Health Dynamics over the Life Course

The Effect of Childhood Health on Adult Health and Disability

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Abstract

This paper models health over the life course using data from the Panel Study of Income Dynamics. It explores whether infant and childhood health have lasting effects on various measures of adult health including: self-reported health, chronic disease, disability, and changes in health over time. I find that low birth weight (LBW) individuals experience substantially worse childhood health. Furthermore, LBW and poor childhood health are associated with worse adult self-reported health, increased odds of having a work-limiting disability or a chronic disease. Those with poor early-life health are also more likely to experience a decline in their health across waves. I find no evidence that the experience of LBW and poor childhood health are further compounded by disadvantaged social background. Though education continues to be associated with adult health, this relationship is attenuated somewhat with the inclusion of childhood health into the model suggesting some effect of health selection.

INTRODUCTION

The nominal purpose of this study is to estimate the determinants of childhood health and the relationship between infant/childhood health and various dimensions of adult health including self-reported general health status, the presence of a work-limiting disability, having a physician-diagnosed chronic disease, and changes in self-reported health over time. This paper further highlights several lines of inquiry into the extent to which health over the life course is both a cause and consequence of the social stratification process. First, it seeks to understand the extent to which infant and childhood health are structurally determined by social class of origin. If health in early life is to be considered an important mechanism for the transmission of socioeconomic position from one generation to the next than it must, at least in part, be a function of parental socioeconomic status. Second, it examines whether the effects of poor infant and childhood health are further compounded by disadvantaged social background creating a double jeopardy effect. Individuals from less advantaged backgrounds that additionally face the potentially deleterious effects of poor infant and childhood health may be particularly worse off in terms of their adult health outcomes. Third, the question of health selection is also addressed here indirectly. If the health selection argument has some validity and early life health is an important determinant of both later life health and socioeconomic status then we would expect its inclusion to have an attenuating effect on the SES/adult health relationship. Lastly, the estimated relationships discussed here have important implications for theoretical debates concerning the determinants of health over the life course. Specifically, they provide insight into the utility of the fetal "programming" or fetal origins hypothesis of chronic disease compared to a life course cumulative insult model.

Recent debates over the validity of the fetal origins hypothesis are embedded within larger historical debates on how best to describe and model the determinants of health across the life course. The source of this debate is empirical observations that have pointed to potentially dramatic effects of health very early on in life including during gestational development in determining downstream chronic diseases in late adulthood such as diabetes and cardiovascular disease. However, this debate between the fetal origins or "latent effects" model of health and the "cumulative insult" model is best understood as emerging as part of a long term shift in the central framework used to describe health or conversely the emergence of disease. This shift has challenged both the simplistic binary healthy/sick classification scheme of the biomedical model of health and the implicit notion that each disease is connected to a singular pathogenic vector. The construct of well-being conceptualizes health of an individual as a multiply determined continuum composed of not just its bio-physiological aspects but its cognitive, emotional, and functional dimensions as well. In order to give a more comprehensive view of where the current work fits into the larger literature on health determinants over the life course I now turn to a brief review of how the discourse over the determinants of health and chronic disease has emerged over the last century.

THE BIOMEDICAL MODEL OF HEALTH & ITS LIMITATIONS

The biomedical model of disease emerged in the latter half of the 19th and early 20th century with the discovery of the germ theory of disease. In the biomedical model (see figure 1), individuals were assumed to exist in a natural equilibrium state of good health. Disease therefore constituted a state of disequilibrium caused by the presence of some exogenous factor (pathogen) infecting the individual and leading to a pathologic

reaction in the body. Equilibrium was restored either through the body's own natural immune response or through some curative intervention. In reality, the dramatic decline in mortality that occurred during the 19th and early 20th century had more to do with public health interventions that limited individual exposures to disease vectors than to the heroic actions of medical science (Preston 1980; Szreter 1988). Nevertheless, in a mortality regime in which the major causes of mortality were of an infectious nature, the germ theory of disease and the resulting biomedical model of health provided an invaluable tool to scientists and public health officials seeking to describe the causes of ill-health and ultimately to eliminate them. While the germ theory of disease was highly successful tool in identifying the microbial causes of disease the subsequent shift in the underlying causes of mortality also exposed the limits of its ability to describe the complex processes that determine health and disease generally.

[Figure 1 about here]

Though the germ theory of disease took center stage early in the 20th century, as Kuh and Davey Smith (1997) correctly point out, notions that the keys to understanding adult health in part lay in early life was also a widely held belief. The view that health and vitality in adulthood were directly related to a healthful physical and social environment in early life which promoted the creation of a healthy "constitution" (Fisher 1909 as cited in Kuh & Davey Smith 1997) was in contrast to those within the eugenicist movement, such as Karl Pearson, who saw health as largely determined by genetic inheritance. Beliefs in the importance of early life environment in part motivated many of the social services such as the hygiene movement and mother's pensions undertaken during this period.

The notion that early life health is an important determinant of adult health became overshadowed in the mid 20th century largely due to dramatic changes in the cause of death structure of the mortality regimes in western industrialized nations that has occurred over the last 250 years. During this period, diseases of an infectious nature (e.g. smallpox and tuberculosis) were replaced by degenerative and chronic diseases (e.g. cancer and cardiovascular disease (CVD)) as the major causes of morbidity and mortality (Omran 1982; Vallin 1991). This was particularly apparent during the first half of the 20th century, when coronary heart disease and lung cancer became an endemic feature of the American mortality regime. Unlike communicable diseases whose etiology could almost always be connected with a specific pathogenic vector, the etiology of chronic diseases is more complicated and multi-factorial. Although a few chronic diseases can be traced back to one or two pathogenic factors[1], most chronic diseases are thought to be the long-term result of exposure to a variety of risk factors. The rise of chronic diseases as the major cause of adult mortality and their multi-factorial pathogenesis led epidemiologists and public health researchers to develop the risk-factor model of health (figure 2). The risk factor model of health shifted attention away from pathogenic exposures associated with microorganisms to those associated with individual level lifestyle. This focus on adult lifestyle and behavioral characteristics tended to obscure the role of early life. Health interventions also shifted in their focus away from the physical and social environment in early life and towards individual level medical interventions. The *raison d'etre* of public health researchers therefore was to identify and minimize those factors associated with elevated risk of disease thereby improving the health of individuals and populations.

[1] For example, lung cancer is very rare among those who have never smoked or been exposed to asbestos.

[Figure 2 about here]

Epidemiology has been successful in identifying some of the more salient risk factors associated with chronic diseases such as cancer and CVD. Most of these risk factors, which include smoking, excessive alcohol consumption, sedentary life style, high-fat/low fiber diet, and stress, can be categorized as adult lifestyle or behavioral factors. Though these risk factors are no doubt important determinants of chronic disease they are severely limited in their ability to explain important social variations in health and disability. For example they explain only about 20-30% of social class variation in cardiovascular disease (Marmot et al 1978).

Because of persistent social differentials in health and the inability of known lifestyle risk factors to explain them, researchers have begun to investigate a more comprehensive model of health determination than the traditional focus on health behaviors and medical care. Evans, Barer, and Marmor (1994) have introduced a framework for analyzing the multiple determinants of health. The Evans model introduces some important features into the traditional biomedical model of disease.1) It brings in the social and physical environment as well as the individual's genetic endowment into the model as proximate determinants. Traditionally, epidemiologists have viewed SES more as a confounding variable either outside of or hopelessly distal to the disease process. They acknowledged that SES may be an important upstream variable, but in practical application it was seen as something that clouded up the otherwise clear waters of causal association between etiologic agent and disease. In the Evans model, SES is seen as an important causal agent, although they continue to seek the more proximate physiological mechanisms by which it manifested its effects. 2) The

Evans model also broadened the outcomes of interest to include functioning and overall well-being, not just the presence of specific disease sequelae. 3) In addition, the Evans model incorporates the economic concept of marginal cost. Resources are scarce and interventions to increase the well-being of populations must be judged by their marginal costs and benefits. Whereas health care investments have been the traditional instrument in attempts to increase health and well-being within populations, the marginal return of these must be compared with alternative investments such as education that may yield higher marginal benefits. By implication this suggests a comprehensive re-examination of how health is achieved and points to the potentially important role played by such factors as education, child care, and early childhood health and social conditions. Health researchers therefore have come full circle, rediscovering early 20th century notions of health determination.

As part of this new found interest in early life circumstances and exposures, an increasingly strong body of evidence has recently pointed to the role of early childhood and even prenatal health and social conditions in shaping both socioeconomic status and health later in life. This area of research has found that poor health early in life such as low birth weight and particular childhood diseases can have lasting effects on adult health (Barker 1994; Elo 1998; Blackwell *et al.* 2001), cognitive development, and high school completion (Conley and Bennett 2000; Rich-Edwards *et al.* 1997; Sorensen *et al.* 1997).

