The Effects of Siblings, Parental Socioeconomic Status, Adolescent Aptitude, Educational Attainment, and Wealth on Health in Later Life

Siblings have long been regarded as contributors to the developmental outcomes of children (Downey, 1995; Hertwig, Davis, & Sulloway, 2002; Bjerkedal, Kristensen, Skjeret, & Brevik, 2007). Sibling effects reflect either within or between family processes that promote or constrain inequalities in sibling outcomes. Within-family sibling effects are typically represented by birth order, and the literature indicates that firstborns generally achieve more (Hertwig et al., 2002) and have better health outcomes (Elliott, 1992; Modin, 2002; Angelillo et al., 1999; Barreto & Rodrigues, 1992; Kaplan, Mascie-Taylor, & Boldsen, 1992), than later-born siblings. Between-family sibling effects are typically represented by the number of siblings in a family, or sibship size (Downey, 1995) and the literature indicates that smaller sibships are more likely to produce higher achieving and more intelligent children than larger sibships (Blake, 1989; Falbo & Polit, 1986). Because birth order and sibship size are highly related, many researchers (e.g., Falbo & Polit, 1986; Rodgers, Cleveland, van den Oord, & Rowe, 2000) have argued that both aspects of sibling effects must be considered together in order to understand sibling effects on developmental outcomes. When both birth order and sibship size are considered together, firstborns or those with fewer siblings are generally found to have greater academic achievement (Downey, 1995; Blake, 1989) and better health outcomes (Angelillo et al., 1999; Barreto & Rodrigues, 1992; Li & Taylor, 1993; Lewis & Britton, 1998) than middle or later-borns, especially those with larger numbers of siblings.

While there is an extensive research literature investigating sibling effects on the outcomes of children, there is little research investigating sibling effects on outcomes experienced later in life. Specifically, do these sibling effects on achievement and health during childhood set individuals on life courses that affect their health later, when they are in their 60s? A large and growing body of research (Adler, Boyce, Chesney, Folkman & Syme, 1993; Adler & Snibbe, 2003; Backlund, Sorlie, & Johnson, 1999; Marmot, 2004) has demonstrated that Americans who attain more social status as adults, in terms of greater education and wealth accumulation, live longer and have better health than those who do not. Because firstborns and adults with fewer siblings are more likely to attain more education (Blake, 1989; Falbo & Polit, 1986) and accumulate more wealth as adults (Keister, 2003), it seems likely that they achieve higher status and consequently experience better health outcomes later in life.

There are three broad goals of our research. First, we consider if sibling effects on health in later life can be found and whether these effects are direct or indirect, mediated by the adult's own educational attainment and wealth. Second, we develop and test alternative models that consider characteristics of the adults' family of origin and their personal characteristics as adolescents. In particular, the family characteristic considered here represents the socioeconomic status of the adults' parents. This family characteristic has been found to influence not only the health of children in the family, but also their educational attainment (Bradley & Corwyn, 2002; Brooks-Gunn & Duncan, 1997). The personal characteristic considered here is the graduate's academic aptitude, as measured

in high school. This personal characteristic has also been found to influence not only educational attainment, but also the health of adults (Gottfredson & Dreary, 2004). Third, we created and tested our final model and cross-validate it (c.f., Joreskog & Sorbom, 1993), finally determining if it is equally plausible for men and women.

Mortality Risk and Exceptional Longevity

Recently, two lines of research have emerged that examine sibling effects on mortality risk and exceptional longevity. Taken together, these findings suggest a connection between sibling effects and health in later life, even though their studies focused specifically on mortality risk or exceptional longevity. Clearly, mortality risk, exceptional longevity, and health are inter-related phenomena (MacIntyre, 1986). Poor health is generally thought to increase mortality risk; while, exceptional longevity is caused partly by the avoidance of poor health (Perls et al., 2002).

The first line of research is provided by Modin (2002) who used data from a cohort born between 1915 and 1929 in a Swedish hospital (N = 14,192) to determine whether birth order affected the likelihood of mortality during four life stages: infancy, childhood (age 1-10), adulthood (20-54), and older age (55-80). After adjusting for such factors as mother's marital status, age, and social class, Modin generally found that firstborns had lower mortality risk than later-borns during the first three life stages. However, for the oldest life stage, women demonstrated no birth order effect, while the birth order effect found for men became non-significant after controlling for their own attained social class, education, and income. Modin interpreted these results as indicating the importance of children's birth order in determining mortality risk during all life stages, except the oldest, when sibling effects were mediated by the adult's own attainments.

