

In this paper, we examine how five types of childhood diseases are associated with young adult health – serious illness, diarrhea, anorexia, respiratory and infectious diseases. Measures of childhood morbidity are based on mother's reports of the prevalence of these five disease categories. Mothers were asked how many days their child was sick with each type of illness during three-month periods from birth to 24 months. For each childhood disease, I use these data to calculate the percentage of days in each three month period that the mother reported the child to be ill with the disease.

Data on the prevalence of childhood morbidity is not available for all children in all eight time periods. The prevalence of serious illness, diarrhea and anorexia are recorded for all eight periods in the first two years of life for 48% of the sample and for respiratory and infectious diseases for 58% of the sample. In order to take advantage of all data values, I use a relative measure of morbidity in our models. I constructed z scores for each child for each type of disease in each of the eight time periods. I then calculated the average of a child's z score over the eight time periods for each type of morbidity. The summary score for each type of disease is the intensity of disease experience for each individual relative to his/her peers. Therefore in our analysis we use all 425 respondents for whom we have any data on these five childhood diseases. I also estimate our models with a smaller sample for which we have data for all eight periods and the results do not differ.

Figure 1 reports the mean prevalence of illness for our sample over the first two years of life. As shown in Table 1, respiratory diseases are the most common childhood illness in our sample. Newborns are sick with respiratory diseases about 30 percent of the time and this increases throughout the first two years of life to about 40%. Serious illness, diarrhea and anorexia each have an inverted U shape in these two years. Prevalence of these diseases is generally low, 5-15% for the first few months, and then prevalence increases as foods other than breast milk are introduced. These levels of childhood diseases are much higher than those in affluent countries, but common for poor developing countries. However, infectious diseases are relatively uncommon, with the mean prevalence less than one percent. The level of infectious disease reported here is unnaturally low for this context, possibly resulting from the availability of high-quality health services made available to the communities taking part in the nutritional intervention from 1969-1977.

Childhood morbidity is correlated across time and some diseases are correlated with one other. First, I examine how each type of morbidity is serially correlated across the eight periods from five correlation matrices in which each cell shows the correlation between the intensity of the disease in one period and that in another. Table 1 in the appendix presents the mean, standard deviation, and range of the 28 correlation coefficients for each disease category. Serious illness, diarrhea, anorexia and respiratory diseases are moderately correlated across time. The mean correlations are between .25 (diarrheal disease) and .42 (respiratory). However, infectious disease prevalence in one time period

appears to be independent of that in another, possibly resulting from acquired immunity which is not relevant for the other disease categories. Table 2 in the appendix presents correlations between the average z scores for different illnesses. Diarrheal disease and anorexia are both highly correlated with serious illness, leading us to believe that the disease category serious illness includes the most serious forms of both of these morbidities. Diarrhea and anorexia are moderately correlated (.35), as are respiratory diseases with serious illness (.30) and with anorexia (.34). Infectious disease prevalence seems to be independent of other types of morbidities examined here.

#### Control Variables

In our models, we control for confounding factors throughout the life course, including age, sex, exposure to INCAP's high protein nutritional supplement, socioeconomic status in childhood and adulthood, as well as smoking behavior and adult achieved height. Age is measured in exact years from the time of birth until the time the outcome of interest is measured. The respondent's sex is coded as "1" for females and "0" for males. Exposure to the high protein nutritional supplement in utero or the first two years of life is coded as "1" and exposure to the vitamin drink without protein is coded as "0"<sup>4</sup>. Birth weight is coded as a six category variable, for five quintiles of birth weights and one category for missing data. Mother's literacy is used as a measure of childhood socioeconomic circumstances. This information was reported in the 1969-1977 round of data collection, when subjects were children. Parents' education is not used because the level and frequency of formal education is so low for this generation. Socioeconomic status in

<sup>&</sup>lt;sup>4</sup> The first two years of life is the time period in which the supplement was found to have the greatest positive impact on health.

adulthood is measured with a continuous variable for the number of grades of education attained by the age of 21. We introduce a dichotomous measure of smoking behavior indicating whether the respondent was a past or present smoker (coded as 1) or a nonsmoker (coded as  $0)^5$ . Adult height is an average of two measurements taken at different times during the course of the 2002-2004 follow-up data collection by a trained interviewer to account for variation depending on the time of day.

