Gender Differences in the Link between
Childhood Socioeconomic Position and Heart Attack Risk in Adulthood

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Numerous studies have shown that childhood socioeconomic position (SEP) is predictive of disease risk in later life, with those from disadvantaged backgrounds experiencing the greatest likelihood of poor health outcomes (e.g., O'Rand and Hamil-Luker 2005). Most research on the association between early life environments and adult health outcomes, however, is based on middle-aged male populations (Barker et al. 2001; Blane et al. 1996). If women are included in samples, the usual approach is to "control" for gender, ignoring the possibility that the complex interplay of biologic, social, psychological, economic, and behavioral processes that shape health trajectories may operate differently for men and women. Furthermore, most research in this area has focused on the correlation of childhood factors with various adult health outcomes, paying insufficient attention to the pathways between childhood risks and specific adult disorders. This paper contributes to the growing body of knowledge of the link between childhood SEP and adult health by examining gender differences in heart attack risk trajectories and the mechanisms by which early life exposures affect future disease risk.

Why Study Heart Attacks?

We choose to study the relationship between childhood conditions, adult pathway mediators, and heart attack risk trajectories for several reasons. First, we recognize that different diseases and sub-diseases have varying configurations of causal variables (Galobardes et al. 2004). In particular, the contribution of child and adult SEP varies across diseases, with the relationship between childhood disadvantage and later poor health especially strong for cardiovascular disease (CVD) and mortality (Beebe-Dimmer et al. 2004; Hayward and Gorman 2004; Naess et al. 2004; Pensola and Martikaninen 2003; Singh-Manoux et al. 2004; Steptoe and Marmot 2002). Because CVD is a group of diseases that vary in incidence, risk factors, causes, treatments, epidemiology, and mortality rates, we focus on one form of CVD: heart attacks.

Second, heart attacks occur in sufficient numbers to warrant investigation and attention. The American Heart Association (2005) estimates that in 1999-2002 over 7 million Americans had experienced a heart attack. Since 1900, heart attacks and related CVD have been the leading cause of death among American women and men every year but 1918 (AHA 2005). Only 13 percent of American women consider CVD their greatest health risk, but more deaths are caused by CVD than by the next six causes of death combined (AHA 2005). Thus, a better understanding of gender differences in life course risk factors for heart attack is needed.

Third, there is evidence that risk factors for poor health differ between men and women, and this is particularly true for CVD and mortality risks (e.g. Chandola et al. 2004; Wister and Gee 1994). Although not the main focus of empirical investigation, a small number of studies have documented gender differences in the contribution of childhood SEP to adult health. In the 1946 British birth cohort followed until age 55, childhood SEP more strongly predicted adult mortality among women than men (Kuh et al. 2002). Similarly, Rahkonen et al. (1997) found that deleterious effects of economic problems during childhood and father's low education were stronger for women than men. Clausen et al. (2003) also found that women's risk for CVD was more strongly related to childhood living environments than men's. Gender differences in the pathway from childhood circumstances to adult health trajectories, however, are not well understood.

Finally, diseases of the heart serve as a good example of health problems that are influenced by exposures during fetal development, childhood, adolescence, and adulthood. CVD begins in utero, evolves through childhood, and emerges in middle and older age (Li et al. 2003; Wizemann and Pardue 2001). For example, poor maternal nutrition has been shown to slow fetal development and growth in childhood, leading to accumulated risks of increased blood pressure in adolescence, which in turn increases the likelihood of heart attacks in adulthood (Montgomery et al. 2000). A growing body of evidence illuminates the multiple social, environmental, biologic, and genetic factors that contribute to heart attack risk over the life course. Although this accumulating knowledge has contributed to a steady decline in mortality rates from heart disease over the last decade, we have yet to understand fully the extent to which childhood environments contribute to heart attack risk trajectories and whether these effects differ for men and women.

Theories Linking Childhood Socioeconomic Position to Poor Adult Health

There are two main explanations for the relationship between early life exposures and health trajectories across adulthood. First, the notion of biological imprinting, as theorized by David Barker's work, (1992, 1993), suggests that factors during early childhood, especially during fetal development, make a biological imprint on the human organism in a way that makes it more susceptible to illness later in life. Also known as the *latency model*, this theory proposes that early life exposures can program long-term or permanent changes in biological and behavioral systems (Hertzman 1999; Halfon and Hochstein 2002). Barker (1999), for example, suggests that the fetus may adapt to malnutrition or an excess of maternal stress hormones by permanently altering cardiovascular, endocrine, and metabolic systems in ways that promote atherosclerosis later in life. Hertzman (1999) hypothesizes that conditions in utero and early life may also affect the structure and functioning of the central nervous system, which interacts with the immune, hormone, and clotting systems to increase or decrease susceptibility to disease. In support of the latency model, several studies using primates and rodents have found that early environmental exposures alter physiological regulator systems in permanent ways (see McEwen and Seeman, 1999, for a review). In humans, low birth weight and other measures of slow prenatal growth have been found to be inversely associated with blood pressure, serum cholesterol level, and CVD mortality in later life (Davey Smith et al. 2000; Martyn et al. 1996; Miura et al. 2001; Rich-Edwards et al. 1997).

In contrast to the latency model that links early exposures to adult health outcomes independently of intervening life circumstances, the accumulation of risk or *pathway model* proposes that childhood circumstances set individuals on diverse social, economic, and behavioral trajectories that in turn affect health. Early social disadvantage initiates a sequence of negative influences that together lead to illness or premature death in adulthood. Ben-Shlomo and Kuh (2002) have discussed the pathway model in terms of "chains of risk" whereby one adverse exposure tends to lead to another. Childhood experiences of poverty, inadequate housing, and stressful family conditions, for example, may lead to unhealthy behaviors and poor school performance in adolescence, limited job opportunities and income in adulthood. Inadequate socioeconomic resources and stressful life circumstances in adulthood in turn are risk factors for morbidity and mortality.