The second line of research suggesting a connection between sibling effects and health in later life involves investigations of sibling effects among samples of centenarians. Specifically, Gavrilova & Gavrilov (2005) reported a link between exceptional longevity and birth order in a sample of centenarians (N=991) born in the U.S. between 1875 and 1899. They found that first-born daughters were more likely to survive to age 100 than were later-born daughters. However, for men, they found a U-shaped relation between birth order and likelihood of surviving to the age of 100. Among men, firstborns and last-borns were more likely to survive to age 100.

In explaining these sibling effects in exceptional longevity, Gavrilov and Gavrilova (2000) favored biological factors. Specifically, they suggested that the advantage of firstborns might be explained by the relatively younger age of their parents. Since parents are older when they have their later-born children, the quality of their reproductive cells is lower (due to the accumulation of deleterious mutations in the parental germ cells), and this contributes to a biologically-based disadvantage for later-borns that is expressed by a shorter life. On the other hand, Gavrilova and Gavrilov (2005) have explained the advantage of last-born men in terms of the positive association between parity and birth weight (Magnus, Berg, & Bjerkdal, 1985), which may protect them from developing such conditions as heart disease and diabetes later in life (Barker, 1992; 1994).

The research literature regarding sibling effects on mortality risk and extreme longevity also provides contrasting explanations for possible sibling effects on health in later life. Modin (2002) suggested that sibling effects on mortality risk in later life are

expressed indirectly via attained social status, as reflected in the educational attainment and accumulated wealth of the older adult. In contrast, Gavrilov & Gavrilova (2000) argued that sibling effects on exceptional longevity are based partly on causal factors occurring before birth. This suggests that some sibling effects on exceptional longevity are direct, without mediation from the adult's attained social status.

Method

Participants

The longitudinal data used to test our models comes from the Wisconsin Longitudinal Study (WLS: Sewell, Hauser, Springer, & Hauser, 2004), which has been supported since 1991 principally by the National Institute on Aging. Originally, the WLS sample consisted of a random selection of 10, 317 high school graduates from the total pool of 1957 Wisconsin graduates (Hauser, 2005). Born primarily in 1939, these graduates have been surveyed in 1957, 1964, 1975, 1992, and most recently in 2004, when they were re-interviewed first by telephone and then by mail-in questionnaire. About 85% of the surviving graduates participated in the telephone phase of the 2004 survey. In terms of the most recent wave of data collection, the Institutional Review Board at the University of Wisconsin-Madison approved all instruments and operations. Consent was obtained by telephone at the start of the most recent interview and participants were assured of confidentiality. Previous survey data about the graduates have been supplemented by such information as the earnings of their parents from state tax records and the graduates' adolescent ability test scores and rank in their high school class from educational records. Overall, the WLS sample is portrayed as broadly representative of white, non-Hispanic Americans who have completed at least a high school education (Hauser & Roan, 2006).

Although about 13% of the graduates have died since 1957, our analyses indicated that the likelihood of death, as of October 2005, was not significantly related to either sibship size or birth order. Graduates were eliminated from our final sample if they had missing data on any of the variables used in the structural equation modeling analyses reported in this paper. Only graduates who reported growing up with both of their parents (as 90% of the graduates did) were included in the final sample. Ultimately, our final sample consisted 3,968 of the graduates (specifically from WLS version 12.00, released November 1, 2006). Our final sample was randomly divided into the calibration (n=1,984) and validation (n=1,984) sub-samples.

Analysis Plan

We used latent variable structural equation modeling (SEM) to determine the magnitude of sibling effects on health and to consider alternative models of family and personal characteristics that influence health later in life (Byrne, 2001; Bollen, 1989; Keith, 2006). SEM is a method for determining the magnitude of multiple possible causes on multiple outcomes. The linkage of causes and outcomes is defined by models, developed based on theory and previous research, and then tested and evaluated in terms of fitting the data from the calibration sub-sample. When the results of our SEM analyses warranted the addition or deletion of causal pathways, we changed our model and then re-evaluated it using data from the calibration sub-sample. Once we arrived at our final model, we cross-validated it using a multi-group modeling approach and data from the validation sub-sample. Once we cross-validated our model, we checked for structural

invariance across men and women. We input correlations and standard deviations into the structural equation statistical program we used, AMOS, version 7.0 (Analysis of Moment Structures: Arbuckle, 2006).

Variables

The composition of all the latent variables is presented in Table 1, along with the WLS variable name for each indicator. In addition, this table presents alpha coefficients and maximum and minimum scores based on our total sample.