#### Modeling Approach

I use nested logistic regression models to predict metabolic syndrome and each of its five risk factor components. The nested models are organized to evaluate how childhood health is associated with adult morbidity in young adulthood and how this association changes with controls for other variables. The baseline model estimates the effects of individual demographic characteristics, birth weight, exposure to the high-protein nutritional supplement in the first two years of life, socioeconomic status in childhood and childhood morbidity. Model 2 includes adult smoking behavior and adult socioeconomic status in order to examine if childhood morbidity affects adult health through socioeconomic status in adulthood. Model 3 includes achieved height, enabling us to see if adult height captures other aspects of childhood conditions, net of those observed. Model 4 estimates the extent to which adult height serves as a proxy for childhood health or general deprivation. We control for demographic characteristics, adult socioeconomic status, smoking, and adult height, and exposure to the high protein nutritional supplement (because of the high impact of the intervention) and exclude

<sup>&</sup>lt;sup>5</sup> Results do not change if we use a continuous variable for pack years smoked in the subject's life or a categorical variable for pack years smoked.

childhood characteristics not usually observed (birth weight and childhood morbidity). Respondents with missing information on control variables are flagged as missing and as coefficients for these categories are seldom statistically significant, they are not shown in our tables. All models calculate robust standard errors, with siblings grouped by the mother.

#### Results

First I explore the relationship between five childhood disease and metabolic syndrome in adulthood, net of demographic characteristics and childhood conditions. These results are presented in Tables 3 in the column labeled Model 1. As expected we find a significant positive association between serious illnesses and anorexia and metabolic syndrome. For example, a one standard deviation increase in the prevalence of anorexia relative to one's peers is associated with a 58% increase in the odds of metabolic syndrome in young adulthood. The effects of childhood diseases on adult health are not equally powerful for all diseases. Diarrheal diseases and those labeled "infectious" also have a positive relationship with metabolic syndrome, however the relationship was not statistically significant. We found no relationship between respiratory diseases in early childhood and metabolic syndrome in adulthood.

Model 2 adds adult socioeconomic status and smoking behavior in order to examine if childhood morbidity affects adult health through socioeconomic status in adulthood. The associations found between childhood health and morbidity in young adulthood persisted.

As these relationships did not change, we find that the relationship between childhood health and adult health is *not* mediated by adult SES or smoking behavior.

With models 3 and 4, we explore to what extent height captures the effects of childhood morbidity. Model 3 includes childhood conditions, adult socioeconomic status and smoking, as well as achieved height, enabling us to see if adult height captures other aspects of childhood conditions, net of those observed. Here, we see that the odds ratios for childhood illness are not significantly changed when we add adult height. Moreover, in this model, adult height has no significant additional effect on adult health.

With Model 4, we further explore the use of adult height as a proxy for childhood conditions. We exclude those variables that are commonly missing in studies of adult morbidity: childhood morbidity and birth weight. In these analyses, we find that the coefficient for height is very small and not significant. Thus, height seems to be an imperfect measure of childhood conditions in a population with high prevalence of morbidity at young ages.

Next we turn to our secondary outcomes of interest, the five components of metabolic syndrome. While metabolic syndrome, having three or more of the five risk factors is a stronger predictor of cardiovascular disease and type 2 diabetes than any of its components alone, many studies have examined one or more of these components as outcomes, if information on all of them is not available. To compare our results to those

studies, we examine the relationships between childhood morbidity and these risk factors. Results are shown in Tables 4 through 8.

As expected, I find a strong positive association between serious illness and diarrheal disease in childhood and impaired fasting glucose, a precursor of type two diabetes (Nichols, Hillier and Brown 2007). I also find a positive relationship between three types of childhood morbidity and abdominal adiposity in young adulthood. Although this relationship has not been well studied, it fits into the broader literature that documents that poor childhood conditions are associated with diminished adult health generally. Contrary to prior research, mostly on adults older than our sample, I find no relationship between childhood.

#### **Discussion and Conclusions**

This analysis of early childhood morbidity supports the hypothesis that poor health in childhood is associated with a higher probability of metabolic syndrome in adulthood, a predictor of cardiovascular disease and type 2 diabetes. Respondents who experienced higher prevalence of serious illness and anorexia in childhood also had higher rates of metabolic syndrome and abdominal obesity in early adulthood, even after controlling for confounding factors throughout the life course. Respondents who experienced higher prevalence of infectious and diarrheal diseases in childhood also had higher fasting plasma glucose, a predictor of type 2 diabetes although these results were not statistically significant. Moreover, we find relatively strong effects of early childhood diseases on

metabolic syndrome, especially compared to better studied factors such as birth weight and educational attainment.