It is artificial, however, to juxtapose these as competing explanations because they probably work together in complicated ways. Barker et al. (2001), for example, documented the interaction between childhood and adult conditions on predicting coronary heart disease (CHD). Thinness at birth was related to men's CHD only in the presence of low adult social class. Thus, chronic disease may be the long-term outcome of childhood conditions and experiences beginning in utero combined with cumulative exposures across adulthood (Blackwell, Hayward and Crimmins 2001). Susceptibility to diseases is embedded in individuals' biological makeup, but diseases are expressed and maintained in particular social, economic, and cultural environments (Halfon and Hochstein, 2002; Power and Hertzman 1997).

Gender Differences in Cardiovascular Disease

CVD provides a good example of how sex, a key biological factor, interacts with the social environment over the life course to influence susceptibility to disease. Women and men differ in the development, presentation, course, and outcome of CVD (Vaccarino and Mallik 2004). Women experience a lower risk of heart diseases than men, showing symptoms about 10 years later than men (AHA 2005). This is due in part to sex specific genetic mechanisms, such as ovarian hormones that have a protective effect on cardiovascular risk factors and functions, but also gender difference in exposure to and learned coping with environmental stressors (Roberts 2004). The same risk factors that predict CVD for men do so for women, but the relative strength for some factors is gender specific. For example, smoking, diabetes, low HDL-C (high-density lipoprotein cholesterol), and elevated triglyceride levels confer more risk in women than men (Roberts 2004).

Although women's CVD mortality rate lags behind that of men by a decade (AHA 2005), women experience a higher risk of poor outcomes from CVD than men (Schoenberg, Peters, and Drew 2003). After a heart attack, women are more critically ill than men, suffer higher rates of complications, and have higher mortality, especially in the short term (Vaccarino and Mallik 2004). This is due in part to women's older age at clinical manifestation, greater number of co-morbidities, and the competing social demands women face when experiencing health problems (Dong et al. 1998; Schoenberg, Peters and Drew, 2003). Looking beyond personal attributes, however, gender differences in heart attack diagnoses and outcomes are also due to availability of health care, government policies, and gender bias in healthcare delivery (McKinlay 1996). Thus, both internal environments (e.g., sex differences in the size of coronary arteries) and external environments (e.g., hospitals' lower likelihood of providing women lifesaving treatments) influence men and women's risk for heart attack as they age.

This paper recognizes the complex interplay of structural, behavioral, psychosocial, biological and genetic forces that shape individuals' health trajectories. Based on the theoretical frameworks outlined above, we hypothesize that people vary in their susceptibility to heart attacks and that these latent differences emerge early in life. We hypothesize that genetic, biologic, and early environmental forces set the stage for future economic, behavioral, and psychosocial pathways that influence the expression of inherent disease risks. Our analyses try to model this complex layer of intertwined forces, both latent and path-specific, that produce heterogeneity in health outcomes as people age. By conducting our analyzes separately for men and women, we highlight gender differences and similarities in the contribution of childhood SEP and human, social, and health capital to heart attack risks over time. We also acknowledge the heterogeneity of heart attack trajectories among women and men, and model some of the processes that lead to this diversity.

Data and Measures

To examine the association between and mechanisms linking childhood SEP and heart attack risk, we analyze data from the Health and Retirement Study (HRS). The HRS is a national panel study launched in 1992 to collect information on the physical, mental, social, and financial well-being of Americans over the age of 50. Through 2002, respondents have biennially reported stability and changes in their health and marital statuses, employment, and income, with proxy interviews conducted after death. Our sample includes 5166 women and 4594 men born between 1931 and 1941. Thus, respondents were 51 to 61 years old in 1992 when interviews began and aged into their 60s and early 70s over the following decade. We selected the HRS because it is one of the rare data sets that include information on childhood living conditions and health outcomes during young, middle, and older adulthood.

The dependent variable in this analysis is a series of repeated measures of whether or not respondents had experienced a heart attack by time *t*. For each data collection point between 1992 and 2002, we create a dichotomous indicator *heart attack*$_t$ that equals 1 if respondents report that a doctor had ever told them they had had a heart attack by that survey year, 0 otherwise. Models include a linear measure for time, with time=0 in 1992 to time=5 in 2002.

To test the hypothesis that early environments differentially influence men and women's heart attack risk trajectories, we examine seven characteristics of respondents' early life environments. In 1998, respondents were asked to think about their home life from birth to age 16 and report their father's occupation, mother's education, family's socioeconomic position, father's absence, experience of financial difficulties, geographic mobility due to financial problems, and father's unemployment. As explained more fully in O'Rand and Hamil-Luker (2005), we used these measures and latent class cluster analysis to identify distinct groups of respondents with differential exposure to childhood disadvantage. Fit statistics from the latent class cluster models indicated that there were three basic groups into which respondents fell. First, the *fatherless* were respondents who never lived with their father while growing up. Because our sample was born between 1931 and 1941, many fathers probably died during World War II. The *early disadvantaged* were most likely to have low-educated mothers, unemployed fathers, and poor families or origin. Respondents in the *early advantaged* cluster were the least likely to experience poverty and the problems associated with it.

Demographic control variables include *age* in 1992 and race (*white*=1; 0 otherwise). Our human capital indicators include measures for *education* in 1992 (high school dropouts and high school graduates compared to those with at least some college), and income measured at each survey year ($low\ income_t$=1 if total household income, adjusted for family size, is less than half the median income).[1] We include yearly dichotomous indicators of whether or not respondents were working for pay at time t ($employed_t$=1, 0 otherwise). In 1992, 1994, and 1996, respondents reported whether they had been exposed to dangerous chemicals or other hazards at work. Because hazardous or stressful working conditions can negatively affect health (Marmot et al. 1997), we

[1] Models not shown here included logged household income, adjusted for family size, and education measured as the number of years of completed schooling. These measures were not significant predictors of heart attack risk, indicating that it is not each incremental year of schooling or dollar of income that influences health, but being at the margins of the education/income distribution.

compare those who reported any exposure (*hazardous occupation*=1; 0 otherwise) to those who had not worked in such an environment. We also include time-varying measures of whether or not respondents were employed in *service occupations*$_t$ and *lacked health insurance*$_t$.