Sibling Effects. The latent variable Sibling Effects was represented by two indicators, one representing the graduate's order of birth and the other, representing the number of siblings within the graduate's family of origin. This information was obtained from the graduate during a telephone interview in 1975. As shown in Table 1, the Coefficient H (Hancock & Mueller, 2001) calculated for the latent variable, Sibling Effects, was above the recommended minimum of .70. Documentation regarding all the indicators used in the analyses reported in this paper can be found at the WLS web site (www.ssc.wisc.edu/wlsresearch/documentation).

Health. The latent variable of Health was represented by four indicators from data collected in 2004. The first two indicators were based on information obtained from the telephone interview. The first indicator measured self-rated health; the graduates were asked to rate their health on a 1-5 scale, with 1 being excellent. This single item is regarded as an excellent indicator of health and is widely used in health assessments (Lorig, Stewart, Ritter, Gonzalez, Laurent, & Lynch, 1996). The second indicator of health was based on our combining the graduates' reports regarding whether a doctor had told them they had high blood pressure, diabetes, cancer (or a malignant tumor), a heart attack (or coronary heart disease, angina, congestive heart failure), arthritis (or rheumatism), high blood sugar, mental illness, or a stroke. For more information regarding the health indicators assessed during the telephone interview, go to Appendix A. The third and fourth health indicators were created by the staff of the WLS based on information obtained from the mail-in questionnaire. For more information about these health indicators, go to Appendix B. Specifically, the third indicator of health was a composite of the graduates' responses to questions regarding 25 physical symptoms. The fourth indicator of health was also a composite of the graduates' responses to questions regarding 14 diagnosed illnesses. We reverse coded the scores of all four indicators so that high scores represented better health. As shown in Table 1, the Coefficient H calculated for the Health latent variable was very close to the recommended minimum of .70.

Educational Attainment and Wealth. The latent variable of Educational Attainment had two indicators, representing the number of years of education the graduate completed, assessed in 1975 and again in 1992. These variables were constructed by the WLS, based on the graduates' responses to telephone interviews. The Wealth latent variable had two indicators. Both were based on data collected via the telephone interview in 2004 and were provided as composite scores by the WLS. One of the indicators of wealth was the total household income, which represented the combined income from all household members. For more information about the components of this indicator, go to Appendix C. The second indicator of wealth was net worth, which represented the graduates' reports of their current assets minus their current debts. For more information about the components of this indicator, go to Appendix D. As shown in

Table 1, the H Coefficients calculated for the Educational Attainment and Wealth latent variables were above the recommended minimum.

Parental Socioeconomic Status and Adolescent Aptitude. Alternative models considered in this study added two latent variables to the simple model of sibling effects. These latent variables were Parental Socioeconomic Status (Parental SES) and Adolescent Aptitude. Parental SES had three indicators, two representing the number of years of education completed by the graduate's mother and father, respectively, and the third representing parental income in 1957. The parent education information had been collected in 1957 from the graduates when they were still in high school, and the parental income variable was based on state tax records. For more information about the indicators of Parental SES, go to Appendix E. Adolescent Aptitude had two indicators, the graduates' IQ scores, which were based on a mapping of raw Henmon-Nelson test scores obtained from high school records. For more information about IQ scores and the graduates' high school grades, which were percentile ranked, go to Appendix F. As shown in Table 1, the H Coefficients for these two latent variables were above the recommended minimum.

Results

Description of Participants

Table 2 shows the background demographic characteristics of the two sub-samples: specifically, their sex, sibling characteristics, and their parents' level of education. Information collected in 2004 from the graduates indicated that 99% of them described their race/origin as White, non-Hispanic. Table 3 presents the correlations of the indicators included in the SEM analyses, separately by the calibration and validation sub-samples. Means and standard deviations for all variables used in the SEM analyses are also shown in Table 3.

Simple Model

Figure 1 presents the results from our testing the simple Sibling Effects and Health model using data from the calibration sub-sample. The fit of the data to the model was adequate: the CFI was .990, the TLI was .985, the RMSEA was .037, and the CMIN/DF was 3.7. None of the Modification Indices (MIs) was above 100. All the standardized regression weights associated with each indicator of the Sibling Effects, Educational Attainment, Wealth, and Health latent variables were statistically significant. As shown in Figure 1, the standardized regression weight associated with the direct path from Sibling Effects to Health was not significant ($p = .45$), while the standardized regression weight associated with the path from Sibling Effects to Educational Attainment was significant ($p < .001$), as were the standardized regression weights associated with the paths from Educational Attainment to Wealth ($p < .001$) and Wealth to Health ($p < .001$).