THE FETAL ORIGINS HYPOTHESIS

One theory of the development of chronic disease in mid-life that has received considerable attention lately involves the notion of fetal programming or "fetal origins". This theory is most notably associated with Barker and colleagues (dozens of articles

summarized in Barker 1994). The basic idea behind fetal origins is that poor maternal nutrition at various critical periods during the development of the fetus has long-term impacts on the risk of CVD and diabetes. Poor nutrition leads to growth-retardation of the fetus and fetal adaptations which enable its survival. These adaptations are thought to "program" later life disease by altering the structure and function of important tissues. The biologic plausibility of programming is largely drawn from animal studies (Winick and Noble 1966; Cella *et al.* 1990; Person and Jansson 1992; see also Harding 2001 for a summary).

Two specific mechanisms link fetal adaptations to adult chronic disease. The first is that growth-retardation is associated with insulin resistance and impaired beta-cell development which are linked to both type-II diabetes, and cardiovascular disease. Secondly, growth-retarded fetuses tend to have high levels of cortisol in the blood which elevates fetal blood pressure and may lead to lifelong hypertension thereby increasing the risk for a cardiovascular event.

Dozens of studies have attempted to test the fetal origins hypothesis. Using both geographical correlations and cohort data, Barker and associates have shown that poor nutrition during gestation as evidenced by low birth weight, infant thinness relative to length, the ratio of head to chest circumference and various other anthropometric measures are associated with lifetime elevated blood pressure, impaired lipid metabolism, and the risk of mortality from CVD (Forsdahl 1977; Barker 1994). These effects remain after controls for later life SES are introduced. However, critics of fetal origins have charged that early life SES is not adequately controlled for (Paneth and Susser 1995). The

implication of the fetal origins model of chronic disease is that interventions later in the life course are not likely to yield beneficial results.

The fetal origins hypothesis has been the subject of great controversy as of late. In a meta-analysis of studies on the association between low birth weight and systolic blood pressure Huxley, Neil, and Collins (2002) find that previous findings of a strong inverse relationship are likely to have been severely overstated. The actual association they find is on the order of 0.6 mm Hg per added kilo gram of infant body weight (Huxley, Neil, and Collins 2002). This is substantially smaller than the 2-4 mm Hg typically found (Law and Shiell 1996). In addition to a lack of sufficient control for exogenous factors such as family SES and other household characteristics mentioned above, the population attributable risk associated with low birth weight is largely insufficient to explain the prevalence of either cardiovascular disease or diabetes mellitus. Even if the associations between low birth weight and adult chronic disease are indeed causal, the notion of fetal origins as the dominant causal determinant of coronary heart disease or diabetes mellitus cannot be sustained.

A third line of criticism is that associations between low birth weight and adult health outcomes often are strengthened or only appear once controls for current body mass have been included (Paneth and Susser 1995; Lucas, Fewtrell, and Coale 1999). This raises the question of whether low birth weight *per se* is what is determinant or if it is the combination of retarded fetal growth followed by later life overweight or obesity that is really the culprit (Paneth and Susser 1995). In an attempt to get at this problem the analysis presented below tests for the general effect of low birth weight on various adult health measures including physician diagnosis of cardiovascular disease and diabetes

with and without controls for current BMI. Also, a separate analysis tests for differences in the effect of later life body mass conditional on low birth weight status.

The fetal origins hypothesis is an example of critical period or latent effects model in the life course literature (Keating & Hertzman 1999; Kuh & Ben-Shlomo 1997; Goldman 2001). The idea of critical periods is that negative events that occur during periods that are particularly important may permanently alter the trajectory of the life course (sort of a path dependence argument). It is not until much later do these effects manifest themselves in disease pathologies.

An alternative to the critical periods approach is the life course, "pathways", or "cumulative insult model". This model suggests that exposures accumulate over the life course and that it is this lifetime accumulation that is important. This approach is not necessarily inconsistent with the existence of critical period effects, however it takes the position that there are social, environmental, and behavioral exposures that over the life course alter an individual's risk of disease in addition to critical period effects (Kuh & Ben-Shlomo 1997). For the most part researchers from the life course perspective accept the robust association linking low birth weight and adult chronic disease. However, they are critical of interpretations of fetal origins that are overly deterministic and that otherwise dismiss the contribution of exposures that occur later in life such as from smoking, diet, and sedentary lifestyle. They suggest that there may be important interaction effects between fetal and later life exposures. This issue of low birth weight followed by adult overweight and obesity discussed above is an example of such interaction.

THE LONG ARM OF CHILDHOOD

In addition to research concerning the fetal origins hypothesis there is also a growing literature on childhood social and economic conditions, psychological adjustment, and physical health as determinants of adult health outcomes in what Hayward and colleagues refer to as the "long arm of childhood" (Hayward, Gorman, and Robinson 2001). Much of what is known about early life influences on health come from the 1946 British national birth cohort study (MRC National Survey of Health and Development) (Wadsworth and Kuh 1997). A number of analyses using this and other data have connected adult health outcomes to the social and economic characteristics of the family of origin.

Social and Economic Characteristics of Family of Origin

One characteristic that has received a fair amount of investigation (primarily because it is often held up as an alternative to the fetal origins hypothesis) is social class of family of origin. As an alternative to the fetal origins hypothesis, social class of origin may act as a common upstream determinant of both low birth weight and adult social position (and subsequently adult health). This common cause therefore may create a spurious association between low birth weight and adult chronic disease.

Numerous studies have found that those who come from disadvantaged backgrounds tend to have higher morbidity and mortality in adulthood (Notkola *et al.* 1985; Peck and Lundberg 1995; Kuh and Wadsworth 1993; Brunner *et al.* 1996; Leon *et al*. 1996; Hayward, Gorman, and Robinson 2001; Blackwell *et al*. 2001; Peck 2002). Similarly those who lived in crowded housing conditions at age 2 had reduced peak respiratory flow rates at age 36 (Mann, Wadsworth, and Colley 1992).

Another dimension of the childhood social environment that researchers have examined for potential effects on adult health, mortality, and health-related risk behaviors is childhood family structure and experience of family disruption. For example Schwartz *et al*. (1995) find that parental divorce increased mortality hazard rates after age 20 for both men and women. Hayward, Gorman, and Robinson (2001) find the opposite. They find that some forms of family disruption actually lower mortality risk. Relative to those who lived with their biological mother and father, men who lived with their father and step mother, their father only, or a male relative had lowered mortality risk over a 24 year period. Parental divorce or separation prior to age 16 has been found to be associated with increased smoking among men and significantly higher alcohol consumption among women (Kuh and Maclean 1990; Wadsworth, Maclean, Kuh, and Rodgers 1990).

Childhood Health and Development

Researchers, most notably those associated with the 1946 British national birth cohort study has also linked the experience of childhood illness to adult physical health. For example, Colley and colleagues (1973; 1976) have observed that the experience of lower respiratory illness in the first two years of life increased the risk of symptoms associated with chronic obstructive pulmonary disease (COPD) (Colley, Douglas, and Reid 1973; Kiernan, Colley, Douglas, and Reid 1976). The experience of serious illness between ages 5-24 was also found to be associated with poor health at age 36 measured by having at least one of the following: any disability, any life threatening health problem, hypertension, obesity, or being in the lowest decile of peak respiratory flow rate (Kuh and Wadsworth 1993).

More recently Blackwell, Hayward, and Crimmins (2001) investigated the relationship between childhood health and chronic morbidity in the Health and Retirement Study (HRS) sample of near elderly. As with the analysis presented here, the indicator of childhood health used by Blackwell *et al.* is based on retrospective reports of childhood health. In an experimental module of the HRS respondents were asked whether or not as a result of a health condition they had 1) ever missed one month or more of school, 2) were restricted from participating in sports for three or months, or 3) if they had to remain in bed at home for one month or more. Those respondents that answered affirmatively were then asked to name the specific condition from which their incapacity resulted. Despite the limited sample size in the experimental module, significant associations were found between childhood and adult health. Those who experienced a serious infectious disease in childhood had 1.7 times greater odds of reporting a physician diagnosis for cardiovascular disease at age 55-65. They also had 1.8 times greater odds of arthritis/rheumatism, 3.6 times greater odds of cancer, and 4.1 times greater odds of lung conditions. These effects were net of age, race, gender, social background, current SES, height, and co-morbid conditions.

