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Childhood Morbidity and Health in Early Adulthood: Life course linkages in a high morbidity context

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Abstract

This paper examines whether morbidity in early and later childhood is associated with health later in life. I investigate the relationship between five types of childhood morbidity and risk factors for cardiovascular disease among Guatemalan adults who experienced high levels of morbidity in childhood. The analysis is based on the Human Capital Study (2002–2004), a recent follow-up of the INCAP Longitudinal Study conducted between 1969 and 1977. I find that most types of childhood morbidity are associated with poorer adult health, independent of family background, adult socioeconomic status, and health behaviors. Higher levels of infections in childhood were associated with a low level of high density lipoprotein (HDL), and higher level of triglycerides, plasma glucose, waist circumference, and obesity (but not hypertension). These results are consistent with the literature that finds that childhood morbidity is associated with increased morbidity and mortality at older ages. However, diarrheal disease in later childhood was associated with lower levels of some risk factors, as measured by triglycerides and plasma glucose, suggesting that exposure to bacteria after infancy may be beneficial for some measures of adult health.

Keywords

childhood conditions; childhood health; adult health; life course; socioeconomic status; Guatemala

INTRODUCTION

Research on health and mortality has expanded from focusing on adult characteristics such as education, employment, and health behaviors, to a life course approach which examines exposures and patterns throughout the life cycle (Blackwell, Hayward, & Crimmins, 2001; Elo, 2009; Hayward & Gorman, 2004; Palloni, 2006). Recent work has found that conditions early in life are associated with adult socioeconomic position, morbidity, and mortality (Costa, 1993; Elo & Preston, 1992; Haas, 2007; Hayward & Gorman, 2004). The specific causal mechanisms involved are still up for debate (Blackwell et al., 2001), however our understanding of the life course processes benefits from mapping associations in various contexts and populations in order to generate hypotheses (Palloni, 2006).

Childhood conditions have been operationalized as various factors, such as conditions in utero, childhood health, familial socioeconomic status, and nutritional status. In this paper, I focus on childhood health, examining how various types of childhood morbidity are associated with health in young adulthood. Most of the research conducted on early life

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morbidity and adult health outcomes comes from studies conducted in now developed countries (Blackwell et al., 2001; Kuh & Wadsworth, 1993; Palloni, 2006), with more recent evidence emerging from studies about developing countries (Huang & Elo, 2009; Kohler & Soldo, 2005; Zeng, Gu, & Land, 2007). This analysis examines a Guatemalan cohort that experienced high rates of childhood infections and has a high prevalence of chronic diseases in young adulthood.

There are two major models which map how early life conditions are thought to connect to adult health outcomes — the *latency model* and the *pathway model* (Zhang, Gu, & Hayward, 2008; Preston, Hill & Drevenstedt, 1998). The *latency model* suggests that early life circumstances have a direct association with later life outcomes, which can be either positive or negative. Most research has found that childhood disease has a direct negative association with later life health. Poor childhood nutrition, manifested in low birth weight or growth retardation in childhood, has been thought to increase morbidity and mortality from chronic diseases such as cardiovascular disease and diabetes (Barker, 1997; 1998). Childhood infections, such as tuberculosis, hepatitis B, and rheumatic heart disease, may scar survivors, increasing death rates at older ages (Elo & Preston, 1992), while other infections early in life are associated with higher levels of cardiovascular disease, cancer, diabetes, and respiratory diseases (Almond & Mazumder, 2005; Blackwell et al., 2001; Buck & Simpson, 1982; Costa, 2000; Haas, 2008; Hall & Peckham, 1997; Matthews, Whittingham, & Mackay, 1974). One possible biological explanation for this relationship was proposed by Crimmins and Finch (2006). Their cohort morbidity phenotype hypothesis posits that higher levels of infections at younger ages will be positively associated with inflammation and cardiovascular diseases at older ages because early infectious exposures may increase the activation of inflammatory pathways throughout the life course (Finch & Crimmins, 2004). High-sensitivity CRP, a biomarker for inflammation, has since been found to be positively associated with type 2 diabetes (Pradhan et al., 2001), cardiovascular disease (Ridker et al., 1998), metabolic syndrome (Ridker et al., 2003), and mortality (Jenny et al., 2007).

There is some evidence that childhood morbidity may have a direct positive association with health later in life through acquired immunity to specific diseases. Early exposure to infections may be important for guiding the development of the immune defenses. Reduced exposure to microbes in infancy and childhood due to rising standards of sanitation and hygiene have been associated with elevated rates of atopic diseases and other diseases related to immune dysregulation later in life (Illi et al., 2001; McDade et al., 2009; 2001; Rook & Stanford, 1998; Strachan, 1989), higher rates of autoimmune diseases (Paunio et al., 2000) and higher death rates from influenza at older ages (Lee, 1997).

The *pathway model*, on the other hand, sees childhood conditions as indirect influences on adult health through attained socioeconomic or health behaviors (Case, Fertig, & Paxson, 2005; Case, Lubotsky, & Paxson, 2002; Currie & Madrian, 1999; Elo, 2009; Zhang, Gu, & Hayward, 2008). Children ill in childhood may reach adulthood in poorer health, with less education, and fewer marketable skills. Poor health in childhood might also affect adult health outcomes through lifestyle factors, if it increases the propensity to exercise less, smoke more, and eat less healthily (Lynch, Kaplan, & Salonen, 1997; Power, Matthews, & Manor, 1996; Van de Mheen et al., 1998). Thus early life circumstances may influence health outcomes in adulthood both directly and indirectly, mediated through their effect on adult characteristics and health behaviors (Elo, 2009).

The Timing of Exposures

Epidemiologists have argued that the timing of childhood morbidity may be important for how it affects later life outcomes, encouraging researchers to test for critical and sensitive periods during which exposures can have either adverse or protective effects on later life

health (Ben-Shlomo & Kuh, 2002). However, testing hypotheses about the timing of early life exposures on adult health outcomes has been extremely difficult because of the scarcity of rich life course data. Few studies include measures of the timing and severity of diseases in childhood and detailed data on adult health outcomes. Therefore researchers have employed proxies for childhood conditions such as height (Elo & Preston, 1992; Fogel, 1993; Fogel & Costa, 1997; Huang & Elo, 2009; Kohler & Soldo, 2005) or used retrospective reports of childhood illness (Blackwell et al., 2001; Haas, 2007; 2008). However, proxies for childhood conditions and retrospective data do not give us detailed enough information to test theories about critical and sensitive periods, and retrospective data introduce bias into the estimates of the relationship between early life factors and adult health outcomes (Galobardes, Lynch, & Davey Smith, 2004; Kauhanen et al., 2006). In this paper, I am able to distinguish between two periods in childhood, early childhood (age 0–2) and later childhood (age 2–7) — periods thought to be critical or sensitive for later life health (Case et al., 2005; Martorell, 1975; Martorell et al., 1995; McDade et al., 2009)¹.