Because sibling effects have sometimes been found to be non-linear (e.g., Gavrilova & Gavrilov, 2005), we considered the possibility that the latent variable Sibling Effects would be better represented by linear and non-linear indicators of birth order and sibship size. We conducted three additional SEM analyses in which both direct and indirect pathways from Sibling Effects to Health were represented in the model. The first SEM analysis had four indicators of sibling effects: two linear representations of birth order and sibship size, and two non-linear representations of birth order and sibship size. The fit of this first model was much worse than the fit of the simple model portrayed

in Figure 1. The CFI was .797, the TLI was .726, the RMSEA was .180, and the CMIN/DF was 64.9. Given this poor fit, we removed one of the two non-linear indicators and conducted two additional SEM analyses. The goodness of fit measures indicated a better fit. When Sibling Effects was represented by linear versions of birth order and sibship size and a non-linear indicator of birth order, the CFI was .992, the TLI was .988, the RMSEA was .036, and the CMIN/DF was 3.58. When Sibling Effects was represented by linear versions of birth order and sibship size and a non-linear indicator of sibship size, the CFI was .992, the TLI was .989, the RMSEA was .034, and the CMIN/DF was 3.24. None of the MIs produced by these analyses were above 100. All the standardized regression weights produced by these analyses and associated with each indicator of the Sibling Effects, Educational Attainment, Wealth, and Health latent variables were statistically significant.

Overall, these findings indicated that regardless of whether the indicators of Sibling Effects included the two linear and either of the two non-linear indicators, the standardized regression weight associated with the direct pathway from Sibling Effects to Health was not significant, while the standardized regression weights associated with the indirect pathways from Sibling Effects to Health, by way of Educational Attainment and Wealth, were significant. Because adding either of the non-linear indicators of Sibling Effects did not substantially improve the fit of the model to the data, and because adding non-linear indicators did not alter the findings about direct or indirect pathways, we proceeded with solely linear indicators of Sibling Effects in our models.

Overall, these results suggest that there are sibling effects on health in later life and that sibling effects generally influence health indirectly via educational attainment and wealth accumulation.

Alternate Models

A continuing debate within the research literature on sibling effects concerns the extent to which the effects attributed to siblings may be misplaced. Specifically, some researchers have argued that factors such as the socioeconomic status of the individual's family of origin (e.g., Downey & Condron, 2004) or the individual's academic aptitude (Rodgers, 2001) may be causing the variations in the outcomes attributed to sibling effects. For example, Guo and Van Wey (1999) argued that families that produce many children are likely to be socioeconomically disadvantaged, and the reduced outcomes of these children may be due to their socioeconomic disadvantage, rather than their large number of siblings. Because socioeconomic status of origin and adolescent aptitude may influence not only individuals' educational attainment, but also their health in later life (American Psychological Association, 2007; Gottfredson & Deary, 2004), we created another model that controlled for the graduates' socioeconomic status of origin and adolescent aptitude.

This model portrayed Parental SES and Adolescent Aptitude as covariates and retained the paths between Sibling Effects and Educational Attainment, Educational Attainment and Wealth, and Wealth and Health. While all the standardized regression weights associated with each indicator of the Sibling Effects, Educational Attainment, Wealth, Health, Parental SES and Adolescent Aptitude latent variables in this model were statistically significant, the fit of the data from the calibration sub-sample to this model was poor. The CFI was .930, and the TLI was .912, the RMSEA was .068 and the CMIN/DF was 10.15. More troubling, the analyses indicated that two of the MIs were

extremely high, suggesting that the model may be misspecified. Specifically, the results indicated that adding paths between Adolescent Aptitude and Educational Attainment (MI = 358.43) and between Parental SES and Educational Attainment (MI = 221.20) would improve the fit of the data to the model. Therefore, we added these two structural paths to our model and tested its fit using data from the calibration sub-sample.

The results are presented in Figure 2. The fit of the data to this model was better. The CFI was .985, the TLI was .980, the RMSEA was .032, and the CMIN/DF was 3.05. Furthermore, none of the MIs was above 100. All the standardized regression weights associated with each indicator of the Sibling Effects, Educational Attainment, Wealth, Health, Parental SES, and Adolescent Aptitude latent variables were statistically significant. As shown in Figure 2, the standardized regression weights associated with the pathways between Parental SES and Educational Attainment ($p < .001$) and between Adolescent Aptitude and Educational Attainment ($p < .001$) were statistically significant. However, with these new pathways added to the model, the standardized regression weight associated with the pathway between Sibling Effects and Educational Attainment ($p = .739$) was no longer significant.