Because social relationships and support are key risk factors for health (House 2002), our next group of covariates includes measures of social capital. We compare those who reported in 1992 that they were very or somewhat *dissatisfied with their friendships* to those who reported they were very or somewhat satisfied. Because the strains of marital conflict and dissolution may undermine physical health (Williams and Umberson 2004), we compare those who had who were *ever divorced* in 2002 to all others and compared those who were very or somewhat *dissatisfied with their marriage* to all others. Previous research has shown that difficulty managing work and family responsibilities is linked to poor health and health damaging behaviors (Frone, Russell and Cooper 1997). To examine potential gender differences in the health effects of work-family responsibilities, our models include dichotomous repeated measures of whether or not respondents were *working parents*$_t$, defined as having children at home and being in the labor force.

The final group of covariates measures health capital. Because adult co-morbidities are known risk factors for CVD, we use data from each survey year to compare those who reported they had ever had *cancer*, *diabetes*, and *high blood pressure* to those who had not experienced these health problems. Early disadvantage may increase later disease risk in part because children from lower socioeconomic backgrounds adopt detrimental health behaviors and attitudes that persist from childhood into adulthood and increase CVD risk (Aboderin et al. 2002; Lawlor et al. 2004). Our models test this hypothesis by including time-varying indicators of key health behaviors that are risk factors for cardiovascular disease: *current smoker*$_t$, *does not exercise*$_t$, and *obese*$_t$, defined as a body mass index of 30 or greater.

Methods

Because of the nested structure of our data with repeated observations clustered within HRS respondents over time, we use non-parametric hierarchical models. As shown by Vermunt and Van Kijk (2001), these models are similar to traditional hierarchical models (also known as multilevel, growth curve, and random coefficient models), but offer many methodological advantages that allow us to test our theoretical framework. Foremost, non-parametric hierarchical models allow us to handle dependent observations in longitudinal data without making strong assumptions about the distribution of random coefficients (Vermunt 2003). Because non-parametric hierarchical models do not assume that random effects come from a multivariate normal distribution, we can analyze a repeatedly measured dichotomous dependent variable in a relatively short computational time.[2]

We hypothesize that individuals differ in their inherent susceptibility to heart attacks and that this heterogeneity begins early in life. Because disease risk cannot be observed directly but only inferred from observable variables, our models must estimate and test relationships between latent variables (such as genetic susceptibility) and manifest indicators (whether or not respondents reported a heart attack by 1992, 1994, 1996, 1998, 2000, and 2002). Non-parametric hierarchical models are a form of latent class (LC) analysis that makes it possible to reveal and statistically test a latent structure for explaining the data (Bouwmeester, Sijtsma, and Bermunt 2004). Also known as multilevel LC models, they assume there are a finite number of discrete groups who differ in a latent trait or behavior pattern (Wedel and DeSarbo 1994). Using this approach, we examine heterogeneity in latent susceptibility to heart attack by testing for and identifying groups of individuals that differ in the timing and experience of heart attacks. Because cross-sectional population-based studies reveal gender differences in the development and course of heart attacks,

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[2] We attempted to verify our results by estimating the models with the standard hierarchical approach in SAS using GLIMMIX. Because the procedure incorrectly computes how much memory is required in repeated measures models, the models would not converge even when specifying the largest memory size allowed.

we test whether men and women are distinguished by different heart attack risk trajectories as they age. We hypothesize that childhood SEP has an important influence on the formation of the latent classes. More specifically, those disadvantaged early in life may have heart attack risk trajectories characterized by higher incidence, early age of onset, and steeper rates of rising risk over time. Multilevel LC models may include covariates that predict latent cluster membership, making it empirically possible to test the influence of childhood SEP on the formation of latent classes. Thus, the first stage of analysis tests the proposition derived from the latency model that early unobservable and observable measures of early life conditions will influence heart attack risk trajectories over the life course.

In the second stage of analysis, we test the pathway model by examining the extent to which the accumulation of human, social and health capital decreases heart attack risks over time. Like traditional hierarchical models, non-parametric hierarchical models allow time-varying predictors of a dependent variable measured repeatedly over time. Unlike the standard approach, however, multilevel LC regression models relax the traditional assumption that the same model holds for all cases. Thus, in addition to examining gender differences in the effects of adult pathway variables on heart attack risk over time, we can test whether identified subgroups among women and among men differ with respect to the estimated regression parameters.

We are interested in modeling the probability density of observing a particular set of **y** values (whether or not individual *i* reported a heart attack at time *t*) given a particular set of covariate measures of childhood SEP (denoted by $\boldsymbol{z}^c$) and a particular set of path-specific and time-varying predictor values (denoted by $\boldsymbol{z}^p{}_t$), that is $f(\mathbf{y}|\, \boldsymbol{z}^c\, \boldsymbol{z}^p{}_t)$. The non-parametric hierarchical model may be denoted as (Vermunt and Magidson 2004):

$$f(\mathbf{y}|\, \boldsymbol{z}^c\, \boldsymbol{z}^p{}_t) = \sum_{\mathbf{x}} \pi\, (\mathbf{x}|\, \boldsymbol{z}^c) \prod_{t=1}^{T} f(y_t|\, \boldsymbol{x}\, \boldsymbol{z}^p{}_t).$$

Here, π (x| z^c) is the probability of being in a particular latent class given observed values for the vector of covariate measures of childhood SEP. The distribution of scores for the dependent variable y_t given a person's values on the predictor variables $z^p{}_t$ and latent class membership x is denoted by f(y_t| x $z^p{}_t$). In this model specification, latent classes differ with respect to their underlying susceptibility to heart attacks as well as to the degree to which measures of human, social, and health capital influence risk of heart attack. Using maximum likelihood estimation, the model approximates the distribution of random coefficients by a limited number of mass points that identify latent classes. The maximum likelihood estimator is obtained by increasing the number of latent classes until a saturation point is reached and model fit no longer improves (Vermunt and Van Dijk 2001). Various fit statistics, such as log likelihood and Bayes Information Criteria, allow researchers to test which model provides the best fit to the data.