A number of studies also examine the relationship between childhood and adult health and mortality by using height as a proxy for early life health and nutritional status. Achieved adult height is often used in the absence of direct measures of childhood health because it has been shown to be a fair indicator of child health and development (Floud Wachter, and Gregory 1990). Such studies have found significant associations between achieved adult height and adult morbidity and mortality (Waaler 1984; Marmot 1986; Peck and Vågerö 1989; Allebeck and Bergh 1992; Vågerö and Leon 1994; Peck and

Lundberg 1995). It is generally the case that short stature is associated with increased morbidity and mortality, as is the case for cardiovascular and respiratory disease (Waaler 1984; Yarnell *et al.* 1992; Rich *et al.* 1995). However, among women, tallness is associated with increased risk of breast cancer (Hunter and Willett 1993).

THE CURRENT STUDY

The present study adds to the growing literature on early life influences on adult health in a number of important ways. 1) It examines the effects of infant and childhood health on several dimensions of adult health simultaneously. Rather than focusing on 1 or two specific health outcomes or dimensions, this present study provides a broad overview of how health in early life affects adult health generally. Rarely do studies include measures of both low birth weight status and childhood health 2) By utilizing multiple indicators of childhood health including low birth weight status, generalized health in childhood, and achieved adult height, the present study can tease out differential effects of the prenatal period from those occurring in childhood. 3) Rather than simply adding social background as a control variable to get an unbiased estimate of infant and childhood health, the present study goes further, actually investigating the presence of interaction or double jeopardy effects of both disadvantaged social background and poor infant and childhood health. 4) In addition to examining the relationship between early life health and that at one point in adulthood, the present study also examines the effect of infant and childhood health on changes in adult health over time. This allows a better understanding of how early life shapes not just the overall level of adult health but health trajectories as well.

DATA & METHODS

Data

Data for this analysis come from the *Panel Study of Income Dynamics* (PSID). The PSID is a nationally representative longitudinal survey of households begun in 1968. The PSID was designed to investigate the effects of poverty and policies directed at its amelioration. The data detail the economic and demographic dynamics of households and include information on topics such as work, income, wealth, and housing. This analysis focuses on data derived from the 1999 and 2001 waves because the measure of childhood health was only gathered in these waves. I use a very select subset of the more than 50,000 individuals on which data have been collected, namely those that were heads and wives in the 2001 wave who also have valid information of birth weight status. In order to have information on weight at birth a respondent must be a child born to one of the original 1968 households (though they may have been born prior to 1968). Roughly 2800 current heads and wives have information on birth weight. I further limited the analysis to those aged 18-64. With the exception of a few variables discussed below, cases with missing data on either explanatory or outcome variables were deleted in a list wise fashion. Because some heads in the sample are the siblings of other heads there is clustering by original 1968 household. In the analyses standard errors have corrected for this clustering.

Measurement

Low Birth Weight

Information on weight at birth is provided by maternal reports. Previous research has shown maternal recall of child's birth weight to be accurate (Walton *et al.* 2000). In order to have information on weight at birth a respondent must be a child born to one of

the original 1968 households (though they may have been born prior to 1968). Low birth weight is defined in the standard fashion as weighing less than 88 ounces (5.5 Pounds) at birth. Those weighing less than 88 ounces at birth were assigned values or 1 and those weighing 88 ounces or more at birth were assigned values of 0.

Childhood Health

The measure of childhood health used in this study is based on response to the following question: "Consider your health while you were growing up, from birth to age 16. Would you say that your health during that time was excellent, very good, good, fair, or poor?" Values of 1(excellent) - 5 (poor) were then assigned to these categories. A similar question was asked in an experimental module in wave 3 of the Health and Retirement Study (HRS). Elo's (1998) analysis of these data found that responses had a high level of internal consistency between the report of general health and reports of specific long-term health limitations in childhood. While global measures like these are often dichotomized, because of the use of structural equation models I keep it in its original ordinal metric. To maximize sample size, the childhood health measure is based on the 2001 report except for a small number of cases that had missing data in 2001. These cases use the 1999 reports.

My analysis of retrospective reports of childhood health across the two waves suggests that the retrospective reports used here are fairly reliable over time, especially when the measure is dichotomized into a good/very good/excellent vs. fair/poor comparison (Haas 2003)[2]. Analysis of the effect of low birth weight on childhood health shown below, demonstrates that low birth weight exerts rather strong adverse effects on

[2] Across all groups the gamma measure of association was 0.597 for the 5-level variable. When dichotomized, the gamma increases to 0.904.

childhood health. Given the consistent relationship between birth weight and objective measures of childhood health (Brooks *et al.* 2001; Vohr *et al.* 2000; Stevenson *et al.* 1998), the fact that a similar relationship is found using retrospective reports lends support for their validity. Childhood health is measured on a five point ordinal Likert type scale ranging from 1 (excellent) to 5 (poor). So odds-ratios greater than 1.0 reflect an increase in odds of poorer health and odds ratios less than 1.0 reflect decreased odds of poorer health. Results based on a dichotomized version of the childhood health measure do not change substantively.

Self-Reported Adult Health 1999 & 2001 Waves

As with childhood health, self-reported adult health is measured on a five point ordinal Likert type scale ranging from 1 (excellent) to 5 (poor). Odds-ratios greater than 1.0 reflect an increase in odds of poorer health and odds ratios less than 1.0 reflect decreased odds of poorer health. In order to assess the effect of infant and childhood health on adult health longitudinally, an indicator of decline in self-reported adult health is also included in these analyses. This indicator is given a value of 1 if self-reported health declined at least 1 level between the 1999 and 2001 waves and 0 if not.

Work-Limiting Disability

An indicator of work limiting disability are based on responses to the following question- Do you have any physical or nervous condition that limits the type of work or the amount of work you can do? Affirmative responses are coded 1 and negative responses 0.

Physician Diagnosed Chronic Disease

Any Chronic Condition

Respondents were asked if a physician had ever diagnosed any of the following conditions: stroke, high blood pressure or hypertension, diabetes or high blood sugar, cancer or a malignant tumor, chronic lung disease such as bronchitis or emphysema, heart attack, coronary heart disease, angina, congestive heart failure, any emotional, nervous, or psychiatric problems, arthritis or rheumatism, asthma, permanent loss of memory or loss of mental ability, a learning disorder. Responses were assigned 1 if they answered in the affirmative to any of these conditions and 0 if they responded negatively to all.

Though these measures are not completely objective, for example they are still subject to recall error and other forms of selective response, they refer to the presence or absence of an objective clinical diagnosis of a specific set of disease sequeale. Previous research has shown that these reports have a high degree of agreement with medical records (Bush, Miller, Golden, and Hale 1989; Pasty *et al.* 1995). Furthermore, analysis of chronic conditions generally, and cardiovascular disease (CVD) and diabetes specifically, are most relevant to assessing predictions derived from the fetal-origins hypothesis.

Cardiovascular Disease

Regarding the presence of a physician diagnosis of CVD, respondents were assigned 1 if they answered in the affirmative to any of the following conditions: stroke, high blood pressure or hypertension, heart attack, coronary heart disease, angina, or congestive heart failure and 0 if not.

Diabetes

As with cardiovascular disease, those who gave affirmative to responses to a question asking whether a doctor has ever told them they had diabetes or high blood

sugar were assigned values of 1 and those with negative responses were assigned values of 0.

Other Statistical Controls

The analyses also include a standard set of control variables. These include information on demographic background such a series of age dummy variables (19-25, 26-35 (reference), 36-45, 46-55, and 56-64), and indicators of race and ethnicity, and current marital status. In the analyses based on the low birth weight head sample and indicator for gender is included in the model while in the analyses based on the full sample, all models are run separately by gender. Measures of both current SES (years of completed education), and SES of the family of origin (parental educational attainment) are also included. The analyses also control for the potential confounding effect of health-related risk factors including overweight (BMI 25-29), obesity (BMI $\geq$ 30), and indicators of being a current or former smoker. In addition as it is often used as a proxy measure for childhood health, nutrition, and development, I also include a measure of achieved adult height in inches.

Descriptive statistics for are presented in table 1. The average age of the sample is 39.2 for men and 38.3 for women. More than sixty percent of the sample is between the ages of 36 and 55. As would be expected given this relatively low mean age, the average health of the sample is pretty good. Only 5.9% of men and 7.7% of women report themselves as being in fair or poor health in 1999 while 7.8% and 8.1% report having a disability that limits either the type or amount of work they can perform. Though the sample is in generally good health, it is also clear that significant health problems are beginning to emerge. Almost a third of the sample reports any physician diagnosed

chronic disease with about 13% reporting cardiovascular disease. In addition, about a quarter of the sample experienced a decline in their self-reported general health of at least one point between the 1999 and 2001 waves. The sample respondents also have high rates of health related risk factors. For example, almost two-thirds of men and more than half of the women in the sample is either overweight or obese (BMI > 25) and just under half are current or former regular smokers.