Based on previous literature, I expected to find a positive relationship between childhood morbidity and several of the components of metabolic syndrome, mainly fasting plasma glucose and blood pressure. I found a strong positive relationship between childhood morbidity and high fasting plasma glucose and abdominal obesity in young adulthood. However, I found no statistical association between childhood morbidity and blood pressure or cholesterol levels in young adulthood. This null result could be explained by the fact that our sample is composed of young adults under the age of 35. Perhaps this relationship does not emerge until some later point in the life course.

I also hypothesized that the relationship between childhood health and adult health would be partially mediated by adult socioeconomic status and health behaviors. However, the association found between childhood health and morbidity in young adulthood persisted even when we included statistical controls for socioeconomic status in childhood, educational attainment and smoking behavior in adulthood in our models. As these relationships did not change, we find that childhood health exerts an independent influence on metabolic syndrome in adulthood.

In this analysis, I also attempt to validate the use of adult height as a proxy for childhood conditions, since height has often been used to predict chronic morbidity and mortality. I find that it does not capture the effects of childhood diseases on metabolic syndrome in

young adulthood and therefore is not an effective proxy. Thus, studies that include height but no direct measure of childhood morbidity in populations with high prevalence of childhood diseases are likely to underestimate the effects of child health on later life outcomes.

Additionally, as noted in Blackwell et al. (2001), our results suggest that it is important to distinguish between types of childhood diseases if possible. I found that serious illness and anorexia are associated with metabolic syndrome and abdominal obesity in adulthood. Moreover, we found a positive association between infectious diseases and fasting glucose, a key predictor of type 2 diabetes. The overall levels of infectious diseases in early life in this sample are low. This could be because of almost universal breastfeeding which protects children against infections or because of the availability of health care in local centers associated with the nutrition intervention. We did not obtain statistically significant results between childhood infectious diseases and metabolic syndrome, however the magnitude of the resulting coefficients suggests that such a relationship might be found in a larger sample. As various infectious and non-infectious diseases are associated with different adult health outcomes, it would be helpful to differentiate between these whenever possible in prospective studies.

Our analysis has several limitations. First, the size of our sample limits the power of our analysis. This may explain the lack of statistically significant relationship between infectious diseases and adult health, although the coefficients are in the expected direction. Second, it was not clear from our data exactly which diseases were coded as

"serious illnesses". However, we can assume that this category includes some amount of diarrheal, anorexia and infectious diseases of childhood, as these categories are correlated and have similar patterns over time. When comparing our results to those of other studies, "serious illness" should be compared to infectious diseases of childhood generally. Our data were collected as part of a nutrition intervention, which included health services in each of the villages. This is one reason that the level of infectious diseases is much lower than we would imagine. This also partially explains why we have some statistically insignificant results from "infectious diseases" in this analysis.

Sample attrition is a common phenomenon in longitudinal studies. Because respondents lost to follow-up tend to be nonrandomly distributed, attrition may bias means and coefficient estimates in regression models. The results of this study could be affected by attrition in two ways. If the weakest members of the original sample died in childhood, the remaining sample to interview in young adulthood will be slightly more robust than the original sample. Likewise, if the most robust respondents emigrated from Guatemala and were not included in the sample of young adults, then our sample might be slightly less healthy than the original cohort.

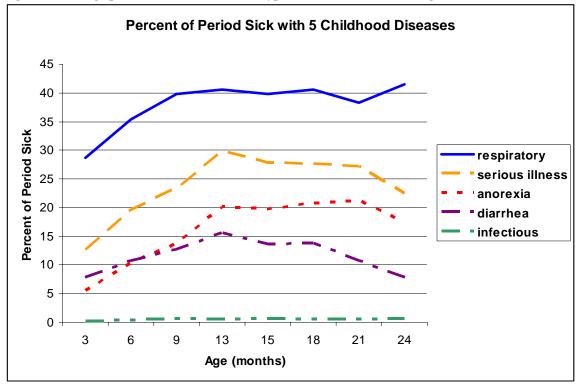
This analysis contributes to the literature in several ways. First and foremost, I use prospective data from a poor developing country with relatively high prevalence of childhood morbidity. Most studies on this topic use data from developed countries, for which the infectious disease burden is lower than in poor developing countries. Our estimates of the relationships between childhood and adult health have high external validity for contexts similar to rural Guatemala. Although the estimates may be less relevant for more affluent contexts, since the proposed mechanisms for this relationship are physiological, our results are useful for rich countries as well. Second, while many studies of aging use adult height or retrospective reports of childhood morbidity to estimate childhood conditions, our data allow us to get more detailed estimates of morbidity throughout the first two years of life. Our data allow us to differentiate childhood morbidity into five categories for the first two years of life, which turns out to be important because we find different relationships between our five childhood diseases and adult health outcomes. Last, there has been relatively low attrition in this sample, allowing us to look at a high proportion of this birth cohort.