Because sample selection is a major problem in panel studies of health outcomes, handling of missing data is a key issue in this analysis. Respondents lost-to-follow up tend to have characteristics predictive of poor health, resulting in samples biased toward healthier older survivors. To address this problem, we use multiple imputation (MI) procedures to produce consistent, asymptotically efficient, and asymptotically normal estimates (Allison 2002). First, using the variables discussed above, we impute missing data five times to generate five complete data sets. We then analyze these data sets, combining results to produce inferential results. Because MI represents a random sample of the missing values, it results in valid statistical inferences that properly reflect the uncertainty due to missing values (Rubin 1987). Results derived from data generated through MI did not substantially differ from results obtained using data with missing values for the dependent variables, covariates, and predictors.

Results

Table 1 presents descriptive statistics for the variables used in our analyses, illustrating gender differences and similarities in the bivariate relationships between childhood SEP and human, social, and health capital in adulthood. In 1992, approximately 8 percent of men of an average age of 56 had experienced a heart attack. By 2002, 15-18 percent had. For men, experiencing socioeconomic advantage in childhood is not associated with a lower likelihood of having a heart attack in adulthood. In contrast, fatherless and early disadvantaged women have a greater risk of heart attack in their 50s than early advantaged women. Over the decade, the absolute size of the gap between the three groups increased. For both men and women, childhood advantage is associated with higher education and income and reduced risk of hazardous occupations, diabetes, high blood pressure, and lack of exercise. Women who grew up without a father are more likely to have experienced divorce or be in a dissatisfying marriage. Marriage trajectories of fatherless men, however, are indistinguishable from those who grew up with a father.

INSERT TABLE ONE ABOUT HERE.

Next, we examine heart attack risk trajectories for the average HRS man and woman, displayed graphically in Figures 1 and 2 as the thick, bold lines. Both men's and women's risk of heart attack increases as they age. At all time points, men are more likely to have experienced a heart attack than women. Figures 1 and 2 also depict subgroup trajectory differences among men and women as identified in non-parametric hierarchical models. Based on BIC statistics, the best fitting model for women specified two latent classes. Ninety-four percent of the women in our sample fall into the "low risk" cluster, experiencing virtually no risk of heart attack as they aged into their early 70s. In contrast, 6 percent of the sample is clustered in the "increasing risk" class. Approximately 30 percent of this cluster reported a heart attack in 1992; ten years later, almost all women had.

As shown in Figure 2, non-parametric hierarchical models identified three discrete groups of men with distinctive heart attack risk trajectories. Similar to HRS women, 76 percent of the sample of men was at low risk of heart attack over the decade. The increasing risk trajectory among women is also identified among men. Comprised of 13 percent of the sample, none of the men in this cluster reported a heart attack in 1992. Ten years later, 75 percent had experienced a heart attack. The final identified trajectory, which we label high risk, includes 11 percent of the sample of men. This trajectory is characterized by early age of heart attack onset (75 percent had a heart attack by their 50s), and the highest probability of experiencing a heart attack.

INSERT FIGURES 1 AND 2 ABOUT HERE.

Figures 1 and 2 highlight the importance of identifying heterogeneity in heart attack risk trajectories among women and men as they age. If we only examined the average risk across all women, for example, we would conclude that women have a low risk of heart attack. Indeed, over 85 percent of the 1931-1941 cohort had not experienced a heart attack by 2002. In contrast, the specification of latent classes allows us to identify a group of women who have an alarming risk for heart attack. Similarly for men, the varying age of onset and likelihood of heart attack across subgroups is masked by the overall trajectory.

Next, we use a non-parametric hierarchical model to examine latent and path-specific mechanisms among women that contribute to the diverse heart attack risk trajectories depicted in Figure 1. Table 2 displays the maximum likelihood parameter estimates from the model predicting heart attack risk trajectories as a function of women's childhood SEP and human, social, and health capital measured in adulthood. The first panel includes estimated coefficients from a logit model that tests whether childhood SEP predicts women's membership in one of the two clusters. Large positive and statistically significant numbers are predictive of membership in that cluster. To obtain odds ratios, the parameter estimates may be exponentiated then the ratio between the two clusters computed. Women from socioeconomically disadvantaged backgrounds, for example, are 44 percent more likely than the early advantaged [exp(.18)/exp(-.18)=1.20/.84=1.44] to belong to the cluster of women with steeply increasing risk of heart attack. Similarly, fatherless women are 56 percent less likely than early advantaged women to have heart attack risk trajectories characterized as low risk. These findings lend support to the latency model that women differ in their underlying propensity to have a heart attack and that childhood SEP is predictive of differing levels of risk.

INSERT TABLE 2 ABOUT HERE.

To test the pathway model, the second panel in Table 2 examines heterogeneity within and across identified clusters by adding predictor variables of heart attack risk over time. The Wald statistic indicates whether the difference in parameter estimates of the dependent variable across clusters is statistically significant. The estimates for time, for example, show that the rate of increase between 1992 and 2002 in reported heart attacks is greater for women in the increasing risk cluster than in the low risk cluster. In contrast, the effects of age, income, and dissatisfied friendships are the same for both clusters, with older women, those with low incomes, and dissatisfaction with their friendships more likely to report a heart attack. Cancer, diabetes, and the role of working mother are associated with elevated heart attack risk for both clusters, but race, hazardous occupations, divorce and dissatisfying marriages do not predict heart attacks for either group. For those in the increasing risk trajectory, low education, employment in the service sector, and lack of health insurance is associated with increased chances of heart attack. This is not the case, however, for women in the low risk trajectory. For these women, high blood pressure and unhealthy lifestyle behaviors increase heart attack risk. These results show that even after controlling for latent population differences in susceptibility to heart attack, measures of human, social, and health capital in adulthood influence the likelihood of experiencing a heart attack. The effects of path-specific mechanisms, however, differ across subgroups of women.