An unanswered question in the literature is identifying what it is about childhood health that influences adult health outcomes (Palloni, 2006). Most studies do not allow us to test many aspects of childhood health and proxies such as height do not tell us *which* diseases or exposures matter. An additional strength of these data is that they include rich measures of early life morbidity making it possible to distinguish between five types of childhood illnesses: diarrhea, anorexia, fever, serious illnesses (e.g., extreme gastrointestinal problems and nutritional diseases), and infectious diseases (e.g., measles, mumps, rubella, tetanus, hepatitis, and whooping cough).

The Context of Exposures

The direction of the relationship between early life exposures and later life health may differ by context. The majority of research on early life exposures and later life health has been conducted on populations in Western Europe or North America and has found that higher rates of morbidity in childhood are associated with higher rates of morbidity, disability, and mortality in adulthood (Almond & Mazumder, 2005; Blackwell et al., 2001; Buck & Simpson, 1982; Costa, 2000; Haas, 2008; Hall & Peckham, 1997; Matthews et al., 1974). However, recent research conducted in the Philippines, a context with a high prevalence of infectious diseases, has found that higher levels of infections early in life are associated with stronger immune function (McDade et al., 2001) and lower levels of inflammation in early adulthood, independent of important confounders among young adults (McDade et al., 2009). Findings may differ by context because of different prevalence, intensity, or types of infectious diseases, or because of the age at which health outcomes are measured.

Guatemala is an important context in which to examine the relationship between childhood disease and adult health because many other developing countries are undergoing similarly rapid changes in their disease burden. From the time when the examined birth cohort was young in the 1970s to young adults in their 30s, life expectancy in Guatemala increased from 52 to 70 years (UNICEF, 2010). The mortality reduction, driven by the decrease in infectious diseases, was followed by an increase in degenerative and man-made diseases, in which increased fat and caloric intake, widespread tobacco use, and chronic disease deaths exceed mortality from infectious diseases and malnutrition (Gaziano, 2005). Similar changes are occurring in countries where more than seventy percent of the world's population lives (Gaziano, 2005). If morbidity in childhood has a causal impact on health later in life, then public health initiatives targeting childhood will have additional payoffs when the affected cohorts are healthier adults with lower rates of chronic diseases which are costly to treat.

¹The correlations between types of childhood illnesses in early and later childhood are available in Table 8.

THE PRESENT STUDY

In this paper, I investigate associations between five types of childhood morbidity and adult health outcomes at ages 29 to 34 using the INCAP Longitudinal Study (1969–1977) and Human Capital Study (2002–2004). The data allow for four improvements to prior research. First, unlike most research which has focused on adult health in middle-aged or older adults, I investigate health in early adulthood. Unlike the relatively healthy young adults in low mortality populations, the young adults in this sample exhibit risk factors for cardiovascular disease. Another strength of these data is that adult health outcomes are measured with biomarkers and clinical measurements rather than self-reports which improve accuracy and can lead to a clearer understanding of the pathways through which morbidity develops (Crimmins & Seeman, 2001). I also test for the critical and sensitive periods of early and later childhood and distinguish between five types of childhood illness. Last, the rich life course data allow me to control for many factors that often go unobserved, such as family background.

Based on the literature on life course health, I developed the following hypotheses. First, childhood morbidity would be associated with increased odds of exhibiting risk factors for cardiovascular disease in young adulthood: elevated glucose, dislipidaemia, hypertension, and obesity. Second, the association between childhood morbidity and adult health would be partially mediated by adult socioeconomic status (SES) and lifestyle factors.

METHODS

Data

As noted above, the analyses are based on the Institute of Nutrition of Central America and Panama (INCAP) Longitudinal Study conducted 1969–1977 and the 2002–2004 Human Capital Study. This sample consists of individuals exposed to a randomized community nutrition supplementation trial which was carried out in four villages in Eastern Guatemala between 1969 and 1977². The trial was designed to assess the effect of improved protein intake in early life on the growth and cognitive development of children. The “treatment” drink, called *atole*, was a type of hot gruel, high in protein. The “control” drink, called *fresco*, was devoid of protein but included vitamins, iron, and fluoride. Each drink was assigned to two villages and was available in those villages twice a day. Medical clinics were set up in each of the four villages offering free preventative and curative services that were unrelated to participation in the study (Martorell et al., 2005; Martorell, Habicht, & Rivera, 1995). Detailed information on childhood morbidity was collected from July 1970 to February 1977. In 2002–2004, a multidisciplinary team representing Emory University, the University of Pennsylvania, the International Food Policy Research Institute (IFPRI), and the Institute of Nutritional of Central America and Panama (INCAP) led the Human Capital Study, did a follow-up, to examine the effect of the intervention on adult wages and health. The cohort of individuals who were young children or born during the original study were between ages 26 and 42 in 2003.

In this analysis, I focus on a subsample of respondents born in the early part of the study (1969–1974). Table 1 presents the demographic and childhood characteristics of children born in the study villages and the subsamples for whom childhood and adult morbidity data were collected. Of the 1,125 children born between 1969 and 1974, I focus on those with substantial childhood morbidity data available for early and later childhood. Respondents were included if morbidity data was collected during at least one year of their early

²Ingesting the supplement was not necessary to take part in the intervention.

childhood (ages 0–2) and one and a half years of later childhood (ages 2–7). For this subsample of 558 respondents, 50% of the original cohort, morbidity data was collected for on average 86% of early childhood and 59% of later childhood until respondents were censored when data collection ended in 1977. This subsample is shown in the second column and has similar demographic characteristics as the original cohort, but come from more disadvantaged families with lower SES and less literate mothers.

Of the respondents for whom we have childhood morbidity histories, 458 (82%) were re-interviewed in 2002–04. The respondents who were not re-interviewed in the Human Capital Study either died (4.3%), were lost to follow up (0.5%), or were not interviewed (13.1%).³ Shown in the third column, this is the possible subsample from which the analytic subsamples are taken. Respondents that were re-interviewed are similar on demographic characteristics but more disadvantaged than the original cohort as shown by lower family SES and maternal literacy. Of the sample that was re-interviewed, 339 (74%) provided a full blood test which provides information on a range of biomarkers and 364 (79%) respondents completed blood pressure and anthropometric measurements. These data comprise the two analytic subsamples used in the analysis depending on the outcome of interest, and are shown in the last two columns in Table 1. The analytic samples have substantial overlap and as shown in Tables 1 and 2 are similar with respect to all variables of interest.