[Table 1 about here]

As would be expected given that these represent the children born to original 1968 households the average age of respondents is slightly lower it is for household heads in the full sample. Because of this age advantage this sample is also slightly more highly educated and is in better overall health. They were also slightly less likely to report a work-limiting disability, a chronic health condition, or to experience a decline in their health across waves. This sample also report slightly better childhood health than those in the full sample. Five percent of men and 8.9% of women in the sample were born low birth weight (less than 88 ounces).

Analytic Strategy

The results resented here are based on logistic regression models. In the case of ordinal outcome variables such as childhood health and self-reported current adult health these are based on ordered logit models. For all other outcome variables parameter estimates are based on binary logistic regression models. For the analysis of the effect of infant and childhood health on adult health outcomes I employ a series of nested models. In these nested models model 1 estimates the bivariate relationship between educational attainment and adult health. Model 2 adds measure(s) of infant and childhood health.

Model 3 further controls for socio-demographic characteristics such as race/ethnicity, age, and marital status. Model 4 adds the effects of parental education, while model 5 (full model) adds controls for health related risk factors such as obesity and smoking behavior.

To examine whether the effect of low birth weight and poor childhood health vary by social class of origin a separate analysis presents the effects of infant and childhood health stratified by both mother's and father's educational attainment. Similarly I also employ analysis stratified by low birth weight status to examine the differential effects of health-related risk factors by low birth weight status.

RESULTS

The Determinants of Childhood Health

Table 2 presents estimates from an ordered logistic regression model of childhood health. From these estimates several observations can be made. First, for men there is a large positive relationship between birth weight and childhood health. Those that were born low birth weight have substantially increased risk of poor childhood health. While low birth weight also increases the odds of poor health in women this is not statistically significant. Second, males report significantly better health in childhood than do women based on retrospective reports. Third, there does not appear to be significant racial differences in childhood health. However, because of sample size constraints it is not possible to estimate differences between Asians, Latinos and white, and may also limit the model's power to detect such differences between whites and blacks. In addition, being born to a married mother appears to exert a protective effect on childhood health for men though this was not statistically significant. Finally, those who come from

families with higher paternal education experienced healthier childhoods. Those fathers with at least some post secondary education have children with significantly lower odds of being in worse health during childhood. Although they are generally in the expected direction, other effects of parental education fail to achieve statistical significance. Again this may be due to sample size constraints.

The Effect of Infant & Childhood Health on Self-Reported Health (1999)

[Table 2 about here]

Table 3 presents the results (odds-ratios) of a series of nested ordered logistic regression models with self-reported global health from the 1999 wave as the dependent variable. Confirming the findings of most previous studies, I find a significant educational gradient in global self-reported adult health. Though this relationship is attenuated when various known proximate determinants such as health related risk factors are added to the model it remains large and statistically significant in the full model. Importantly, when measures of infant and childhood health are added to the model (model 2) the relationship between education and health is attenuated somewhat. Though there is not a significant effect of being born low birth weight, there is a large and statistically significant relationship between childhood and adult health. A one unit increase in the childhood health measure doubles the odds of falling into a worse adult health category. This association persists even after other known health determinants are added to the model.

[Table 3 about here]

Whites and those above the age of 35 also report better overall health though the racial differentials become statistically insignificant when social background and risk factors are included in the model. Men whose fathers had missing educational data or

respondents whose father had failed to complete high school were also at increased risk of poorer adult health. No significant effect of maternal education was found. In addition, obesity and being a current smoker were each associated with worse adult health.

The Effect of Infant & Childhood Health on Work-Limiting Disability

Results of Binary logistic regression in the form of odds-ratios are presented in table 4. Again, there is a distinct educational gradient in the likelihood of having a work limiting-disability. Those with higher levels of educational are significantly less likely to report such a disability. As with self-reported health this relationship is somewhat attenuated by childhood health. Poorer health in childhood is associated with an increase in the likelihood of reporting a disability though this relationship is only statistically significant among women. A one unit increase in the childhood health is associated with a 20-50% increase in the odds of having a disability. There is no statistically significant relationship observed between low birth weight and disability. As with self-reported health, to the extent that low birth weight has adverse effects on disability in adulthood these are indirect, acting through impaired health in childhood.

[Table 4 about here]

Disability generally increases with age with those between the ages of 36-55 at significantly increased risk of disability. Currently married individuals are also 35% less likely to report being disabled relative to their non-married peers. Female former smokers have a nearly 80% increased odds of reporting a physical or nervous condition that prevents or limits the type or amount of work they are able to do while among men the risk is nearly 2.5 times larger. There also seems to be a race/gender interaction with black

females having their odds of reporting a disability only half that of white women. This is not the case among black males.

The Effect of Infant & Childhood Health on Chronic Disease

The results discussed above clearly demonstrate a substantively non-trivial, statistically significant association between childhood and adult health outcomes. However, despite the fact self-reported health has been shown to have substantial predictive validity in regard to mortality rates (Mossey and Shapiro 1982), self-reported global health and self-reported disability are subjective measures of health. Perhaps a better test of whether childhood health continues to impact health in adulthood would be to use more objective measures of health. One such set of measures involves physician reported chronic conditions.

Any Physician Diagnosed Chronic Condition

Table 5 presents the results of binary logistic regression models of having any physician diagnosed chronic disease. Several important findings are present. First, though there is a significant educational gradient in the likelihood of chronic disease this effect is attenuated and is no longer statistically significant when measures of low birth weight, childhood health, and other controls are added to the model. Second, in the full model (model 5) low birth weight has a large and significant effect on the likelihood of having a chronic disease for men. Men who weighed less than 88 ounces at birth have 1.76 times greater odds of having a chronic disease than or normal weight births. While low birth weight increases the odds of a chronic condition for women this is not statistically significant. This would seem to provide support for the fetal origins hypothesis of chronic disease etiology. However, General health in childhood also has a significant impact on

chronic disease. Experiencing poorer health in childhood is associated with an increase in the odds of having a diagnosed chronic condition. Given that childhood health is in part determined by birth weight status, low birth weight thus also exerts effects on chronic conditions indirectly via childhood health. Therefore some of the effects of retarded fetal growth are mediated by what happens later on in childhood. In addition, chronic conditions continue to be influenced by health related risk factors such as obesity and smoking behaviors. These results provide support for both the fetal origins and the cumulative insult models of chronic disease. They further suggest that these two approaches are not necessarily mutually exclusive.

[Table 5 about here]

Cardiovascular Disease

Having demonstrated the long term effects of low birth weight and childhood health on chronic disease generally, I now turn to specific chronic diseases to get a better sense of which conditions are most sensitive to poor health early in life. The fetal origins theory would predict cardiovascular disease (CVD) to be *the* chronic condition most likely to be impacted by birth weight.

If the estimate of low birth weight on any chronic disease represents the effect of LBW averaged across all conditions, and if CVD is presumed to be most influenced by LBW then we would expect that LBW would exert a larger effect on CVD than for all chronic conditions together. This turns out to be the case particularly for men as seen in table 6, which presents the parameter estimates (odds-ratios) from binary logistic regressions of CVD. The effect of low birth weight for men is to increase the odds of reporting a doctor diagnosis of cardiovascular disease by 2.76 times. As you'll recall, low

birth weight increased the odds of any chronic condition among men by only about 75% (OR=1.761). This confirms an important prediction of the fetal origins model. Again, though low birth does increase the risk of cardiovascular disease among women, this is not statistically significant.

[Table 6 about here]

However, the cumulative insult model is also supported. Net of the effect of fetal development, health status during childhood, and adult socioeconomic and health related characteristics continue to have lasting impacts on the risk of cardiovascular disease diagnosis. Every additional year of schooling is associated with about a 7% decrease in the odds of CVD.

Diabetes

The analysis of diabetes in the smaller sub-sample of heads is made prohibitively difficult by the extremely small sample size and the relative rarity of diabetes cases in the data. Only 3.5% or 97 of the 2805 cases report having a physician diagnosis for diabetes. Therefore there is only adequate statistical power to detect the largest of effects. As seen in table 7 poorer childhood health is associated with significantly increased odds of diabetes among women. For men the only variables that achieve statistical significance are age and obesity, in which the effects are very large with odds ratios of the order of 3 or larger. Although I am reluctant to draw any strong conclusions, the estimated effects of low birth weight, though not statistically significant, are in the expected direction as to be consistent with results of CVD.

[Table 7 about here]

Global Self-Reported Health (2001 Wave)-Autoregressive Models

Parameter estimates derived from ordered logistic regression models of global self-reported adult health from the 2001 wave are presented in table 8. Again health in the period of childhood continues to act as an important determinant of later life health. This is true even when a control for poor adult health in the previous wave is included in the model suggesting a lagged effect of childhood health on adult health. A one unit increase in childhood health (reflecting worse health) increases the odds of falling into a higher (and thus worse) health category in adulthood by a factor of 2.4 times for men and 2.1 times for women. As with self-reported health in the previous wave poorer self assessments of ones health is associated with being black, older, and obese.