Overall, these results suggest that graduates who came from families of higher socioeconomic status and who themselves had greater aptitude in high school were more likely to attain more education and accumulate more wealth as adults, leading to better health later in life. Sibling effects were not significant once the structural pathways between parental socioeconomic status and educational attainment and between adolescent aptitude and educational attainment were added to the model. This finding suggests that the effects of parental socioeconomic status and adolescent aptitude on health in later life may be more fundamental than sibling effects.

Final Model and Validation

We could have selected the model in Figure 2 as our final model because it adequately fits the data. However, we decided instead to deepen our knowledge of the effects of Adolescent Aptitude and Educational Attainment on health by adding two direct paths to our structural model.

The first direct path was between Adolescent Aptitude and Health. Gottfredson (2004) has posited that general intelligence is the fundamental cause of the relation between socioeconomic status and health. She argued that because intelligence reflects an individual's fundamental ability to learn, reason, and solve problems, people with greater intelligence acquire more health knowledge and pursue healthier lifestyles and are more likely to avoid disease and injury. Ultimately, she argued that greater intelligence is associated with longer and healthier lives. Her perspective suggests that Adolescent Aptitude has a direct path to Health, perhaps in addition to its indirect path to Health, via Educational Attainment and Wealth.

The second direct path was between Educational Attainment and Health. Mirowsky and Ross (2003) have argued that educational attainment is the fundamental factor underlying the relationship between the socioeconomic status and health. They marshaled evidence indicating that greater education not only promotes employment and wealth accumulation, but also the capacity to control one's own life outcomes. In their view, greater educational attainment can mitigate the effects of low income on health. This perspective suggests that Educational Attainment has a direct path to Health, perhaps in addition to its indirect path to Health, via Wealth.

Thus, we created our final model and tested it with data from the calibration sub-sample. The results are presented in Figure 3. The fit of the data to the final model was good. The CFI was .987, the TLI was .983, the RMSEA was .030, and the CMIN/DF was 2.74. Furthermore, none of the MIs was above 100. The SEM results indicated that the standardized regression weight associated with the direct path between Educational Attainment and Health was significant ($p < .001$). Consistent with the results from the tests of the previous models, the standardized regression weight associated with the path from Educational Attainment to Wealth was also significant ($p < .001$). However, the standardized regression weight associated with the path from Wealth to Health was not significant ($p = .373$). Thus, the SEM results indicate that Educational Attainment has a direct, but not indirect effect on Health.

In contrast, while the standardized regression weight associated with the path from Adolescent Aptitude and Educational Attainment was significant ($p < .001$), as found in the intermediate model, the standardized regression weight associated with the path from Adolescent Aptitude and Health was not ($p = .222$). Thus, the SEM results indicate that Adolescent Aptitude has primarily an indirect, not a direct effect on health in later life.

We proceeded to cross-validate our final model by testing for structural invariance across the two sub-samples. To do this, we followed the approach offered by Byrne (2001) pooling the calibration and validation samples and conducting a baseline analysis in which the final model was fitted to both samples without constraints. Then, we re-ran the analysis, constraining the structural paths to be equal. We conducted a chi-square difference test to determine if the baseline and constrained models were significantly different. They were not ($\Delta\chi^2 = 6.76$, $\Delta df = 7$, $p = .344$). Therefore, we concluded that the structural model is invariant between the calibration and validation sub-samples.

Comparing Men and Women

The research literature regarding sibling effects, mortality risk, and extreme longevity suggested that models of health in later might be different for men and women. In order to determine if our final model was equally plausible for men and women, we tested for structural invariance across data from the men ($N = 1,779$) and women ($N = 2,189$) in our total sample. As we did in the cross-validation analyses described above, we pooled the male and female sub-samples and conducted a baseline analysis in which the final model was fitted to both sub-samples without constraints. Then, we re-ran the analysis, constraining all the structural paths to be equal. We conducted a chi-square difference test to determine if the baseline and constrained models were significantly different. In this comparison, the baseline and constrained models were significantly different ($\Delta\chi^2 = 115.10$, $\Delta df = 7$, $p < .001$). This finding indicates that the final model is not equally plausible for men and women.