Several recent studies have examined the effects of the 1969–77 intervention on the adult outcomes of the cohort born during the study. Those exposed to the protein supplement were found to have higher economic productivity (Hoddinott et al., 2008), higher educational attainment and cognitive skills (Maluccio et al., 2009), better reading comprehension (Stein et al., 2008), and even improved anthropometric outcomes for their children (Behrman et al., 2009). Some prior research has assessed the effect of the nutrition intervention on health outcomes. Most of this research has found small, if any, differences in adult health between the groups that received *atole*, the “treatment”, and *fresco*, the “control”. For example, Stein et al., (2002; 2006) documented few significant associations between the nutrition supplement and cardiovascular risk factors in early adulthood. Others found slightly lower fasting glucose levels among those born in villages exposed to *atole* (Conlisk et al., 2004) compared to those exposed to *fresco*, but no difference in blood pressure between the two groups (Webb et al., 2005). Adult characteristics, such as occupation and place of residence, were more strongly associated with the cardiovascular disease risk factors than the type of early life supplementation (Stein et al., 2002; Torun et al., 2002). To the author’s knowledge, no study has analyzed the associations between childhood morbidity and adult health with these data.

Young Adult Health

My dependent variables consist of six clinically-measured aspects of adult health which are risk factors for cardiovascular disease. Plasma glucose and two measures of dislipidaemia are based on a blood sample obtained by finger prick after an overnight fast. The level of fasting plasma glucose measures the health of the metabolic system, with elevated glucose levels being associated with heart disease risk (Reaven, 1988), higher mortality (Fried et al., 1998), and poorer cognitive function (Craft et al., 1993; Gradman et al., 1993; Manning, Hall, & Gold, 1990). A low level of high-density lipoprotein (HDL), often called “good cholesterol”, is associated with increased risk of heart disease (Wilson et al., 1998). An elevated level of triglycerides is another component of dislipidaemia. The level of triglycerides is usually inversely correlated with HDL, and high levels of triglycerides in the

³A full description of the tracking of individuals and data collection for the 2002–04 Human Capital Study can be found in Grajeda et al. (2005).

bloodstream are associated with atherosclerosis, heart disease, and stroke. I analyze both the absolute level of plasma glucose, HDL, and triglycerides as well as the risk of being above established cut points for elevated plasma glucose (≥ 100 mg/dl or drug treatment for elevated glucose), elevated triglycerides ($\text{TG} \geq 150$ mg/dl), and low HDL ($\text{HDL-C} < 40$ mg/dL for males, < 50 mg/dL for females) (Grundy et al., 2005).

Hypertension, an indicator of the health of the cardiovascular system, is my fourth measure of adult health. Three measurements of systolic and diastolic blood pressure were taken at three to five minute intervals and I use the mean level of these three measurements. Participants were asked to refrain from tobacco, alcohol, or caffeine use during 30 minutes preceding measurement. I analyze the risk of having hypertension, defined as having blood pressure $\geq 130/85$ mmHg (Grundy et al., 2005) or taking blood pressure medication.

The last two adult health measures are anthropometric- waist circumference and obesity. Waist circumference was measured to the nearest 0.1 cm at the umbilicus and I use the mean of two consecutive measurements. I analyze the risk of abdominal adiposity, for which the clinical cut-point is defined as waist circumference ≥ 102 cm for males and ≥ 88 cm for females (Grundy et al., 2005). I categorize BMI based on guidelines set forth by the NIH (National Heart, Lung, and Blood Institute, 1998) and the World Health Organization (2000), with obesity defined as $\text{BMI} \geq 30$. I use the mean of two height and weight measurements and calculate BMI as weight (kg) divided by height (m) squared.⁴

Childhood Morbidity

Data on childhood morbidity were collected from July 1970 to February 1977. Mothers or primary caregivers were interviewed every two weeks about the health of their children. They were asked to recall any symptoms their children younger than age seven had had during the two-week period prior to interview. During the study, interviewers collected data on 44 distinct symptoms and coded these into various types of illnesses⁵.

The mother-reported morbidity measures were evaluated in two ways. First, researchers compared the levels of illness reported on the day of interview to the days during the two weeks before the interview and found that fewer days of illness were reported earlier in the period. Martorell et al. (1975) interpreted this pattern as underreporting due to the mother's memory loss and estimated a 22% underreporting rate for diarrhea and 37% for fever. The data were evaluated a second time when in a sub-study mother's reports on the day of interview were compared with independent assessments by a physician. This comparison found that 66% of the children whom the physician reported having diarrhea were also reported by mothers to be ill with diarrhea and 99% of the children whom the physician found without diarrhea were also reported free of diarrhea by the mother. Thus, there may be some underreporting of diarrhea (see Martorell et al., 1975; 1976 for more detail)⁶.

Based on prior literature (Blackwell et al., 2001; Elo & Preston, 1992; McDade et al., 2001; 2009) and data availability, I have chosen five types of childhood morbidity that have been hypothesized to predict adult health outcomes: diarrhea, anorexia, fever, infectious diseases, and serious illnesses⁷. Diarrhea and fever were defined by the mother. Anorexia refers to the

⁴See Ramirez-Zea et al. (2005) for more detail on the data collection of adult health measures.

⁵Respondents in the analytic samples exposed to *atole* experienced significantly fewer days with serious illness and anorexia in early and later childhood, and infectious diseases in later childhood than those exposed to *fresco*. There were however, no significant differences in the time spent with diarrhea or fever in either early or later childhood or with infectious diseases in early childhood (results not shown).

⁶The physician reported data are not available to this researcher.

⁷These five disease categories were constructed by the data team and the raw data for each specific type of illness are no longer available.

marked reduction of appetite and/or decrease in the amount of food eaten at home. Infectious childhood diseases include measles, mumps, rubella, chickenpox, tetanus, hepatitis, and whooping cough. Serious illnesses refer to extreme gastrointestinal problems such as mucus or blood in stool, nutritional diseases, extreme vomiting, inflammation of the eyes, nose and ears, and serious wounds.

I capture the intensity and recurrence of disease in childhood with two types of measures. The intensity of disease in *early* childhood is coded as the number of days that respondents were ill with each type of disease from 15 days to two years of age. Morbidity in *later* childhood is coded as the number of days respondents were reported to have been ill from age two to seven. Illness in these two periods is coded as number of days rather than a rate in order to easily compare the relative strength of associations between childhood health and the adult health outcome, given that the periods are different lengths⁸. To adjust for the fact that children had varying number of days reported on, all analyses control for the number of days for which morbidity is reported in the relevant time periods. The recurrence of disease is measured as the total number of years during which respondents experienced each illness at least once, ranging from zero to seven.