[Table 8 about here]

Decline in Self-Reported Health 1999-2001

While analyses described above present a description of the relationship between childhood health and various dimensions of adult health at different points in time it is also important to investigate what relationship it has to changes in adult health over time. This section provides a glimpse into the effect that childhood health has on health trajectories in adulthood.

In addition to having adverse effects on adult health at various cross sections table 9 demonstrates that poor health in childhood also increases the likelihood of a decline in health across waves. In a seemingly anomalous finding, those who were in poor health in the 1999 wave are markedly less likely to experience a decline in their health across waves. However, given that these cases are already in the two worse health states that are possible on the ordinal scale it makes sense that they are less likely to experience a decline in their health. After all, in terms of the scale they cannot get much worse off if at

all. With the exception of obesity among women other socio-demographic characteristics and health related risk factors do not appear to increase the risk of a health decline to a statistically significant degree.

[Table 9 about here]

Summary

The analyses presented above illustrate that indeed infant and childhood health have substantial impacts on adult health measured along various dimensions. Those who report worse childhood health have increased odds of reporting poorer current subjective health, are more to have a work-limiting disability, and to have a physician diagnosed chronic disease. They are also more likely to experience a decline in their self-reported health longitudinally. The estimated long-term effects of low birth weight are less consistent. No statistically significant effects are found for either current or longitudinal changes in self-reported health. Similarly those born low birth weight do not spear to have increased risk of work-limiting disability. However, as has been found in previous studies associated with the fetal origins hypothesis, low birth weight was found to be significantly predictive of physician-diagnosed chronic disease, especially cardiovascular disease.

The preceding analysis also suggests a role (albeit perhaps a small one) for selection mechanisms in determining the relationship between educational attainment and adult health. The addition of infant and childhood health routinely results in the attenuation of the effect of educational attainment on health. This attenuation is generally modest and also varies by the particular health outcome being examined. Nevertheless,

some of the covariance between adult health and educational attainment appears to stem from their jointly being a function of childhood health.

Do the Effects of LBW and Poor Childhood Health Vary by Social Class of Origin?

While the effects of infant and childhood health described above are net of both current and childhood SES they are also estimates pooled over all social backgrounds. An important question is whether these effects are consistent across social class of origin or are those from (dis)advantaged backgrounds more adversely affected by poor infant and childhood health? To answer this question, analysis of the effects of infant and childhood health were stratified by both mother's and father's educational attainment. The results of these analyses are presented below.

Table 10 presents the effects (odds-ratios and associated 95% confidence intervals) of low birth weight and childhood health on self-reported health, disability, any chronic disease, cardiovascular disease, and decline in self-reported health across waves stratified by both mother's and father's educational attainment. Because only 3.5% of observations report a physician diagnosis of diabetes I am unable to estimate these models stratified by parental education. Regarding low birth weight, a couple of findings stand out. First, as in the pooled analyses, low birth weight does not have statistically significant effects on self-reported health and disability among those from any social background. Second, the confidence intervals associated with these estimates tend to be quite large due to sample size constraints almost always overlap. Therefore, the effect of low birth weight does not appear to vary by parental education, at least not to a statistically significant degree.

[Table 10 about here]

Similar results hold for the effect of childhood health. Though, the parameter estimates would suggests, counter intuitively, that poor childhood health was worse for those that come from relatively advantaged backgrounds. However, as with those for low birth weight, the confidence intervals all overlap so there are no statistically significant differences by social background.

From the preceding analysis there do not appear to be significant double jeopardy effects by which the adverse effects of low birth weight or lowered childhood health are further compounded by socioeconomic disadvantage of family of origin. In all but a small minority of cases the confidence intervals associated with differential estimates by parental education overlap. In those cases where there are statistically significant differences, these are in the opposite direction than would be expected. Poor childhood health in these cases is particularly adverse for those from more advantaged backgrounds.

Life Course Interactions between Low Birth Weight and Health-Related Risk Factors

Previous analysis discussed above illustrates the adverse consequences associated with low birth weight. Consistent with the fetal origins hypothesis I find that low birth weight is associated with increased risk of chronic disease, particularly in the case of cardiovascular disease. Resent debates about the fetal origins hypothesis have highlighted the need to separate out the so-called fetal effects from those associated with later growth and obesity. In addition because retarded fetal growth is associated with impaired lung development and lung capacity it may also be that the effect of subsequent smoking behavior is more detrimental to adult health among the low birth weight. To test these assertions I estimated the effects of BMI category (normal BMI<25; overweight BMI 25-29; obese BMI≥ 30) and smoking history (never, current, former) on adult the adult

health measures separately by low birth weight status. The results of these analyses are presented in table 11.

[Table 11 about here]

In looking at these results a couple of findings stand out. First, as would be expected, obesity in adulthood appears to be associated with adverse health outcomes in adulthood independently of low birth weight status. For example, obesity significantly increases the odds of reporting a cardiovascular condition even among the non-low birth weight respondents. Second, the deleterious effects of obesity on adult health are further compounded by retarded growth *in utero*. The effect of obesity on adult health is consistently larger among those born low birth weight than for those not born low birth weight. In the case of any chronic disease the effect is 36% larger for low birth weight respondents than it is for the non-low birth weight. The results for cardio vascular disease are even more pronounced. Respondents that weighed less than 88 ounces at birth and who were subsequently obese in adulthood had 75% higher odds of reporting a cardiovascular condition than non-obese low birth weight respondents. This compares to much smaller 2.5 times increased odds of CVD among obese non-low birth weight respondents.

The analysis of interaction between low birth weight and subsequent smoking behavior does not indicate the consistently larger effects of smoking among the low birth weight respondents as was seen for obesity. While current and former smokers have consistently worse health outcomes than do those that have never been regular smokers, and that the estimated effects of smoking are generally larger among the low birth weight sample, these differences are much less pronounced than those for obesity.

While these analyses show a clear and consistent pattern of interaction between low birth weight and later life obesity in the risk of poor adult health outcomes and hint at one with later life smoking behavior, they should nonetheless be taken very cautiously. Because of the relatively small number of low birth weight respondents in the data the confidence intervals that bound the estimated odds ratios are quite large and always overlap.

DISCUSSION

The analysis presented here illustrate the importance of taking exposures over the life course into account in order to better understand the determinants of adult health. They further highlight the importance of childhood health and early life social environment in the genesis of socioeconomic differentials in health. The results confirm that socioeconomic status of family of origin and low birth weight (men only) are important determinants of childhood health. Those from less well educated families report worse general health over the period of their childhood as do men that weighed less than 88 ounces at birth. The latter association lends indirect support to the notion that retrospective reports of childhood health may be reasonably valid indicators of childhood health.

It is also shown that infant and childhood health continue to have lasting effects on very dimensions of adult health. Poorer childhood health is associated with worse self-reported adult health in the 1999 wave. In addition, those whose childhood experience was unhealthy also were more likely to report a work-limiting disability and the presence of a physician-diagnosed chronic condition. In addition to these effects at one point in time, poor childhood health is also associated with increased odds of experiencing a

decline in self-reported health status across waves. Therefore, early life health appears to alter both the overall level of adult health as well as its trajectory.

Similarly, as would be predicted by the fetal origins hypothesis, low birth weight is associated with adverse adult health outcomes. Being small at birth is associated with increased risk of having any chronic condition. This is especially true for cardiovascular disease where low birth weight nearly doubles the odds of CVD. As with previous analyses of fetal origins, I find that the addition of controls for current body mass increases the size of this association. However, for cardiovascular disease, this association is still large and statistically significant even without these controls. Further analysis stratified by low birth weight status suggests that there is indeed a strong interaction effect between retarded fetal growth and subsequent obesity. The effect of obesity on the odds of CVD was four times larger among those with retarded fetal growth than fro those normal size at birth. This would seem to suggest that the very adaptations that allow the fetus to survive in a nutrient poor environment (albeit with retarded growth), are also detrimental to survival in which there is relative abundance.

While low birth weight continues to be an important determinant of adult chronic disease, the notion of low birth weight as *the* central factor in the genesis of chronic disease is not supported here. In the full model the relative risk associated with low birth weight is smaller than that associated with obesity. In addition, as we've seen above the effect of low birth weight is further compounded by adult obesity. Low birth weight also exerts an indirect effect on adult health through it adverse effects on childhood health. The findings reported here confirm that even the risk associated with fetal development is

best understood within the context of the life course and the accumulation of exposures over time.

Contrary to expectation, the deleterious effects of low birth weight and poor childhood health do not appear to be compounded by disadvantaged social class of origin. It was postulated that low birth weight and poor childhood health may be particularly deleterious for those with the added disadvantage of low socioeconomic status of family of origin. In fact, there is some indication that the opposite pattern may be present. That is the long term consequences of poor health in early life may be relatively worse for those from more advantaged backgrounds. However, for the most part differences in the effect of low birth weight and poor childhood health are not statistically significant.