In order to determine where the differences might be located, we examined the standardized regression weights generated by each of the six paths in the SEM analyses conducted without constraints. These weights are presented in Table 4 and suggest that the biggest difference between men and women could be found in the path between Adolescent Aptitude and Educational Attainment. The standardized regression weight for men was much higher than that for women. Note also that all but one of the standardized regression weights in Table 4 were statistically significant. That is, for men

the standardized regression weight for the path from Wealth to Health was not significant.

The information in Table 4 suggested that the differences between men and women might be located in two paths. To determine if there was equivalence on the other four pathways in the final model, we re-ran the analysis constraining all but the two pathways to be equal and conducted a chi-square difference test to determine if the initial and constrained models were different. They were not ($\Delta\chi^2 = 7.34$, $\Delta df = 5$, $p = .20$). This finding suggests that the difference between men and women did not reside in the structural paths between Parental SES and Educational Attainment, Educational Attainment and Health, Educational Attainment and Wealth and Adolescent Aptitude and Health.

To test whether the gender difference resided in the structural path from Adolescent Aptitude to Educational Attainment, we re-ran the analysis constraining this path to be equal, while allowing the other paths to be unconstrained. We conducted a chi-square difference test to determine if the initial and the one-path constrained models were different. They were ($\Delta\chi^2 = 90.72$, $\Delta df = 1$, $p < .001$). This supports the idea that one path that differed for men and women was the path from Adolescent Aptitude to Educational Attainment.

We conducted similar analyses to determine if the path from Wealth to Health was different for men and women. Specifically, we re-ran the analysis constraining this path to be equal, while allowing all the other paths to be unconstrained. We then conducted a chi-square difference test to determine if the initial and the one-path constrained models were different. They were ($\Delta\chi^2 = 4.86$, $\Delta df = 1$, $p = .03$). This finding supports the idea that another path that differed for men and women was the path from Wealth to Health.

These results suggest that the final model was not equally plausible for men and women. Specifically, the structural paths between adolescent aptitude and educational attainment and between wealth and health were different for men and women.

Discussion

From the outset, we acknowledge that the generalizability of our findings is limited by the relatively narrow range of sociodemographic characteristics present in our sample. While the original sample was based on a random selection of over 10,000 graduates from a pool of all Wisconsin high school graduates of 1957, it is at best reflective of the experiences of non-Hispanic Whites who have at least a high school education. In addition, we limited our sample to solely those graduates who had lived with both parents in order to avoid the effects of possible confounding factors, such as growing up in a single-parent household. It is possible that our findings might be different if we tested our models on data from a more heterogeneous sample.

Note also that the sample used to test our models had just matured into "later life," arriving at the age of 65 years during the 2004 data collection. Other studies of sibling effects, such as, Modin (2002) and Gavrilova and Gavrilov (2005), have included older adults in their samples. It is possible that tests of models of sibling, parental socioeconomic status, adolescent aptitude, educational attainment, or wealth effects on health in later life might yield different results if the sample included participants who were older. It is generally understood that diseases follow varying developmental trajectories (Chen, Matthews, & Boyce, 2002), and some may not have emerged yet in

this sample. Further, even though the health indicators reflected a broad range of diseases and symptoms, the results of this study are limited by the fact that all our health indicators were based on self-descriptions. It is possible that the tests of our models might have yielded different results if we used as indicators information from health professionals and we focused on specific illnesses, such as diabetes or cardiovascular disease.

With these caveats in mind, the findings from our tests of the simple model suggested that sibling effects on health in later life can be found, but only when the model did not include the factors parental socioeconomic status or adolescent aptitude as contributors. When sibling effects were found, they influenced health indirectly. That is, we found that adults who were born earlier and/or came from smaller sibships were more likely to attain more education, which was related to greater wealth accumulation and, ultimately, better health in later life. However, when our models included the contributions of parental socioeconomic status and adolescent aptitude to educational attainment, our results indicated that the indirect influence of sibling effects diminished to non-significance.

Also, when we tested our simple model, we did not find a significant direct effect of siblings on health in later life, nor did we find that adding non-linear indicators of sibling effects improved the fit of the model or change our findings about direct pathways to health. The rationale for these expectations had been based on biological theories (e.g., Barker, 1992, 1994; Gavrilov & Gavrilova, 2000) of sibling effects on health in later life. It is possible that direct and/or non-linear sibling effects on the development of particular disease clusters might exist, but our analyses with the WLS data did not find them.