Family Background and Adult Characteristics

I adjust for three sets of independent variables: sociodemographic, family background, and adult characteristics. Sociodemographic variables include age, sex, and the village of origin. Family background is measured by family SES and maternal literacy. Family socioeconomic status is coded into tertiles: low, middle, and high. It is based on a household wealth scale computed for all families in the four villages, and is based on household consumer durable goods such as refrigerators and radios, and housing characteristics such as toilets/latrines, and quality of roof, floors, and walls (Maluccio, Murphy, & Yount, 2005). Maternal literacy is coded as literate, partially literate, and illiterate.

I measure adult SES and risk factors that have been hypothesized to influence adult health outcomes (Bjartvelt & Tverdal, 2005; Britton & McKee, 2000; Elo 2009; Preston & Taubman, 1994; Rimm et al., 1993). Adult SES is measured with educational attainment coded as: no education, lower primary school (1–3 years), upper primary school (4–6 years), and seven or more years. Marital status is measured as married in a formal or informal union, never married, and widowed or divorced.

Behavioral factors include cigarette smoking and alcohol consumption. I construct a three-category variable for smoking to capture past and present smoking behavior: never smoker, former smoker, and current smoker. Alcohol consumption is coded as whether the respondent drinks alcohol or not at all.

Analytical Approach

To examine the relationship between childhood morbidity and adult health, I perform a series of maximum likelihood regression models to examine whether the childhood morbidity measures independently predict each of the outcome variables: dislipidaemia (HDL and triglycerides), high plasma glucose, hypertension, high waist circumference, and obesity. I perform nested models to test whether there is an association between each type of childhood morbidity and adult health outcomes and then whether it is decreased by family background and adult characteristics. The first equation models adult health as a function of each type of childhood morbidity which is significantly associated with the adult health outcome⁹, controlling for age, sex, and the village of origin which controls for village fixed

⁸Results are similar if a variable for percentage of the period experiencing illness is used.

effects and the type of supplement received. The second model adds family background and the third adds adult characteristics and health behaviors. In the first three models each type of childhood morbidity is included a separate model to examine its independent association with each outcome. The fourth model includes *all* measures of childhood morbidity together with family background and adult characteristics to examine whether they predict adult health outcomes independently of each other.

I use binary logistic regression for each dichotomous outcome or risk factor and OLS regressions to predict the level of HDL, triglycerides, and plasma glucose which are also important and sensitive indicators of the metabolic and cardiovascular system¹⁰. To adjust for the non-independence of siblings in the data set, all models are estimated using robust standard errors¹¹. To control for all fixed characteristics of the four villages that might affect health, village fixed effects are represented by dummy variables for three of the four villages (Hoddinott et al. 2008). All models also control for the number of days for which childhood morbidity was reported, from ages zero to two and from ages two to seven.

The two analytic subsamples include respondents for which childhood morbidity histories and adult health outcome data are available. Of the 410 respondents in the two subsamples, less than one percent is missing data on maternal literacy and educational attainment, two percent for educational attainment, and six percent for adult marital status and health behaviors (Shown in Table 1). In order to retain the sample size, I use multiple imputation to impute missing values for these missing control variables, assuming these data are missing at random, conditional on the observed covariates in the imputation model (Allison 2001;Royston 2004).

RESULTS

Table 2 presents descriptive adult characteristics for the members of the birth cohort with childhood morbidity histories who were followed up in the Human Capital Study, and the two analytic subsamples. At the time of the 2002–2004 survey, the respondents were young adults, ages 29 to 34 with a mean age of 31.5 (sd 1.4). Both the respondents and their mothers had low levels of education. Only one third of the mothers were literate and one in five was partially literate. Although just over 10% of the respondents had no formal education, a quarter had gone to lower primary school, and most (60%) had more than four years of formal schooling. Most respondents (80%) were married or partnered and the vast majority (87%) of the women had begun childbearing. Many respondents also reported having used cigarettes or alcohol. Fewer than one in five reported being a current smoker with one in ten reported being a former smoker. About 40% of the respondents reported that they currently drank alcohol.

Table 3 presents descriptive statistics for the outcome variables. Compared to young adults in the U.S., this Guatemalan cohort has much higher levels of dislipidaemia and elevated plasma glucose, similar levels of hypertension, and slightly lower proportions overweight and obese (National Center for Health Statistics, 2009;Ogden et al., 2006). Dislipidaemia is very common. Close to 80% of respondents have low HDL and half have high triglycerides. However, high plasma glucose and hypertension are less common. Eighteen percent have a fasting plasma glucose level of 100 or higher and only 13% have hypertension. Many

⁹I tested whether each outcome was significantly associated with each of the five types of childhood morbidity- in early childhood, later childhood, and the recurrence of the illness.

¹⁰The results are similar when excluding extreme outliers for the days experiencing illness.

¹¹The results are extremely similar when calculating standard errors in two alternate ways: 1- The Huber-White method which allows for heteroscedasticity of unknown form, but not for clustering and 2- Allowing for correlations within village-birth year cohorts.

respondents are overweight, with close to 50% being either overweight or obese. More than a third have a high waist circumference.

Table 4 presents sample characteristics for childhood morbidity. Diarrhea, anorexia, and serious illnesses were very common for this cohort when they were young. Between ages 0–2, half of the children spent 55 days or more with diarrhea, 39 days with anorexia (lack of appetite) and 37 days with serious illnesses (gastrointestinal problems, nutritional diseases, or extreme inflammation). Although the amount of time spent with diarrhea decreased in later childhood, it was similar for time spent with anorexia and increased for serious illnesses. Moreover, the vast majority of children experienced these illnesses at some point in early or later childhood. Only 5% did not experience diarrhea or anorexia at all or did so in only one year during childhood, while the vast majority experienced these symptoms in two or more years. About 10% were chronically sick with diarrhea, anorexia, and serious illnesses, experiencing these symptoms in six or seven years between birth and age seven. Fever and infectious diseases were somewhat less prevalent. The median number of days spent with fever was 16 for ages 0–2 and 14 for ages 2–7. Almost half of children also experienced one of the infectious diseases in early or later childhood. The levels of illness for later childhood and the recurrence of disease reported in Table 4 are underestimated, as the data are censored for most respondents. The bottom of the table shows that data for available for most (86%) of early childhood, but only 59% of later childhood.