The results presented here show clearly that substantial gains in understanding adult health outcomes can be had from better understanding the determinants of health over the life course. They also suggest that the broad parameters of health trajectories may be forged very early in life as unhealthy children become unhealthy adults and healthy children healthy adults. This is not to imply that later life exposures and insults do not matter. On the contrary, it implies that the key to understanding health status over the life course lies in understanding how health trajectories are forged and how later life exposures influence these trajectories. Therefore rather than representing a theoretical sea change as the fetal origins hypothesis would suggest, the observation that low birth weight and childhood health have long-term effects on adult health can be easily incorporated into the existing cumulative insult framework.

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Fig. 1 The Simple Biomedical Model of Health

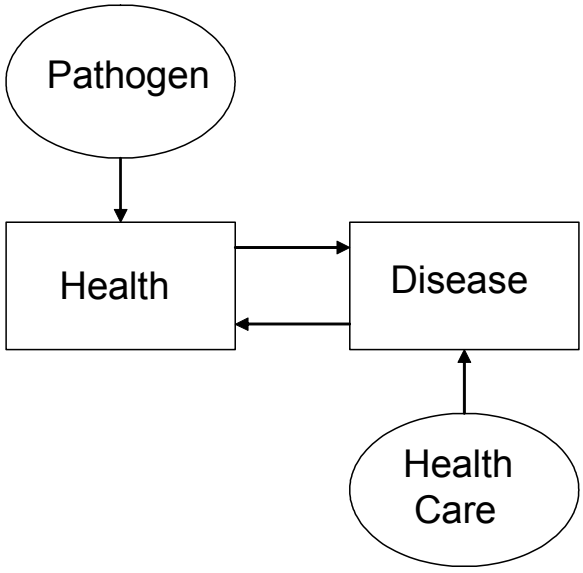

Fig. 2 The Risk Factor Model of Health

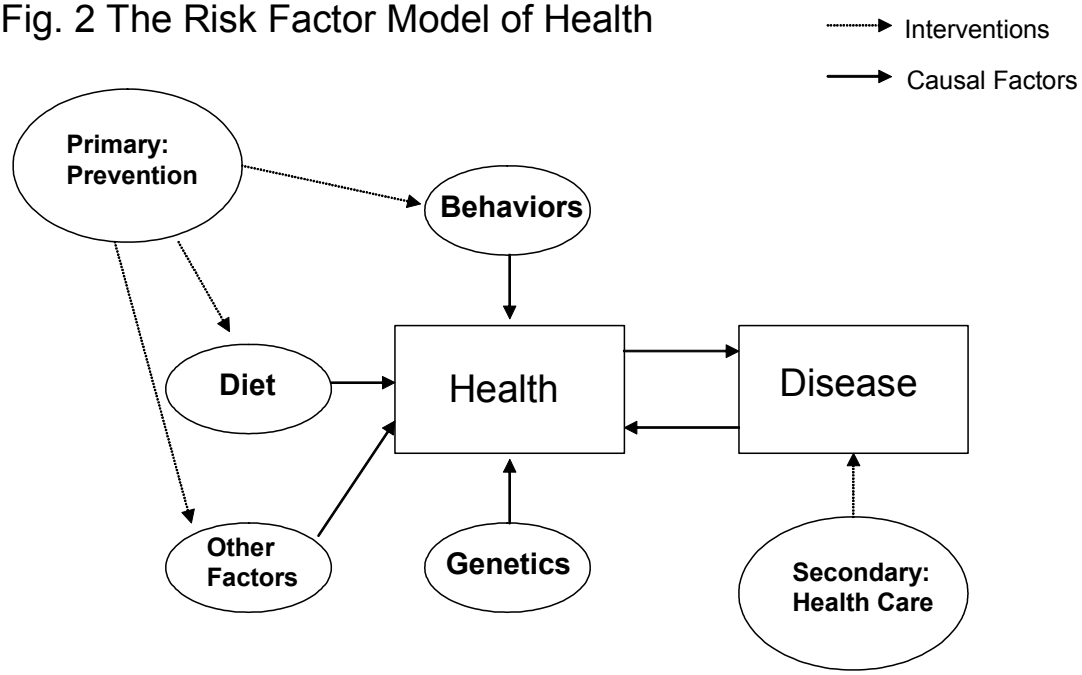

Table 1 Descriptive Statistics for Birth Weight Sample

	Men			Women		
	%	Mean	S.D.	%	Mean	S.D.
Outcome Variables						
Adult Self-Reported Health 1999		2.038	0.92		2.222	0.924
Poor Health 1999 (Fair or Poor)	5.9			7.7		
Adult Self-Reported Health 2001		2.093	0.928		2.257	0.935
Work Limiting Disability	7.8			8.1		
Physician Diagnosed Chronic Disease	28.7			33.7		
Cadiovascular Disease	13.5			13.4		
Diabetes	3.3			4.1		
Childhood Health		1.579	0.785		1.757	0.866
Health Declined 1999-2001	25.4			21.1		
Predictor Variables						
Low Birth Weight	5.0			8.9		
Mother Married at Birth	89.6			83.9		
Socio-Demographic						
Education (years)		13.510	2.132		13.390	2.066
Black	23.9			41.6		
Married	66.7			55.2		
Age		39.193	8.818		38.259	8.372
19-25	6.2			6.6		
26-35	29.3			31.0		
36-45	36.6			40.0		
46-55	26.7			21.9		
56-64	1.2			0.4		
Parental Education						
Dad Missing	8.1			11.7		
Dad < High School	23.1			26.4		
Dad High School Grad	39.4			37.9		
Dad > High School	29.5			24.0		
Mom Missing	5.3			3.9		
Mom < High School	16.9			26.3		
Mom High School Grad	50.9			45.5		
Mom > High School	26.9			24.3		
Risk Factors						
Normal Weight = BMI < 25	33.8			48.9		
Overweight = BMI 25-29	46.2			27.0		
Obese = BMI = 30	20.0			24.1		
Never Smoked	52.2			58.5		
Current Smoker	27.6			22.0		
Former Smoker	20.2			19.5		
Height (inches)		70.807	2.813		64.958	2.802

Table 2 Ordered Logit Model of Childhood Health

	Men		Women	
	Odds-Ratio	95% C.I.	Odds-Ratio	95% C.I.
Low Birth Weight	1.692*	(1.04-2.76)	1.166	(0.82-1.65)
Black	0.971	(0.73-1.30)	1.238	(0.97-1.58)
Mom Married at Birth	0.713	(0.49-1.05)	1.151	(0.86-1.55)
Father's Education				
Missing	1.054	(0.63-1.76)	1.080	(0.74-1.57)
< H.S.	0.929	(0.69-1.26)	1.189	(0.91-1.56)
H.S. Grad	1.000	—	1.000	—
> H.S.	0.721*	(0.54-0.97)	0.990	(0.75-1.31)
Mother's Education				
Missing	1.434	(0.80-2.58)	1.186	(0.66-2.13)
< H.S.	1.280	(0.92-1.77)	1.088	(0.83-1.42)
H.S. Grad	1.000	—	1.000	—
> H.S.	0.902	(0.68-1.20)	0.882	(0.68-1.15)
-2 Log Likelihood	-1300.5		-1631.6	
df	9		9	
N	1303		1445	

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 3 Ordered Logit Model of Self-Reported Adult Health (1999)

	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Education (years)	0.833***	0.862***	0.854***	0.872**	0.917**	0.811***	0.820***	0.846***	0.866***	0.896**
Low Birth Weight	—	1.253	1.145	1.119	1.163	—	0.862	0.856	0.866	0.888
Childhood Health	—	2.130***	2.109***	2.114***	2.158***	—	2.094***	2.073***	2.067***	2.061***
Sociodemographic										
Black	—	—	1.361*	1.226	1.209	—	—	1.971***	1.770***	1.624***
Married	—	—	0.857	0.845	0.925	—	—	0.889	0.887	1.007
Age										
19-25	—	—	0.898	0.928	1.077	—	—	1.169	1.195	1.265
36-45	—	—	1.453**	1.358*	1.298*	—	—	1.699***	1.617***	1.584***
46-55	—	—	2.147***	1.966***	1.773***	—	—	2.298***	2.110***	2.065***
56-64	—	—	1.731	1.320	1.699	—	—	2.181	1.792	2.282*
Dad Missing	—	—	—	1.968*	2.113**	—	—	—	1.278	1.227
Dad < H.S.	—	—	—	1.472**	1.372*	—	—	—	1.448**	1.341*
Dad > H.S.	—	—	—	1.208	1.248	—	—	—	0.961	0.974
Mom Missing	—	—	—	0.968	0.872	—	—	—	1.022	0.969
Mom < H.S.	—	—	—	1.143	1.120	—	—	—	1.043	1.049
Mom > H.S.	—	—	—	0.953	0.968	—	—	—	1.019	1.024
Risk Factors										
Overweight	—	—	—	—	1.279*	—	—	—	—	1.314*
Obese	—	—	—	—	2.433***	—	—	—	—	2.522***
Current Smoker	—	—	—	—	2.137***	—	—	—	—	1.669***
Former Smoker	—	—	—	—	1.308	—	—	—	—	0.884
Height (inches)	—	—	—	—	0.951**	—	—	—	—	1.01
-2 Log Likelihood	-1654.7	-1591.6	-1570.7	-1562.9	-1525.2	-1875.5	-1796.2	-1749.7	-1744.5	-1659.8
df	1	3	9	15	20	1	3	9	15	20
N	1329	1329	1329	1329	1325	1476	1476	1476	1476	1428