Our findings regarding the lack of sibling effects on health in later life are partially consistent with the findings of Modin (2002). That is, Modin reported that sibling effects on mortality risk were found in all age groups studied, except the oldest (55-80 years). For women in this group, no sibling effects were found, but for men, sibling effects became non-significant after controlling for their attained social class, education, and income. In contrast, the centenarian data examined by Gavrilov and Gavrilova (2000) and Gavrilova and Gavrilov (2005) did not include information about the adolescent aptitude, educational attainment, or wealth of the centenarians. Consequently, they were unable to determine if these variables affected the size of sibling effects they found.

The results of the tests of the final model point to the importance of adolescent aptitude and educational attainment for health in later life. Specifically, the results from tests of our final model indicated that educational attainment was directly related to health in later life. The finding of a direct effect is consistent with the Mirowsky and Ross (2003) position that educational attainment is more responsible than wealth accumulation for the link between the social gradient and health. Summarizing the available literature, Mirowsky and Ross (2003) concluded that better education had positive consequences on health throughout life, and these benefits accumulate over the life course, as better-educated individuals more consistently avoid working and residing in undesirable environments and pursue health-promoting lifestyles that include marriage and employment. Furthermore, they argued that higher education leads to benefits that affect the individual's biology, so that over time, the aging process of better-educated persons is slower than that of less educated persons (Ross & Wu, 1996). From this

perspective, research using future waves of the WLS data should observe even greater effects of educational attainment on health as the WLS graduates mature further into their later lives.

Tests of the final model also indicated that educational attainment did not have an indirect effect on health in later life, mediated via wealth accumulation. One of the clear benefits of higher education is employment in more financially rewarding jobs, creating more opportunities to accumulate wealth, making economic hardship less likely (Ross & Mirowsky, 1999). While tests of our final model with data from both male and female graduates indicated that the effect of educational attainment on health was partly mediated by wealth, these results were not the same for male and female graduates. For men, the link from wealth to health was not significant, suggesting that wealth was less important for their health. Future research will investigate further the gender differences in sibling, parental socioeconomic status, adolescent aptitude, educational attainment, and wealth effects on health in later life.

Our results indicated that adolescent aptitude had no significant direct effects on health in later life. This finding is inconsistent with the results from the Scottish Mental Surveys of 1932 and 1947 (Deary, Whiteman, Starr, Whalley, & Fox, 2004), which reported that, after controlling for attained socioeconomic status, the childhood intelligence scores of Scottish citizens were predictive of substantial differences in adult morbidity and mortality (Gottfredson & Deary, 2004).

The results from tests of our final model indicated that adolescent aptitude had an indirect effect on health in later life, mediated by the influence of adolescent aptitude on educational attainment. It is not surprising that adolescent aptitude is strongly predictive of additional educational attainment for high school graduates. Indeed, the point of ability testing and high school ranks is to help high schools and colleges select individual for post-secondary education. Note that our tests of structural invariance between men and women indicated that the influence of aptitude on educational attainment was greater for men than women. This suggests that aptitude may have had more influence on whether men in this sample went on to post- secondary education.

In conclusion, the results of this research suggest that sibling effects on health in later life should be viewed within the context of the complexities of socioeconomic effects on health. Our results indicate that sibling effects on health in later life can be found, but they probably are best understood as reflective of other, more primary factors, such as the parents' socioeconomic situation and the individual's own aptitude.

References

Adler, N.E., Boyce, T., Chesney, M.A., Cohen, S., Folkman, S., & Syme, L. (1993). Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association, 269*, 3140-3145.

Adler, N.E., & Snibbe, A.C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science, 12,* 119-123.

American Psychological Association, Task Force on Socioeconomic Status. (2007). *Report of the APA Task Force on Socioeconomic Status*. Washing, D.C.: American Psychological Association.

Angelillo, I.F., Ricciardi, G., Rossi. P., Pantisano, P. Langiano, E., & Pavia, M. (1999). Mothers and vaccination: Knowledge, attitude, and behavior in Italy. *Bulletin of the World Health Organization, 77,* 224-229.

Arbuckle, J.L. (2006). *Amos 7.0 User's Guide*. Chicago: SPSS. [available online: http://www.amosdevelopment.com/download]

Backlund, E., Sorlie, P.D., & Johnson, N.J. (1999). A comparison of the relationships of education and income with mortality: The National Longitudinal Mortality Study. *Social Science and Medicine, 49,* 1373-1384.

Barker, D.J.P. (1992). *Fetal and infant origins of adult disease*. London: British Medical Journal.

Barker, D.J.P. (1994). *Mothers, babies, and disease in later life*. London: BMJ Publishing Group.