Tables 5, 6, and 7 present results from nested multivariate logistic and OLS regression models predicting the young adult health outcomes. First I examine models predicting low high-density lipoprotein (HDL) and the level of HDL, shown in Tables 5 and 6. Higher levels of childhood morbidity are associated with a higher risk of dyslipidaemia, measured by low HDL and a lower level of HDL. A higher number of days with diarrhea in early childhood and the recurrence of serious illness exhibit a significant association with a higher risk of low HDL, with and without controls for family background and adult characteristics. Similarly, four types of illness are significant predictors of a lower *level* of HDL — diarrhea, fever, serious illness, and anorexia. When all childhood morbidities are controlled for simultaneously in Model 4, none are statistically significant due at least in part to the correlation between childhood health measures, which range from 0.09 to 0.59. However, if we include only the strongest predictors of each disease type then diarrhea remains a significant predictor of the level of HDL, (results not shown). Higher levels of childhood morbidity in both early and later childhood predict a higher risk of low HDL and a lower level of HDL. Diarrhea in early childhood and the recurrence of serious illness predict both measures of HDL, while many childhood morbidity measures predicted the level of HDL. The recurrence of disease were strongest predictors for both low HDL and the level of HDL.

Next I turn to the risk of high triglycerides and elevated glucose and the levels of these biomarkers, also shown in Tables 5 and 6. Most childhood illnesses that were associated with triglycerides were associated with a higher level of morbidity in young adulthood. Fever in later childhood is positively associated with high triglycerides, as well as the level. The number of years experiencing infectious diseases was also positively associated with high triglycerides while the years experiencing serious illness was positively associated with a higher level of triglycerides in adulthood. Similarly, experiencing infectious diseases in childhood was positively associated with high triglycerides and a higher level of plasma glucose. However, in contrast to most studies, higher levels of diarrhea in later childhood are associated with *better* adult health outcomes for high triglycerides, the level of triglycerides, and the level of plasma glucose. Moreover, the negative relationship between diarrhea in later childhood and triglycerides and glucose is independent of the relationship between other types of early life morbidity and adult outcomes.

I now turn to the results for hypertension, abdominal obesity, and obesity in Table 7. Of all health outcomes examined, childhood morbidity was least strongly associated with hypertension. Serious illness in early and later childhood has a significant but very small positive association with higher risk of hypertension.

The last two outcomes of interest are anthropometric- waist circumference and obesity. The number of years that children experienced serious illness has a strong positive association with both the risks of high waist circumference and of obesity. Several other types of childhood morbidity also predicted abdominal obesity such as diarrhea and anorexia in early childhood, the recurrence of diarrhea, and serious illness in later childhood.

DISCUSSION

In this paper, I investigate the relationship between five types of childhood morbidity and risk factors for cardiovascular disease among young Guatemalan adults who experienced high levels of morbidity early in life. I test the *latency* and *pathway models*, two life course models through which early life conditions may be associated with later life health (Zhang et al., 2008; Preston et al., 1998). Most results give some support to the *latency model* which predicts that most types of childhood morbidity are directly associated with later life health (either positively or negatively), as controls for adult characteristics did little to change the size of the associations. Most types of childhood morbidity were associated with higher morbidity in adulthood, independent of adult socioeconomic status and health behaviors. Higher levels of infections in childhood were associated with higher risk of dislipidaemia, plasma glucose, waist circumference, and obesity (all outcomes except hypertension). All of the five types of infections were related to negative adult health outcomes. These results are consistent with the majority of the literature that finds that illness in childhood, especially early childhood, scars the body, increasing morbidity and mortality at older ages (Blackwell et al., 2001; Crimmins & Finch, 2006; Elo & Preston, 1992; Hall & Peckham, 1997). Moreover, the negative associations between childhood morbidity and adult health are not insignificant in size, similar to those between education or socioeconomic status and adult health, which have gotten much more attention in the literature (Palloni, 2006). They are also similar to the size of the associations found in another study of young adults in the Philippines (McDade et al., 2001), but smaller than those found in a study of adults ages 55–65 in the U.S. (Blackwell et al., 2001).

However, diarrheal disease in later childhood was associated with better health in adulthood, as measured by dislipidaemia and the level of fasting plasma glucose. To the best of my knowledge, this is the first paper that has shown this association. It can be interpreted in the context of two recent studies conducted on young adults in the Philippines, another high morbidity context, that found that exposure to infectious microbes in infancy was associated with improved immune response and lower levels of inflammation in young adulthood (McDade et al., 2009; 2001). They interpret the results in light of the hygiene hypothesis, which posits that exposure to bacteria help to strengthen the immune system. While triglycerides and fasting plasma glucose are not inflammatory conditions per se, the immune system has been linked to cardiovascular disease, which we previous did not link to immune function (Crimmins & Finch, 2006; Hannson & Libby, 2006). Moreover, a recent trial conducted by Ridker et al. (2008) found that rates of a first major cardiovascular event and death from any cause were significantly reduced by a drug that lowers inflammation among healthy people with normal lipid levels. Treating inflammation led to decreased levels of triglycerides and LDL cholesterol, suggesting that cardiovascular events and risk factors are in some respects disorders of the immune system (Hannson & Libby, 2006; Ridker et al., 2008).

The association between diarrheal disease in later childhood and lower morbidity in adulthood differs from much of the literature cited earlier from now developed countries. This may be because there are differences between the disease burden of North America and Western Europe in the 19th and early 20th centuries and Guatemala and the Philippines in the 1970s and 1980s. It also may be that while childhood diarrheal disease is associated with lower morbidity in young adulthood, this relationship could change as these individuals age. More research is needed to understand the mechanisms through which childhood morbidity affects later life health in both positive and negative ways.

This analysis speaks to several methodological weaknesses in prior literature on the relationship between early life exposures and later life health outcomes. First, with the availability of well-documented morbidity patterns, family background, and adult socioeconomic status and lifestyle factors, I am able to test two models for how early life conditions are associated with later life outcomes. The associations between childhood morbidity and adult health were not decreased much when controlling for adult factors. However, it could be that adult factors had counteracting effects on the outcome of interest, such as educational attainment and health behaviors. The analysis is not causal, as there may be unmeasured factors influencing health and possible feedback effects with health behaviors. However, the extensive relationships presented here should be explored in future research.

The paper also uses more detailed longitudinal data for childhood health than is usually available. I distinguish between five types of illness which are hypothesized to affect later life health — diarrhea, anorexia, serious illnesses, infectious diseases, and fever. I tested the timing of exposures, distinguishing between early childhood (ages 0–2) and later childhood (ages 2–7) in order to capture critical or sensitive periods as well as the recurrence of disease over both these periods. The intensity of disease in early childhood *and* the recurrence of disease throughout early and later childhood were stronger predictors of adult morbidity than the intensity of disease in later childhood. However, diarrheal disease in later childhood was associated with better health for triglycerides and plasma glucose. Overall, if the relationships are found to have a causal impact, then public health initiatives targeting first early childhood but *also* throughout later childhood would make the most impact on later life health.