Table 3 presents parameter estimates and standard errors from a non-parametric hierarchical model predicting men's heart attack risk trajectories. As depicted in Figure 2, the model identified three subgroups of men who differ in their underlying susceptibility to heart attack: those at low, increasing, and high risk. The first panel shows covariate effects of childhood SEP predicting latent cluster membership. The statistically non-significant estimates show that men whose childhood was characterized by early socioeconomic advantages are not more likely to belong to the low risk cluster than men who grew up without a father or men who experienced early disadvantages. In contrast to women whose childhood SEP distinguishes subgroups with varying latent heart attack risks, men's childhood SEP is not associated with membership in a particular heart attack risk trajectory. We discuss potential explanations below.

INSERT TABLE 3 ABOUT HERE.

The second panel in Table 3 presents parameter estimates of path-specific influences on the likelihood of men experiencing a heart attack between 1992 and 2002 within latent clusters. As graphically depicted in Figure 2, those in the increasing risk trajectory experience the steepest rise in risk for heart attack over time. Older age and dissatisfying marriages are predictive of greater risk of heart attack for those in the increasing and high risk clusters. After controlling for latent susceptibility and other path-specific predictors, white men and those dissatisfied with their friendships are more likely to experience heart attack among those in the low and high risk trajectories. Service sector employment, lack of health insurance, and diabetes are associated with greater likelihood of heart attack for those in the high risk trajectory. For all clusters of men, employment reduces and hazardous occupations increase the risk of heart attack.

Comparison of Tables 2 and 3 reveal gender differences and similarities in mechanisms influencing the likelihood of heart attack within latent clusters. Although low income is associated with an increased risk of heart attack for both clusters of women, it is not associated with men's risk of heart attack after controlling for latent susceptibility. Working mothers are 15 to 22 percent [exp(.14) and exp (.22)] more likely to have a heart attack than their counterparts, but employed men who have children at home have a lower risk of having a heart attack. Working fatherhood is the predictor most strongly related to reduced chances of heart attack, with the protective effect holding true across clusters. Similar to women, men with low educations in the increasing risk cluster are at greater risk of heart attack than better-educated men. This is not the case, however, for men in the low and high risk trajectories. Also similar to women, high blood pressure and unhealthy lifestyle behaviors are particularly predictive of heart attacks for those in the low risk trajectory. For both men and women, the accumulation of human, social, and health capital influence the presentation of heart attack in middle and older age even after controlling for latent differences in heart attack risk. The size and direction of the effects, however, differ among and between men and women.

Discussion and Conclusions

We draw four main conclusions from our findings. First, heterogeneity in a population's risk of heart attack is a function of both latent differences in underlying susceptibility to disease and the result of an accumulating cascade of life experiences and stressors from conception to death that mold health trajectories. Such diversity cannot be adequately captured in a single model of the average risk of heart attack over time. By using methods that model both latent and path-specific influences on heart attack risk, we detected distinct heart attack risk trajectories within samples of HRS women and HRS men, revealing clusters who were at low, increasing, and high risk of heart attack between 1992 and 2002.

Second, early exposure to adverse socioeconomic conditions is associated with reduced life course accumulation of human, social, and health capital. Family and financial difficulties during childhood are predictive of lower education and income, hazardous occupations, lack of exercise, diabetes, and high blood pressure in adulthood, with the relationships stronger and more consistent for women than for men. Childhood conditions are thus linked to a web of pathways that shape socioeconomic and lifestyle events that ultimately influence adult health.

Third, childhood socioeconomic position is predictive of latent differences in heart attack risk trajectories for women, but not men. We find that women who grew up without a father and/or under adverse economic conditions are most likely to experience elevated risk for heart attack in their 50s, 60s, and 70s. Women from socioeconomically advantaged backgrounds, in contrast, are clustered in the low risk heart attack trajectory. For men, childhood SEP does not differentiate among those at low, increasing, and high risk for heart attack.

Our findings are consistent with the handful of studies that document a stronger association between early environment and later disease and mortality risk for women than men. It is yet unclear, however, why women's health trajectories would be more strongly impacted by childhood SEP than men's. There could be biological differences between men and women in the body's encoding and storage of harmful early exposures, but these are not yet well documented and understood. We suggest that women may have greater vulnerability to the effects of early disadvantage because they occupy different structural locations than men. As a group, women are less likely than men to be employed and have high incomes, but more likely to work in low-wage service occupations, be single parents and perform domestic labor. Women are exposed to higher levels of demands and obligations in their social roles and experience more stressful life events (de Vries and Watt 1996). The circumstances of women's childhood may more firmly anchor them in future health trajectories because life course exposures in the realms of education, family, and work further solidify latent differences expressed in early life. In contrast, men's childhood SEP may more weakly impact adult health trajectories because their greater access to power, resources, prestige, and knowledge over the life course renders early exposures less important than proximal resources.

Finally, we conclude that some key risk factors for poor health operate differently for men than for women. In particular, we find that work/family obligations have opposite health effects, a finding consistent with previous research documenting gender differences in the effects of parental responsibilities on health (e.g., Dixon, Dixon, and Spinner 1991). In our sample, employed fathers with children at home are much less likely than their counterparts to report a heart attack by their 60s and 70s. For women, however, combining the roles of employee and mother is associated with elevated risk of heart attack. Our findings may be linked to the stress and illness literature showing that men and women's differential exposures to resources and stressors influence gender differences in illness. As measured by stress hormones and cardiovascular arousal, women more often than men are exposed to sustained psychophysiological stress levels (Lundberg and Parr 2000). When men come home from work in the evening, for example, their blood pressure tends to fall. Women, however, are less likely to show decreases in blood pressure at the end of the workday (Lundberg 1996), perhaps because they perform, on average, two to three times as much housework as men (Coltrane 2000). Chronic and repeated exposure to stress hormones can compromise health through wear and tear on the body, such as damaging tissue lining of the arteries that contribute to atherosclerosis (Lundberg and Parr 2000). Acute stress can also cause platelets in the blood to form clots that can induce heart attacks (Muller et al. 1994). Further work is needed to measure and test whether gender differences in exposure and reaction to stressors help explain why childhood SEP more strongly predicts adult morbidity among women than men.