The results of this analysis have implications for the distribution of public health resources in developing countries. The relationships that I find between early childhood health and chronic disease in early adulthood imply that investment in programs to reduce the prevalence of childhood illnesses could have considerable long-term benefits for the health of an adult population like Guatemala's some years later. This is especially relevant for populations facing both high rates of infectious diseases in children and increasing rates of the chronic diseases of aging. Developing countries with limited resources have a double incentive to invest in early childhood health as it improves the immediate health of the young population as well as preventing the development of chronic diseases. If public health programs adopt a life course perspective, then investment in child health programs is a sound policy for the young of today, and the costs of the growing proportion of elderly are reduced (Preston 1984).

	0-2	3-5	6-8	9-11	12-14	15-17	18-20	21-23
	months							
Serious	12.7	19.5	23.4	29.8	27.7	27.7	27.1	22.4
illness	(21.0)	(22.7)	(21.7)	(25.0)	(25.1)	(25.8)	(26.1)	(25.0)
Diarrhea	7.8	10.6	12.6	15.5	13.6	13.7	10.7	7.7
	(16.9)	(18.3)	(15.4)	(16.7)	(15.6)	(18.4)	(15.0)	(12.7)
Anorexia	5.4	10.4	13.7	20.1	19.6	20.7	21.2	17.4
	(14.1)	(19.0)	(20.0)	(24.1)	(24.5)	(24.4)	(25.2)	(24.2)
Respiratory	28.6	35.4	39.9	40.5	39.8	40.5	38.2	41.5
- •	(30.6)	(30.1)	(30.8)	(31.0)	(30.8)	(32.4)	(32.6)	(32.7)
Infectious	0.21	0.36	0.63	0.51	0.57	0.52	0.39	0.57
	(2.2)	(2.6)	(3.9)	(3.0)	(3.2)	(3.2)	(2.8)	(3.9)

Table 1: Average Percent of the Period Sick (sd) with Five Types of Childhood Diseases (n=425)

Figure 1: Average percent of time sick with 5 types of childhood diseases, age 0-24 months



	Percent	Ν
	or mean (sd)	
Dependent Variables		
Metabolic Syndrome (%)	35.0	291
High Fasting Plasma Glucose (%)	17.2	326
High Abdominal Obesity (%)	34.9	375
High Blood Pressure (%)	12.5	409
Low HDL (%)	79.4	326
High Triglycerides (%)	53.4	326
Controls		
Age in years	32.2 (1.32)	425
Female (%)	49.2	425
Birth weight (kg)		282
First quintile	1.5 - 2.56	
Second quintile	2.6 - 2.87	
Third quintile	3.0 - 3.1	
Fourth quintile	3.18 - 3.37	
Fifth quintile	3.5 - 4.75	
Exposure to high protein	52.5	425
nutritional supplement		
in first 2 years (%)		
Mother Literate (%)	19.6	392
Years of Education Attained	4.7 (3.2)	408
Smoker (%)	28.0	425
Adult height (cm)	156.6 (8.49)	381

Table 2: Descriptive Statistics of Dependent Variables and Control Variables (n=425). Percents for categorical variables, mean (sd) for continuous variables, minimum and maximum values for birth weight quintiles.

Table 3: Odds Ratios from Neste	d Logistic Reg	ression Models P	Predicting Meta	abolic Syndrome

	Model 1	Model 2	Model 3	Model 4
Serious illness	1.52 *	1.50 *	1.50 *	-
Anorexia	1.58 *	1.54 *	1.55 *	-
Diarrhea	1.17	1.18	1.19	-
Respiratory	0.87	0.85	0.88	-
Infectious	1.12	1.15	1.14	-
<b>Demographic Characteristics</b>	yes	yes	yes	yes
Childhood Conditions	yes	yes	yes	no
Adult Characteristics	no	yes	yes	yes
Adult Height	no	no	1.00	0.99
n	289	287	285	285