Because researchers do not often have access to detailed morbidity data during such a long period, I present the correlations between the different types and measures of early life morbidity in Table 8. Despite the fact that these conditions might be related to each other, the correlations between symptoms and conditions in the same period and across periods are modest. Future research should attempt to understand the mechanisms through which these conditions may affect the body's development.

Measuring adult health outcomes with clinical measures and biomarkers is another strength of the paper. This population, ages 29–34, already exhibits risk factors for cardiovascular disease. The biomarkers can help us to understand the pathways through which early exposures affect how the body ages (Crimmins & Seeman, 2001). Future work should investigate if the associations found between childhood morbidity and adult health are the same direction and strength at older ages and across populations.

The study has several limitations. The analysis is limited to a relatively small sample, due to the range of years during which morbidity data was collected for children. However, it is comparable to other studies, such as that by Blackwell et al. (2001), which draws on a sample of 654 that experienced a much lower prevalence of childhood infections. This sample, although smaller, includes much higher rates of childhood morbidity and variation

in the timing and intensity of infectious diseases. However, because of the size, running sex-specific models was not possible. Some recent work has found that there are sex differences in the levels of associations between early and later life factors (Zhang et al. 2008) which should be explored in future work.

The design of the study does now allow this analysis to take advantage of the semi-experimental design. While the “treatment” and “control” groups had access to different nutrition supplements, all villages had access to vaccination, preventive, and curative services which were staffed by paramedical personnel under the supervision of a physician. Services were free and unrelated to participation in the study. While there were no significant differences between the treatment and control groups for most childhood infections, we cannot reject the hypothesis that the experiment altered the levels of morbidity in all the villages. I expect that if the level of morbidity was in fact reduced, then without the intervention we may have seen even larger associations between morbidity in early years and later life.

This analysis, conducted on two subsamples of the original cohort (32% and 30%), was subject to selection in the collection of childhood morbidity data, from mortality and survey attrition, and in the collection of adult health information. Most of the selection occurred at the period of collection of childhood morbidity data. Children for whom we had substantial morbidity data were similar on demographic characteristics from the original sample, but were from more disadvantaged families with lower SES and less literate mothers. Similarly, those re-interviewed who completed adult health information came from more disadvantaged families and included a higher percentage of women than the original birth cohort. If these respondents had a higher rate of morbidity than those who did not participate to the same degree, then the sample may have overestimated the rate of morbidity for this original cohort. Moreover, we can say little about how the direction and strength of the associations found in these data are affected by the selectivity of the sample.

Despite limitations, this analysis provides a comprehensive look at the relationship between childhood health and young adult health for a population that experienced high levels of morbidity in early life. The results are associations, not causal effects, and should be interpreted with caution. If however, future research maps specific causal mechanisms, then policies targeting infectious diseases and malnutrition in early and later childhood could have considerable benefits for the health of populations in developing countries. These results are especially pertinent as many countries go through the epidemiological transition rapidly and are burdened by both infectious diseases and increasingly, chronic diseases which are costly to treat. However, the finding that diarrhea in later childhood was associated with lower morbidity in adulthood suggests that less dangerous infections after infancy may be helpful for guiding the development of the immune system. This article provides further evidence that health is linked throughout the life course, however in various ways that may differ by context and period of development.

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Table 1

Demographic and Childhood Characteristics: 1969–74 Birth Cohort, Childhood Morbidity Subsample, Human Capital 2002–2004 Follow-Up Sample, and Two Analytic Subsamples

Variable	Original Sample Born 1969–74 (N=1,125)	Subsample with Substantive Childhood Morbidity Data (N=558)	Subsample with Childhood Morbidity Data and 2002–04 Follow Up (N=458)	Analytic Subsample 1 (N=339)	Analytic Subsample 2 (N=364)
<u>Demographic Characteristics</u>					
Male	53.4	53.6	51.3	43.9 **	48.9
Age (2002–04)	31.2 (1.7)	31.5 (1.4)	31.5 (1.4)	31.5 (1.4)	31.6 (1.4)
<u>Childhood Circumstances</u>					
Exposure to <i>atole</i>	52.9	51.1	50.9	53.4	53.6
Family Socioeconomic Status			*	*	*
Low	33.6	37.5	40.4	41.6	41.8
Medium	33.1	29.0	28.4	27.1	28.6
High	33.3	33.5	31.2	31.3	29.7
Maternal Literacy		**	**	**	**
Illiterate	40.7	45.0	45.2	44.2	44.5
Partially literate	18.4	22.0	22.0	21.5	21.7
Literate	31.8	31.7	32.1	33.9	33.0
Missing	9.1	1.2	0.7	0.3	0.8

^aStars indicate significant differences ($P < .05$) from the Original Sample, with two tailed T-test (mean) or Chi-2 for categorical variables.

Table 2

Adult Sample Characteristics: Human Capital 2002–2004 Sample and Two Analytic Subsamples

Variable	Subsample with Childhood Morbidity Data and Follow Up (2002–04) (N=458)	Analytic Subsample 1 (N=339)	Analytic Subsample 2 (N=364)
<u>Demographic Characteristics</u>			
Male	51.3	43.9 **	48.9
Age (2002–04)	31.5 (1.4)	31.5 (1.4)	31.6 (1.4)
<u>Adult Socioeconomic Status</u>			
Educational Attainment			
None	12.0	11.2	13.7
Lower primary (1–3 years)	24.4	24.5	24.7
Upper primary (4–6 years)	42.8	44.8	43.4
Basic (7 or more years)	15.9	17.7	16.5
Missing	4.8	1.8	1.6
Marital Status			
		*	**
Married/Partnered	71.4	77.9	78.8
Single	12.9	13.0	14.8
Separated/Widowed	5.7	5.0	6.0
Missing	10.0	4.1	0.3
<u>Health Behaviors</u>			
Smoking			
		**	**
Never smoker	61.8	72.3	66.2
Former smoker	9.6	10.6	10.4
Current smoker	16.8	15.3	17.6
Missing	11.8	1.8	5.8
Drinking			
		**	**
Does not drink alcohol	47.6	55.2	51.1
Drinks alcohol	40.4	42.8	42.9
Missing	12.0	2.1	6.0

^aStars indicate significant differences ($P < .05$) from the Follow-Up Subsample, with two tailed T-test (mean) or Chi-2 for categorical variables.