* $p<.05$, ** $p<.01$, *** $p<.001$

Table 4 Binary Logit Model of Work-Limiting Disability (1999)

	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Education (years)	0.868**	0.880*	0.874**	0.836**	0.863*	0.905*	0.916	0.902	0.921	0.955
Low Birth Weight	—	1.078	1.030	1.090	1.047	—	0.638	0.650	0.684	0.704
Childhood Health	—	1.241	1.216	1.228	1.222	—	1.511***	1.542***	1.540***	1.446***
Sociodemographic										
Black	—	—	0.928	1.015	1.033	—	—	0.514**	0.505**	0.521**
Married	—	—	0.624*	0.631*	0.688	—	—	0.592**	0.571**	0.653*
Age										
19-25	—	—	1.047	1.020	1.030	—	—	1.204	1.237	1.212
36-45	—	—	2.224**	2.444**	2.302**	—	—	2.047**	1.968**	1.907*
46-55	—	—	2.230**	2.616**	2.215*	—	—	2.195**	1.981*	2.116*
56-64	—	—	1.554	2.583	2.294	—	—	*a*	*a*	*a*
Dad Missing	—	—	—	1.197	1.194	—	—	—	1.041	1.071
Dad < H.S.	—	—	—	0.627	0.624	—	—	—	1.956**	1.911*
Dad > H.S.	—	—	—	1.335	1.314	—	—	—	0.954	1.030
Mom Missing	—	—	—	0.799	0.767	—	—	—	0.353	0.391
Mom < H.S.	—	—	—	0.833	0.833	—	—	—	0.777	0.793
Mom > H.S.	—	—	—	0.992	1.036	—	—	—	1.000	0.902
Risk Factors										
Overweight	—	—	—	—	0.804	—	—	—	—	0.508*
Obese	—	—	—	—	1.073	—	—	—	—	1.717*
Current Smoker	—	—	—	—	1.646	—	—	—	—	1.788*
Former Smoker	—	—	—	—	2.439**	—	—	—	—	1.722*
Height (inches)	—	—	—	—	0.927*	—	—	—	—	1.078
-2 Log Likelihood	-358.2	-356.6	-349.6	-346.2	-337.9	-413.9	-405.3	-393.7	-388.1	-366.2
df	1	3	9	15	20	1	3	8	14	19
N	1329	1329	1329	1329	1325	1476	1476	1469	1469	1421

* $p<.05$, ** $p<.01$, *** $p<.001$; *a* - dropped due to collinearity

Table 5 Binary Logit Model of Physician-Diagnosed Chronic Condition

	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Education (years)	0.935*	0.957	0.920**	0.924*	0.932	0.921**	0.936*	0.944	0.941	0.985
Low Birth Weight	—	1.487	1.584	1.660	1.761*	—	1.267	1.299	1.302	1.401
Childhood Health	—	1.485***	1.490***	1.496***	1.494***	—	1.491	1.484***	1.482***	1.457***
Sociodemographic										
Black	—	—	0.753	0.671*	0.655*	—	—	1.011	1.002	0.927
Married	—	—	0.815	0.812	0.819	—	—	0.709**	0.715**	0.818
Age										
19-25	—	—	0.847	0.859	0.899	—	—	1.328	1.320	1.549
36-45	—	—	1.426*	1.400	1.336	—	—	1.570**	1.564**	1.584**
46-55	—	—	2.494***	2.497***	2.398***	—	—	2.242***	2.214***	2.189***
56-64	—	—	6.742***	6.683***	6.939***	—	—	3.011	2.831	3.121
Dad Missing	—	—	—	2.744***	2.817***	—	—	—	1.136	1.041
Dad < H.S.	—	—	—	1.134	1.084	—	—	—	1.200	1.052
Dad > H.S.	—	—	—	1.092	1.128	—	—	—	1.073	1.076
Mom Missing	—	—	—	0.676	0.643	—	—	—	0.999	0.911
Mom < H.S.	—	—	—	1.068	1.084	—	—	—	1.000	0.957
Mom > H.S.	—	—	—	1.217	1.218	—	—	—	1.162	1.108
Risk Factors										
Overweight	—	—	—	—	0.870	—	—	—	—	1.500**
Obese	—	—	—	—	1.767**	—	—	—	—	3.118**
Current Smoker	—	—	—	—	1.086	—	—	—	—	1.674***
Former Smoker	—	—	—	—	1.552**	—	—	—	—	1.870***
Height (inches)	—	—	—	—	1.004	—	—	—	—	1.031
-2 Log Likelihood	-793.5	-778.2	-755.8	-748.4	-733.7	-938.9	918.0	-900.9	899.7	-834.0
df	1	3	9	15	20	1	3	9	15	20
N	1329	1329	1329	1329	1325	1476	1476	1476	1476	1428

* $p<.05$, ** $p<.01$, *** $p<.001$

Table 6 Binary Logit Model of Physician-Diagnosed Cardiovascular Disease

	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Education (years)	0.941	0.957	0.923*	0.932	0.922	0.888**	0.896**	0.926	0.939	0.930
Low Birth Weight	—	2.173**	2.211**	2.324**	2.766***	—	1.238	1.272	1.285	1.245
Childhood Health	—	1.269**	1.249*	1.246*	1.239*	—	1.208*	1.159	1.155	1.126
Sociodemographic										
Black	—	—	1.024	0.921	0.877	—	—	2.281***	2.167***	1.973***
Married	—	—	0.857	0.851	0.847	—	—	0.84	0.839	0.916
Age										
19-25	—	—	0.405	0.414	0.447	—	—	0.998	1.017	1.181
36-45	—	—	1.889**	1.847**	1.777*	—	—	1.984***	1.907**	2.030**
46-55	—	—	3.185***	3.138***	3.362***	—	—	3.520***	3.387***	3.533***
56-64	—	—	10.527***	9.879***	12.475***	—	—	6.470*	5.839*	6.616*
Dad Missing	—	—	—	2.925**	3.084***	—	—	—	1.126	0.915
Dad < H.S.	—	—	—	1.195	1.098	—	—	—	1.230	1.070
Dad > H.S.	—	—	—	1.094	1.175	—	—	—	1.288	1.304
Mom Missing	—	—	—	0.488	0.414	—	—	—	1.085	1.029
Mom < H.S.	—	—	—	1.120	1.149	—	—	—	1.035	0.980
Mom > H.S.	—	—	—	1.194	1.184	—	—	—	0.775	0.737
Risk Factors										
Overweight	—	—	—	—	0.888	—	—	—	—	1.541*
Obese	—	—	—	—	2.856***	—	—	—	—	2.685***
Current Smoker	—	—	—	—	0.811	—	—	—	—	0.739
Former Smoker	—	—	—	—	1.101	—	—	—	—	1.241
Height (inches)	—	—	—	—	1.036	—	—	—	—	1.003
-2 Log Likelihood	-525.8	-519.3	-496.4	-491.1	-468.9	-576.9	-574.0	-539.9	538.5	-500.8
df	1	3	9	15	20	1	3	9	15	20
N	1329	1329	1329	1329	1325	1476	1476	1476	1476	1428