Although our findings contribute needed empirical research on gender differences in the link between childhood SEP and adult health, there are several limitations in the current study. First, we are unable to examine in detail the complex interplay of biologic, behavioral, and psychosocial processes that shape men and women's health trajectories from infancy into adulthood. If we are to understand how the body acts as a repository of past experiences, we need data that allow us to study the biosocial interactions between socioeconomic environments and genetic predispositions. Data limitations also restrict our ability to measure accurately early life environments. Similar to most research in this area, our findings are based on respondents' recalled account of their childhood conditions. Although adults do seem to be able to accurately recall their parents' SEP at the time of their childhood (Krieger et al. 1998), measurement error may bias estimates. This paper illustrates the use of latent class analysis as one approach to dealing with the limitation of error in recollection of childhood circumstances. By identifying heterogeneity in early life exposures and human, social, and health capital in adulthood, this paper has shown the importance of theorizing and modeling both latent and path-specific influences on heart attack risk trajectories as men and women age.

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Figure 1: Women's Predicted Probability of Heart Attack by Latent Cluster; Health and Retirement Study 1992-2002 (N=5166)

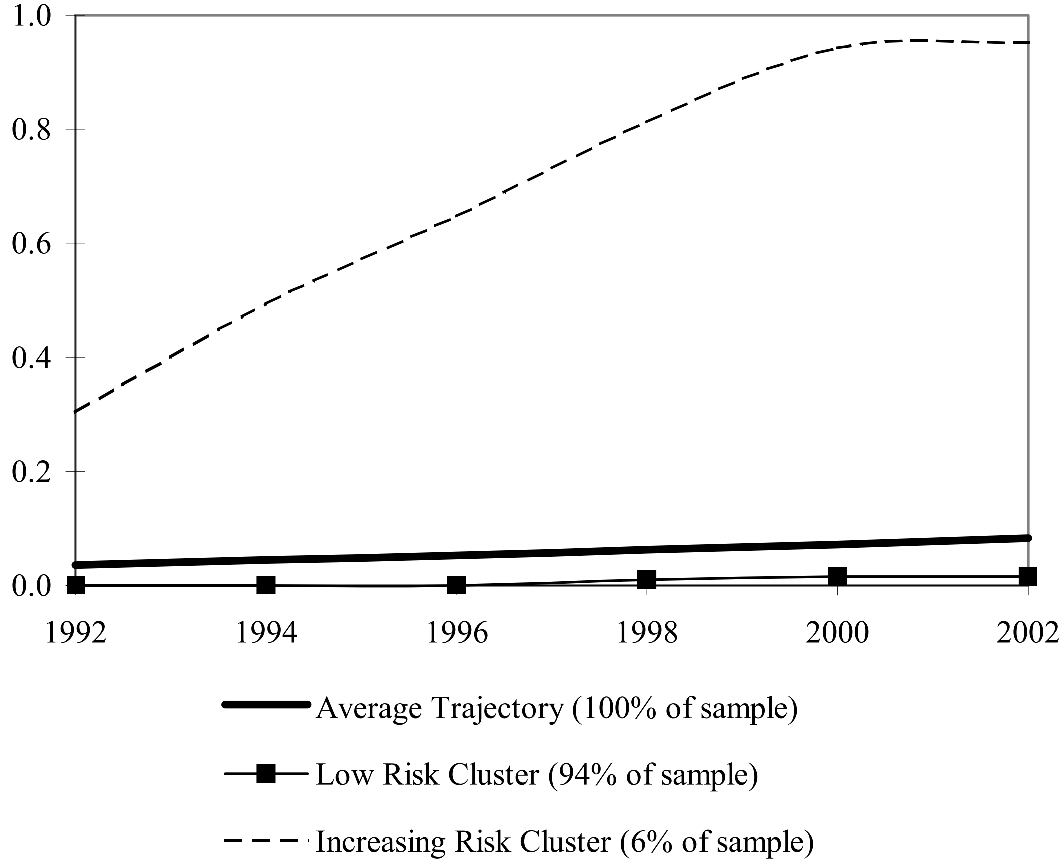

Figure 2: Men's Predicted Probability of Heart Attack by Latent Cluster; Health and Retirement Study 1992-2002 (N=4594)