Each cell is a different regression \* p<.05 \*\* p<.01

	Model 1	Model 2	Model 3	Model 4
Serious illness	1.45	1.55 *	1.63 *	-
Anorexia	1.33	1.37	1.46	-
Diarrhea	1.38	1.51 *	1.57 *	-
Respiratory	1.10	1.07	1.13	-
Infectious	1.51	1.54	1.28	-
<b>Demographic Characteristics</b>	yes	yes	yes	yes
Childhood Conditions	yes	yes	yes	no
Adult Characteristics	no	yes	yes	yes
Adult Height	no	no	0.99	0.98
n	324	319	294	294

 Table 4: Odds Ratios from Nested Logistic Regression Models Predicting High Fasting Plasma
 Glucose

Each cell is a different regression

\* p<.05 \*\* p<.01

# Table 5: Odds Ratios from Nested Logistic Regression Models Predicting Abdominal Obesity

	Model 1	Model 2	Model 3	Model 4
Serious illness	1.69 *	1.60 *	1.56 *	-
Anorexia	1.63 *	1.54 *	1.51	-
Diarrhea	1.44 *	1.42 *	1.38	-
Respiratory	1.04	0.93	0.93	-
Infectious	0.75	0.80	0.83	-
Demographic Characteristics	yes	yes	Yes	yes
Childhood Conditions	yes	yes	Yes	no
Adult Characteristics	no	yes	Yes	yes
Adult Height	no	no	1.03	1.04
n	373	367	365	365

Each cell is a different regression

\* p<.05 \*\* p<.01

# Table 6: Odds Ratios from Nested Logistic Regression Models Predicting Hypertension

	8	0		e
	Model 1	Model 2	Model 3	Model 4
Serious illness	1.00	0.98	1.00	-
Anorexia	0.98	0.94	0.95	-
Diarrhea	0.98	0.96	0.96	-
Respiratory	0.87	0.83	0.83	-
Infectious	1.14	1.21	1.21	-
<b>Demographic Characteristics</b>	yes	yes	yes	yes
Childhood Conditions	yes	yes	yes	no
Adult Characteristics	no	yes	yes	yes
Adult Height	no	no	1.02	1.01
n	404	393	362	362

Each cell is a different regression

\* p<.05 \*\* p<.01

	Model 1	Model 2	Model 3	Model 4
Serious illness	1.09	1.07	1.00	-
Anorexia	1.38	1.34	1.30	-
Diarrhea	0.87	0.86	0.81	-
Respiratory	1.07	0.98	0.94	-
Infectious	0.79	0.95	0.75	-
<b>Demographic Characteristics</b>	yes	yes	yes	yes
Childhood Conditions	yes	yes	yes	no
Adult Characteristics	no	yes	yes	yes
Adult Height	no	no	1.03	1.04
n	324	319	294	294

Table 7: Odds Ratios from Nested Logistic Regression Models Predicting Low HDL

Each cell is a different regression

\* p<.05 \*\* p<.01

#### Table 8: Odds Ratios from Nested Logistic Regression Models Predicting High Triglycerides

	Model 1	Model 2	Model 3	Model 4
Serious illness	1.09	1.07	1.03	-
Anorexia	1.24	1.23	1.21	-
Diarrhea	0.94	0.92	0.86	-
Respiratory	0.89	0.88	0.90	-
Infectious	1.12	1.18	1.31	-
<b>Demographic Characteristics</b>	yes	yes	yes	yes
Childhood Conditions	yes	yes	yes	no
Adult Characteristics	no	yes	yes	yes
Adult Height	no	no	1.01	1.00
n	324	319	294	294

Each cell is a different regression \* p<.05 \*\* p<.01

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# Appendix Table 1: Summary Statistics for Correlations of Morbidity across Time

	Serious	Diarrhea	Anorexia	Respiratory	Infectious
	Illness				
Mean	.32	.25	.31	.42	.03
Sd	.10	.12	.12	.11	.09
Min	.14	.06	.13	.19	05
Max	.51	.48	.61	.64	.24
IVIAA	.91	.+0	.01	.04	.27

# Appendix Table 2: Correlations of Average Z Scores for Different Illnesses

	Serious	Diarrhea	Anorexia	Respiratory	Infectious
	Illness				
Serious	1.00				
Illness					
Diarrhea	0.70	1.00			
Anorexia	0.87	0.35	1.00		
Respiratory	0.30	0.10	0.34	1.00	
Infectious	0.01	-0.06	-0.01	-0.05	1.00