* $p<.05$, ** $p<.01$, *** $p<.001$

Table 7 Binary Logit Model of Physician-Diagnosed Diabetes

	Men					Women				
	1	2	3	4	5	1	2	3	4	5
Education (years)	0.972	0.972	0.951	0.963	0.943	0.997	1.023	1.037	1.109	1.200*
Low Birth Weight	—	1.419	1.351	1.464	1.686	—	1.227	1.263	1.309	1.382
Childhood Health	—	0.962	0.952	0.948	0.919	—	1.669***	1.636***	1.632***	1.582**
Sociodemographic										
Black	—	—	1.084	1.126	1.072	—	—	1.148	0.778	0.685
Married	—	—	0.937	0.916	0.911	—	—	0.758	0.781	0.877
Age										
19-25	—	—	1.814	1.798	2.179	—	—	1.312	1.409	1.403
36-45	—	—	2.938*	2.927*	2.871	—	—	1.956	1.705	1.455
46-55	—	—	4.537**	4.596**	4.985	—	—	2.201	1.778	1.429
56-64	—	—	*a*	*a*	*a*	—	—	*a*	*a*	*a*
Dad Missing	—	—	—	1.143	1.189	—	—	—	1.486	1.620
Dad < H.S.	—	—	—	0.659	0.604	—	—	—	1.397	1.174
Dad > H.S.	—	—	—	0.939	1.023	—	—	—	0.976	0.988
Mom Missing	—	—	—	0.747	0.641	—	—	—	2.270	1.505
Mom < H.S.	—	—	—	0.804	0.777	—	—	—	2.133*	1.838
Mom > H.S.	—	—	—	0.556	0.547	—	—	—	0.958	0.819
Risk Factors										
Overweight	—	—	—	—	0.936	—	—	—	—	2.447
Obese	—	—	—	—	3.045**	—	—	—	—	6.497***
Current Smoker	—	—	—	—	0.647	—	—	—	—	2.482**
Former Smoker	—	—	—	—	0.942	—	—	—	—	2.099*
Height (inches)	—	—	—	—	0.979	—	—	—	—	1.025
-2 Log Likelihood	-193.1	-193.0	-186.8	-185.1	-177.9	-228.4	-222.1	-219.0	-214.5	-191.9
df	1	3	8	14	19	1	3	8	14	19
N	1329	1329	1313	1313	1309	1476	1476	1469	1469	1421

* $p<.05$, ** $p<.01$, *** $p<.001$; *a* - dropped due to collinearity

Table 8 Ordered Logit Model of Self-Reported Health (2001)

	Men	Women
	Odds-ratios	Odds-ratios
Education (years)	0.945	0.981
Low Birth Weight	1.092	1.078
Childhood Health	2.432***	2.141***
Poor Adult Health (1999)	3.319***	4.201***
Socio-Demographic		
Black	1.282	1.406**
Age		
19-25	0.666	0.957
36-45	1.323*	1.338*
46-55	1.468**	1.408*
56-64	1.939	1.391
Married	0.922	0.933
Dad Missing	0.991	0.816
Dad < H.S.	1.075	0.958
Dad > H.S.	0.896	0.967
Mom Missing	0.817	0.852
Mom < H.S.	1.090	1.425**
Mom > H.S.	0.967	0.966
Risk Factors		
Overweight	1.075	1.148
Obese	1.502**	1.598***
Current Smoker	1.226	1.221
Former Smoker	0.912	1.157
Height (inches)	0.993	0.988
-2 Log Likelihood	-1345.8	-1420.1
df	21	21
N	1325	1428

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 9 Binary Logit Model of Health Decline (1999-2001)

	Men	Women
	Odds-ratios	Odds-ratios
Education (years)	0.931	0.964
Low Birth Weight	0.982	0.880
Childhood Health	2.197***	1.791***
Poor Adult Health (1999)	0.325***	0.286***
Socio-Demographic		
Black	1.577	1.176
Age		
19-25	0.598	0.786
36-45	1.117	1.066
46-55	1.098	1.111
56-64	1.805	*a*
Married	1.120	0.855
Dad Missing	0.907	0.743
Dad < H.S.	1.086	1.035
Dad > H.S.	1.123	1.036
Mom Missing	0.889	1.067
Mom < H.S.	1.019	1.411
Mom > H.S.	0.751	0.985
Risk Factors		
Overweight	0.901	0.971
Obese	1.381	1.674**
Current Smoker	1.350	1.407
Former Smoker	1.117	1.027
Height (inches)	1.007	0.996
-2 Log Likelihood	-652.8	-638.4
df	21	20
N	1325	1421

* $p<.05$, ** $p<.01$, *** $p<.001$; *a* - dropped due to collinearity

Table 10 The Effect of Low Birth Weight and Childhood Health on Adult Health Stratified By Parent's Education

	Low Birth Weight				*Childhood Health*			
	Mother's Education		Father's Education		Mother's Education		Father's Education	
	O.R.	(95% C.I.)	O.R.	(95% C.I.)	O.R.	(95% C.I.)	O.R.	(95% C.I.)
				Self-Reported Health 1999 (ordered logit)				
< High School	1.14	(0.67-1.95)	1.33	(0.82-2.17)	1.78***	(1.49-2.14)	1.93***	(1.60-2.33)
High School	0.81	(0.53-1.24)	0.66	(0.40-1.08)	2.08***	(1.79-2.42)	2.02***	(1.74-2.36)
> High School	1.12	(0.55-2.24)	1.02	(0.53-1.97)	2.65***	(2.10-3.36)	2.84***	(2.28-3.54)
				Work Limiting Disability				
< High School	1.93	(0.75-4.98)	1.62	(0.66-3.97)	1.00	(0.73-1.38)	1.31	(0.97-1.76)
High School	0.51	(0.18-1.47)	0.77	(0.23-2.59)	1.45***	(1.17-1.80)	1.29*	(1.00-1.65)
> High School	0.60	(0.14-2.53)	0.41	(0.07-2.29)	1.41	(0.94-2.10)	1.39	(0.98-1.98)
				Any Chronic Disease				
< High School	1.28	(0.72-2.28)	1.24	(0.67-2.31)	1.31*	(1.06-1.61)	1.36**	(1.11-1.66)
High School	1.34	(0.79-2.28)	1.21	(0.70-2.12)	1.57***	(1.35-1.83)	1.55***	(1.31-1.82)
> High School	2.54**	(1.31-4.95)	2.43*	(1.23-4.79)	1.64***	(1.31-2.05)	1.49***	(1.19-1.87)
				Cardiovascular Disease				
< High School	1.78	(0.93-3.42)	2.38**	(1.26-4.52)	1.24	(0.96-1.60)	1.13	(0.89-1.42)
High School	2.08*	(1.10-3.95)	1.44	(0.65-3.20)	1.11	(0.90-1.37)	1.23	(0.98-1.53)
> High School	1.53	(0.59-3.99)	2.28	(0.98-5.33)	1.22	(0.89-1.69)	1.05	(0.75-1.48)
				Decline in Self-Reported Health (1999-2001)				
< High School	1.45	(0.77-2.70)	0.74	(0.37-1.49)	1.32*	(1.06-1.65)	1.41**	(1.14-1.75)
High School	0.98	(0.59-1.61)	1.33	(0.78-2.62)	1.57***	(1.35-1.84)	1.53***	(1.29-1.82)
> High School	0.81	(0.36-1.82)	0.72	(0.32-1.61)	1.27	(0.97-1.65)	1.41**	(1.10-1.81)

* P< .05, ** P<.01, *** P< .001

Table 11 Differential effect of BMI and Smoking on Adult Health by Low Birth Weight Status

	Body Mass Index					
	< 25		25-29		= 30	
	O.R.	(95% C.I.)	O.R.	(95% C.I.)	O.R.	(95% C.I.)
	Self-Reported Health 1999 (Ordered Logit)					
LBW	1.00	—	1.18	(0.60-2.33)	2.20	(0.98-4.97)
Non-LBW	1.00	—	1.32***	(1.12-1.56)	2.48***	(2.01-3.05)
	Any Chronic Disease					
LBW	1.00	—	1.07	(0.46-2.48)	3.32*	(1.17-9.44)
Non-LBW	1.00	—	1.18	(0.94-1.46)	2.44***	(1.92-3.08)
	Cardiovascular Disease					
LBW	1.00	—	2.38	(0.67-8.37)	4.79*	(1.24-18.40)
Non-LBW	1.00	—	1.13	(0.82-1.57)	2.73***	(2.00-3.72)
	Smoking History					
	Never		Former		Current	
	O.R.	(95% C.I.)	O.R.	(95% C.I.)	O.R.	(95% C.I.)
	Self-Reported Health 1999 (Ordered Logit)					
LBW	1.00	—	0.96	(0.41-2.25)	2.00	(0.90-4.44)
Non-LBW	1.00	—	1.05	(0.86-1.28)	1.81***	(1.49-2.19)
	Any Chronic Disease					
LBW	1.00	—	1.35	(0.51-3.58)	2.66*	(1.09-6.51)
Non-LBW	1.00	—	1.69***	(1.35-2.12)	1.32*	(1.04-1.68)
	Cardiovascular Disease					
LBW	1.00	—	0.45	(0.14-1.47)	0.81	(0.27-2.43)
Non-LBW	1.00	—	1.19	(0.89-1.59)	0.77	(0.55-1.08)

* P< .05, ** P<.01, *** P< .001