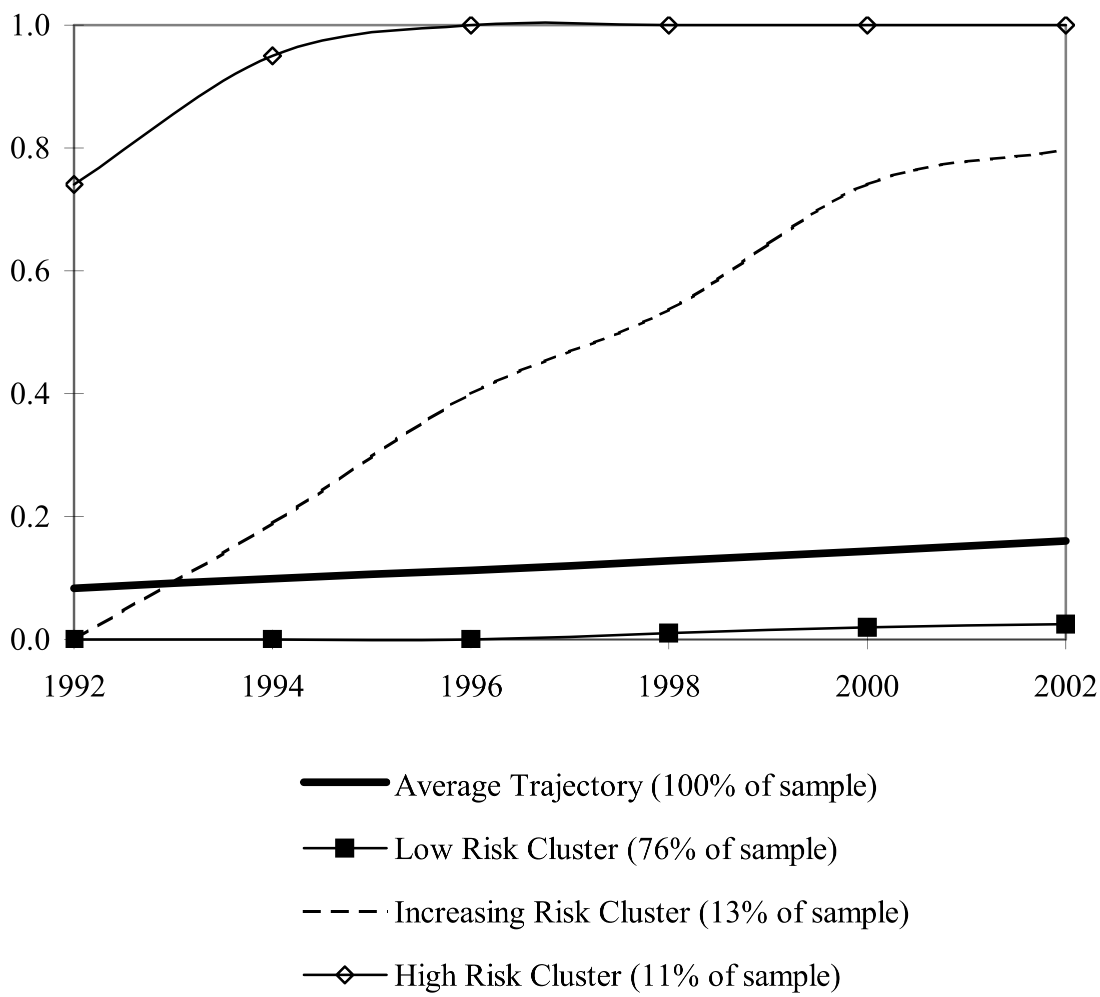

Table 1: Descriptive Statistics by Gender and Childhood Latent Class Cluster: Means and Standard Deviations in Parentheses; Health and Retirement Study 1992-2002

	Men			Women		
	Early Advantaged (N=3057)	Early Dis-advantaged (N=1224)	Fatherless (N=313)	Early Advantaged (N=3416)	Early Dis-advantaged (N=1286)	Fatherless (N=464)
Heart attack by 1992	0.08 (0.27)	0.08 (0.28)	0.09 (0.29)	0.03 (0.16)	0.05* (0.22)	0.06* (0.23)
Heart attack by 2002	0.15 (0.41)	0.18 (0.41)	0.17 (0.40)	0.07 (0.28)	0.12* (0.34)	0.12* (0.35)
Age in 1992	55.82 (3.16)	56.09 (3.19)	55.90 (3.17)	55.80 (3.18)	55.95 (3.13)	56.11 (3.17)
White	0.83 (0.37)	0.78 (0.41)	0.65* (0.48)	0.80 (0.40)	0.75* (0.43)	0.62* (0.49)
No diploma	0.22 (0.42)	0.40* (0.49)	0.35* (0.48)	0.23 (0.42)	0.43* (0.50)	0.42* (0.49)
HS diploma	0.53 (0.50)	0.46* (0.50)	0.49 (0.50)	0.60 (0.49)	0.50* (0.50)	0.50* (0.50)
At least some college	0.25 (0.43)	0.14* (0.34)	0.16* (0.37)	0.17 (0.38)	0.07* (0.26)	0.08* (0.27)
Low income in 1992	0.19 (0.39)	0.26* (0.44)	0.31* (0.46)	0.28 (0.45)	0.40* (0.49)	0.46* (0.50)
Employed in 1992	0.77 (0.42)	0.73 (0.44)	0.67* (0.47)	0.59 (0.49)	0.54* (0.50)	0.55* (0.50)
Hazardous occupation	0.48 (0.50)	0.54* (0.50)	0.54* (0.50)	0.21 (0.41)	0.25* (0.44)	0.26* (0.44)
1992 Service occupation	0.07 (0.26)	0.08 (0.27)	0.09 (0.28)	0.17 (0.37)	0.20 (0.40)	0.23 (0.42)
No health insurance '92	0.20 (0.40)	0.25* (0.43)	0.22 (0.41)	0.21 (0.40)	0.26* (0.44)	0.22 (0.41)

* Means are statistically significant from those of early advantaged cluster at the .01 level of statistical significance.

Table 1, continued: Descriptive Statistics by Gender and Childhood Latent Class Cluster

	Men			Women		
	Early Advantaged (N=3057)	Early Dis-advantaged (N=1224)	Fatherless (N=313)	Early Advantaged (N=3416)	Early Dis-advantaged (N=1286)	Fatherless (N=464)
Dissatisfied friendships	0.02 (0.16)	0.03 (0.16)	0.05 (0.23)	0.03 (0.16)	0.04 (0.20)	0.05 (0.23)
Ever divorced	0.35 (0.48)	0.34 (0.47)	0.39 (0.49)	0.33 (0.47)	0.35 (0.48)	0.46* (0.50)
Dissatisfied marriage '92	0.18 (0.38)	0.16 (0.36)	0.20 (0.40)	0.31 (0.46)	0.34 (0.47)	0.45* (0.50)
Working parent in '92	0.37 (0.48)	0.44 (0.50)	0.37 (0.48)	0.29 (0.45)	0.32 (0.47)	0.34 (0.47)
Ever had cancer	0.14 (0.35)	0.16 (0.37)	0.12 (0.33)	0.16 (0.37)	0.15 (0.35)	0.16 (0.37)
Ever had diabetes	0.20 (0.40)	0.24 (0.43)	0.32* (0.47)	0.19 (0.39)	0.24* (0.43)	0.24* (0.43)
Ever hi blood pressure	0.52 (0.50)	0.58* (0.49)	0.62* (0.49)	0.53 (0.50)	0.60* (0.49)	0.66* (0.48)
Ever smoked	0.73 (0.44)	0.77 (0.42)	0.83* (0.37)	0.54 (0.50)	0.54 (0.50)	0.58 (0.49)
Does not exercise '92	0.44 (0.50)	0.48 (0.50)	0.51* (0.50)	0.52 (0.50)	0.60* (0.49)	0.65* (0.48)
Obese in 1992	0.20 (0.40)	0.23 (0.42)	0.20 (0.40)	0.25 (0.43)	0.27 (0.44)	0.29 (0.45)

* Means are statistically significant from those of early advantaged cluster at the .01 level of statistical significance.