Table 3

Sample Characteristics: Adult Health Outcomes, Human Capital Subsample and Two Analytic Subsamples

	Subsample with Childhood Morbidity Data and 2002-04 Follow Up (N=458)	Analytic Subsample 1 (N=339)	Analytic Subsample 2 (N=364)	T-test or Chi2 ^a p-value	
				Sample 1 vs. Follow-Up Sample	Sample 2 vs. Follow-Up Sample
High-density lipoprotein ^b					
Mean (sd)	37.3 (11.0)	37.3 (11.0)	NA	1.00	NA
>40 (men), >50 (women) (%)	15.5	20.9		0.00	
≤40 (men), ≤50 (women) (%)	58.5	79.1			
Missing (%)	26.0	0.0			
Triglycerides ^c					
Mean (sd)	176.2 (94.4)	176.2 (94.4)	NA	1.00	NA
<150 (%)	34.9	47.2		0.00	
≥150 (%)	39.1	52.8			
Missing (%)	26.0	0.0			
Plasma Glucose ^d					
Mean (sd)	92.4 (16.1)	92.4 (16.1)	NA	1.00	NA
<100 (%)	60.7	82.0		0.00	
≥100 (%)	13.3	18.0			
Missing (%)	26.0	0.0			
Blood Pressure ^e					
Mean systolic (sd)	112.3 (12.4)	NA	112.3 (12.3)	NA	0.97
Mean diastolic (sd)	71.0 (9.6)		71.0 (9.6)		0.95
No Hypertension (%)	79.7		87.4		0.00
Hypertension (%)	11.6		12.6		
Missing (%)	8.7		0.0		
Waist Circumference ^f					
Mean (sd) men	85.9 (8.8)	NA	85.9 (9.0)	NA	0.97
Mean (sd) women	92.3 (11.3)		92.3 (11.3)		0.99
<88 cm (women), <102 cm (men) (%)	53.1		64.8		0.00
≥88 cm women, ≥102 cm (men) (%)	28.4		35.2		

	Subsample with Childhood Morbidity Data and 2002-04 Follow Up (N=458)	Analytic Subsample 1 (N=339)	Analytic Subsample 2 (N=364)	T-test or Chi2 ^a p-value	
				Sample 1 vs. Follow-Up Sample	Sample 2 vs. Follow-Up Sample
Missing (%)	18.6		0.0		
Body Mass Index ^g					
Mean (sd)	25.5 (4.3)		25.6 (4.3)		0.84
Underweight	1.5	NA	1.9	NA	0.00
Normal (%)	41.7		48.1		
Overweight (%)	29.0		34.6		
Obese (%)	12.4		15.4		
Missing (%)	15.3		0.0		

^aTwo tailed T-test (mean) or Chi-2 for categorical variables (P-values indicate statistically significant difference from Follow-up sample)

^bRisk level for low HDL-C is < 40 mg/dL for males, <50 mg/dL for females (Grundy et al., 2005).

^cRisk level for elevated triglycerides ≥ 150 mg/dl (Grundy et al., 2005)

^dRisk level for elevated plasma glucose ≥ 100 mg/dl or drug treatment for elevated glucose (Grundy et al., 2005).

^eHypertension defined as blood pressure ≥ 130/85 mmHg (Grundy et al., 2005) or taking blood pressure medication.

^fRisk level for abdominal adiposity: waist circumference ≥ 102 cm males and ≥88 cm females (Grundy et al., 2005).

^gUnderweight (BMI<18.5), normal (BMI = 18.5-24.9), overweight (BMI = 25.0-29.9), and obese (BMI ≥30) (WHO 2000).

Table 4Sample Characteristics: Childhood Morbidity (N=410)^a

Childhood Morbidity	Median days ill (25th and 75th percentile) Ages 0–2	Median days ill (25th and 75th percentile) Ages 2–7	Respondents experienced illness at least once	
			# of Years (Age 0–7)	% of Respondents
Diarrhea	55 (27–87)	15 (4–38)	0–1	5
			2–3	45
			4–5	42
			6–7	8
Anorexia ^b			0–1	5
			2–3	35
			4–5	48
			6–7	12
Fever	16 (9–27)	14 (7–26)	0–1	5
			2–3	28
			4–5	54
			6–7	13
Serious illnesses ^c	37 (13–100)	51 (15–131)	0–1	6
			2–3	32
			4–5	50
			6–7	12
Infectious childhood diseases ^d			0	56
			1	35
Percent with any infection	25%	24%	2	8
			3	0.5
			4	0.5
Mean % of each period with childhood morbidity reports	86%	59%	NA	NA

Notes

The morbidity data presented are substantially underestimated for later childhood and the recurrence of disease as only 59% of the later childhood period was available.

^a Analytic sample (N=410) includes respondents in both analytic samples because there are no significant differences in childhood morbidity between the two samples.

^b The marked reduction of appetite and/or decrease in the amount of food eaten at home.

^c Extreme gastrointestinal problems, nutritional diseases, extreme vomiting, inflammation of the eyes, nose and ears, and wounds from accidents.

^d Measles, mumps, rubella, chickenpox, tetanus, hepatitis, and whooping cough.

Table 5

Odds Ratios Predicting Low HDL, High Triglycerides, High Plasma Glucose. Human Capital 2002–2004 Follow-Up Study, Analytic Subsample 1 (N=339)

	<u>Model 1</u>	<u>Model 2</u>	<u>Model 3</u>	<u>Model 4</u>
	Childhood Morbidity, Age, Sex, and Village of Origin	Model 1 + Family Background	Model 2 + Adult Characteristics	All Explanatory Variables
(a) Low HDL in early adulthood				
Days with diarrhea (age 0–2) ^c	1.05 [†]	1.05 [†]	1.06 [*]	1.04
Days with serious illness (age 0–2) ^c	1.03 [†]	1.03 [†]	1.03	1.01
Years with serious illness	1.23 [†]	1.24 [†]	1.13 [†]	1.19
(b) High triglycerides				
Days with fever (age 2–7) ^c	1.15 [*]	1.15 [*]	1.16 [*]	1.21 ^{**}
Days with diarrhea (age 2–7) ^c	0.94 [†]	0.95 [†]	0.94 [†]	0.92 [*]
Any infectious disease (age 2–7)	1.63 [†]	1.63 [†]	1.60 [†]	1.16
Years with infectious disease	1.33 [†]	1.34 [†]	1.38 [†]	1.22
(c) High plasma glucose				
None				

Notes

[†] p<.01

^{**} p<.05

^{*} p<.01

The key explanatory variables shown are those types of childhood morbidity that are significantly associated with the outcome. Thus, each outcome may have a different set of predictors.

Models control for the number of days for which morbidity was reported, age 0–2 and age 2–7.