Table 2: Parameter Estimates and Standard Errors from Non-Parametric Hierarchical Model Predicting Women's Heart Attack Risk Trajectories; HRS 1992-2002 (N=5166)

	Low Risk	Increasing Risk	Wald p-value
Predictors of Cluster Membership:			
Intercept	1.13*	-1.13*	--
	(0.05)	(0.05)	
Early disadvantaged	-0.18*	0.18*	--
	(0.03)	(0.03)	
Fatherless	-0.17*	0.17*	--
	(0.05)	(0.05)	
Predictors of Reported Heart Attack:			
Intercept	-8.58*	-2.73*	.001
	(1.45)	(0.92)	
Time	0.47*	0.68*	.04
	(0.03)	(0.10)	
Age	0.06*	0.06*	.95
	(0.02)	(0.02)	
White	0.10	0.05	.54
	(0.07)	(0.06)	
No diploma	0.07	0.25*	.01
	(0.12)	(0.08)	
HS diploma	-0.03	0.19*	.07
	(0.11)	(0.08)	
Low income	0.13*	0.09*	.64
	(0.06)	(0.04)	
Employed	-0.15	-0.12*	.02
	(0.10)	(0.05)	
Hazardous occupation	0.04	0.04	.61
	(0.07)	(0.05)	
Service occupation	0.09	0.19*	.002
	(0.07)	(0.06)	
No health insurance	0.07	0.16*	.002
	(0.09)	(0.05)	

*p<.01

Table 2: Parameter Estimates and Standard Errors in Parentheses Non-Parametric Hierarchical Model Predicting Women's Heart Attack Risk Trajectories; HRS 1992-2002 (N=5166)

	Low Risk	Increasing Risk	Wald p-value
Dissatisfied friendships	0.23*	0.31*	.63
	(0.12)	(0.13)	
Divorced	0.09	0.02	.17
	(0.07)	(0.05)	
Dissatisfied marriage	0.02	0.01	.87
	(0.07)	(0.06)	
Working parent	0.14*	0.20*	.40
	(0.06)	(0.05)	
Cancer	0.19*	0.15*	.63
	(0.07)	(0.07)	
Diabetes	0.30*	0.72*	.001
	(0.07)	(0.28)	
High blood pressure	0.23*	0.05	.05
	(0.08)	(0.05)	
Smoker	0.22*	0.01	.004
	(0.07)	(0.05)	
Does not exercise	0.12*	0.02	.02
	(0.06)	(0.04)	
Obese	0.13*	0.01	.10
	(0.06)	(0.05)	
Log likelihood	-2121.77		
BIC	4628.26		

*p<0.01

Table 3: Parameter Estimates and Standard Errors in Parentheses from Non-Parametric Hierarchical Model Predicting Men's Heart Attack Risk Trajectories; HRS 1992-2002 (N=4594)

	Low Risk	Increasing Risk	High Risk	Wald p-value
Predictors of Cluster Membership:				
Intercept	1.45*	-0.65*	-0.80*	--
	(0.06)	(0.09)	(0.10)	
Early disadvantaged	-0.02	0.05	-0.03	--
	(0.06)	(0.05)	(0.05)	
Fatherless	-0.01	-0.02	0.04	--
	(0.06)	(0.08)	(0.09)	
Predictors of Reported Heart Attack:				
Intercept	-2.75	-6.44	-11.76*	.01
	(1.53)	(7.27)	(2.75)	
Time	0.22*	1.19*	0.53*	.001
	(0.05)	(0.04)	(0.14)	
Age	-0.02	0.05*	0.11*	.01
	(0.03)	(0.02)	(0.04)	
White	0.43*	0.08	0.66*	.001
	(0.13)	(0.07)	(0.17)	
No diploma	0.31	0.19*	0.26	.07
	(0.17)	(0.07)	(0.17)	
HS diploma	0.03	0.23*	0.44	.10
	(0.14)	(0.06)	(0.26)	
Low income	0.16	0.05	0.16	.85
	(0.09)	(0.06)	(0.11)	
Employed	-0.43*	-0.27*	-0.30*	.39
	(0.11)	(0.10)	(0.10)	
Hazardous occupation	0.56*	0.90*	3.34*	.001
	(0.16)	(0.06)	(0.39)	
Service occupation	0.17	0.05	0.35*	.10
	(0.18)	(0.08)	(0.14)	
No health insurance	0.01	0.02	0.41*	.002
	(0.09)	(0.05)	(0.11)	

*p<.01

Table 3: Parameter Estimates and Standard Errors in Parentheses from Non-Parametric Hierarchical Model Predicting Men's Heart Attack Risk Trajectories; HRS 1992-2002 (N=4594)

	Low Risk	Increasing Risk	High Risk	Wald p-value
Dissatisfied friendships	0.29*	0.44	1.17*	.01
	(0.14)	(0.28)	(0.26)	
Divorced	0.12	0.09	0.13	.91
	(0.09)	(0.05)	(0.11)	
Dissatisfied marriage	0.08	0.28*	0.81*	.001
	(0.13)	(0.08)	(0.16)	
Working parent	-1.09*	-4.62*	-1.44*	.001
	(0.15)	(0.72)	(0.30)	
Cancer	0.34	0.01	0.03	.15
	(0.18)	(0.07)	(0.10)	
Diabetes	0.11	0.03	0.34*	.01
	(0.09)	(0.05)	(0.12)	
High blood pressure	0.77*	0.02	0.32*	.001
	(0.17)	(0.05)	(0.12)	
Smoker	0.50*	0.48*	0.12	.001
	(0.12)	(0.07)	(0.42)	
Does not exercise	0.15*	0.01	0.09	.01
	(0.08)	(0.05)	(0.11)	
Obese	0.24*	0.03	0.12	.02
	(0.10)	(0.05)	(0.08)	
Log likelihood	-3077.26			
BIC	6736.33			

*p<0.01