^a In Model 1, each childhood morbidity measure is a separate regression. Controls for age, sex, village of origin, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^b Model 4 includes all childhood morbidity, and control for age, sex, village of origin, family background, adult characteristics, health behaviors, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^c Days with Anorexia, Serious Illness, Fever, and Diarrhea are measured in tens of days

Table 6

OLS Regression Coefficients Predicting the Level of HDL, Triglycerides, and Plasma Glucose. Human Capital 2002–2004 Follow-Up Study, Analytic Subsample 1 (N=339)

	Model 1^a Childhood Morbidity, Age, Sex, and Village of Origin	Model 2 Model 1 + Family Background	Model 3 Model 2 + Adult Characteristics	Model 4^b All Explanatory Variables
(a) Level HDL in early adulthood				
Days with diarrhea (age 0–2) ^c	–0.25 **	–0.25 **	–0.25 **	–0.17
Days with serious illness (age 0–2) ^c	–0.10 †	–0.10 †	–0.09	–0.04
Years with serious illness	–0.92 †	–0.94 †	–0.96 †	–0.10
Days with fever (age 0–2) ^c	–0.82 *	–0.82 *	–0.80 *	–0.40
Days with fever (age 2–7) ^c	–0.63 *	–0.61 *	–0.62 *	–0.04
Years with fever	–1.02 †	–1.02 †	–1.16 *	–0.45
Days with anorexia (age 0–2) ^c	–0.19 †	–0.21 †	–0.22 *	–0.01
Years with anorexia	–1.13 *	–1.16 *	–1.17 *	–0.42
(b) Level triglycerides				
Days with fever (age 2–7) ^c	4.70 †	5.14 †	5.44 *	5.99 *
Days with diarrhea (age 2–7) ^c	–3.22 **	–3.02 **	–3.10 **	–4.95 **
Years with serious illness	8.00 *	8.61 *	9.49 *	11.29 *
(c) Level plasma glucose				
Days with infectious disease (age 0–2) ^c	0.13 †	0.13	0.17 †	0.17 †
Days with diarrhea (age 2–7) ^c	–0.23 †	–0.22	–0.26 †	–0.26 †

Notes

†
p<.01

**
p<.05

*
p<.01

The key explanatory variables shown are those types of childhood morbidity that are significantly associated with the outcome. Thus, each outcome may have a different set of predictors.

Models control for the number of days for which morbidity was reported, age 0–2 and age 2–7.

^a In Model 1, each childhood morbidity measure is a separate regression. Controls for age, sex, village of origin, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^b Model 4 includes all childhood morbidity, and control for age, sex, village of origin, family background, adult characteristics, health behaviors, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^c Days with Anorexia, Serious Illness, Fever, and Diarrhea are measured in tens of days

Table 7

Odds Ratios Predicting Hypertension, Abdominal Obesity, and Obesity. Human Capital 2002–2004 Follow-Up Study, Analytic Subsample 2 (N=364)

	Model 1^a Childhood Morbidity, Age, Sex, and Village of Origin	Model 2 Model 1 + Family Background	Model 3 Model 2 + Adult Characteristics	Model 4^b All Explanatory Variables
(a) Hypertension				
Days with serious illness (age 0–2) ^c	1.02 [†]	1.02	1.02	1.01
Days with serious illness (age 2–7) ^c	1.01 [†]	1.01 *	1.01 [†]	1.01
(b) Abdominal Obesity				
Days with diarrhea (age 0–2) ^c	1.07 **	1.07 **	1.07 **	1.04
Years with diarrhea	1.32 *	1.35 *	1.28 [†]	0.92
Days with anorexia (age 0–2) ^c	1.05 [†]	1.05 [†]	1.06 *	1.01
Days with serious illness (age 2–7) ^c	1.02 *	1.02 *	1.02 [†]	1.00
Years with serious illness	1.59 **	1.64 **	1.57 **	1.52 **
(c) Obesity				
Years with serious illness	1.31 *	1.31 *	1.27 [†]	1.24 [†]

Notes

[†]
p<.01

**
p<.05

*
p<.01

The key explanatory variables shown are those types of childhood morbidity that are significantly associated with the outcome. Thus, each outcome may have a different set of predictors.

Models control for the number of days for which morbidity was reported, age 0–2 and age 2–7.

^a In Model 1, each childhood morbidity measure is a separate regression. Controls for age, sex, village of origin, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^b Model 4 includes all childhood morbidity, and control for age, sex, village of origin, family background, adult characteristics, health behaviors, and number of days for which morbidity was reported, age 0–2 and age 2–7.

^c Days with Anorexia, Serious Illness, Fever, and Diarrhea are measured in tens of days

Correlation of Childhood Disease Measures

Table 8

	Days with Diarrhea (age 0–2)	Days with Diarrhea (age 2–7)	Yrs with Diarrhea	Days with Anorexia (age 0–2)	Days with Anorexia (age 2–7)	Yrs with Anorexia	Days with Fever (age 0–2)	Days with Fever (age 2–7)	Yrs with Fever	Days with Serious Illnesses (age 0–2)	Days with Serious Illnesses (age 2–7)	Yrs with Serious Illnesses	Any Infectious Disease (age 0–2)	Any Infectious Disease (age 2–7)	Yrs with Infectious Disease
Days with Diarrhea (age 0–2)	1.00														
Days with Diarrhea (age 2–7)	.43	1.00													
Yrs with Diarrhea	.41	.56	1.00												
Days with Anorexia (age 0–2)	.48	.26	.22	1.00											
Days with Anorexia (age 2–7)	.33	.50	.43	.51	1.00										
Yrs with Anorexia	.19	.33	.57	.28	.50	1.00									
Days with Fever (age 0–2)	.22	.16	.20	.34	.20	.27	1.00								
Days with Fever (age 2–7)	.19	.32	.36	.14	.45	.52	.42	1.00							
Yrs with Fever	.09	.20	.44	.10	.33	.63	.36	.59	1.00						
Days with Serious Illnesses (age 0–2)	.27	.10	.09	.42	.20	.04	.14	.00	–.03	1.00					
Days with Serious Illnesses (age 2–7)	.24	.48	.32	.30	.58	.33	.13	.21	.20	.36	1.00				
Yrs with Serious Illnesses	.23	.36	.58	.15	.40	.58	.18	.40	.45	.22	.46	1.00			
Any Infectious Disease (age 0–2)	–.03	.04	–.12	.06	.07	.07	.12	0.01	.07	–.02	.03	.05	1.00		
Any Infectious Disease (age 2–7)	–.01	.07	–.04	.00	.14	.20	.02	.25	.25	–.06	.11	.21	–.05	1.00	
Yrs with Infectious Disease	–.04	.07	.34	.04	.16	.20	.11	.23	.25	–.02	.09	.18	.64	.67	1.00