Barreto, T.V., & Rodrigues, L.C. (1992). Factors influencing childhood immunization in an urban area of Brazil. *Journal of Epidemiology and Community Health, 46,* 357-361.

Beier, M.E., & Ackerman, P.L. (2003). Determinants of health knowledge: An investigation of age, gender, abilities, personality, and interests. *Journal of Personality and Social Psychology, 84* (2), 439-448.

Bjerkedal, T., Kristensen, P., Skjeret, G.A., & Brevik. J.I. (2007). Intelligence test scores and birth order among young Norwegian men (conscripts) analyzed within and between families. *Intelligence*. doi:10.1016/j.intell.01.004

Blake, J. (1989). *Family size and achievement*. Berkeley: University of California Press.

Bradley, R.H., & Corwyn, R.F. (2002). Socioeconomic status and child development. *Annual Review of Psychology, 53*, 371-399.

Brooks-Gunn, J., & Duncan, G. (1997). The effects of poverty on children. *Future of Children.* Retrieved from www.futureofchildren.org/information2826/information_show.htm?doc_id=72165

Bollen, K.A. (1989). *Structural equations with latent variables*. New York: John Wiley & Sons.

Byrne, B.M. (2001). *Structural equation modeling with AMOS: Basic concepts, applications, and programming*. Mahwah, NJ: Lawrence Erlbaum Associates.

Deary, I.J., Whiteman, M.C., Starr, J.M., Whalley, L.J., & Fox, H.C. (2004). The impact of childhood intelligence on later life: Following up the Scottish Mental Surveys of 1932 and 1947. *Journal of Personality and Social Psychology, 86,* 130-147.

Dickens, W.T., & Flynn, J.R. (2001). Heritability estimates versus large environmental effects: The IQ paradox resolved. *Psychological Review, 108* (2), 346-369.

Downey, D. B. (1995). When bigger is not better: Family size, parental resources, and children's educational performance. *American Sociological Review, 60*(5), 746-761.

Downey, D.B., & Condron, D.J. (2004). Playing well with others in kindergarten: The benefit of siblings at home. *Journal of Marriage and Family, 66*, 333-350.

Elliott, B.A. (1992). Birth order and health: Major issues. *Social Science and Medicine, 35*(4), 443-452.

Falbo, T., & Polit, D.F. (1986). A quantitative review of the only child literature: Research evidence and theory development. *Psychological Bulletin, 100,* 176-189.

Gavrilova, N.S., & Gavrilov, L.A. (2000). Human longevity and parental age at

conception. In J.M. Robine et al. (Eds.) *Sex and longevity: Sexuality, gender, reproduction, parenthood.* (pp. 7-31). Berlin: Springer-Verlag.
Gavrilova, N.S., & Gavrilov, L.A. (2005). *Living to 100 and beyond: Search for predictors of exceptional human longevity.* Final Report. NORC at the University of Chicago.
Gottfredson, L.S. (2004). Intelligence: Is it the epidemiologists' elusive "fundamental cause" of social class inequalities in health? *Journal of Personality and Social Psychology, 86*(1), 174-199.
Gottfredson, L.S., & Dreary, I.J. (2004). Intelligence predicts health and longevity, but why? *Current Directions in Psychological Science, 13*(1), 1-4.
Guo, G., & Van Wey, L.K. (1999). Sibship size and intellectual development: Is the relationship causal? *American Sociological Review, 64*, 169-187.
Hancock. G.R., & Mueller, R.O. (2001). Rethinking construct reliability within latent variable systems. In R. Cudeck, S. du Toit, & D. Sorbom (Eds.), *Structural Equation Modeling: Present and Future—A Festschrift in honor of Karl Joreskog* (pp. 195-216). Lincolnwood, IL: Scientific Software International, Inc.
Hauser, R.M. (2005). Survey response in the long run: The Wisconsin Longitudinal Study. *Field Methods, 17*(1), 3-29.
Hauser, R.M. & Roan, C.L. (2006). The class of 1957 in their mid-60s: A first look. Retrieved from www.ssc.wisc.edu/wlsresearch/
Hertwig, R., Davis, J.N., & Sulloway, F.J. (2002). Parental investment: How an equity motive can produce inequality. *Psychological Bulletin, 128*(5), 728-745.
Joreskog, K.G., & Sorbom, D. (1993). *LISREL 8: Structural equation modeling with the SIMPLIS command language*. Chicago: Scientific Software.
Kaplan, B.A., Mascie-Taylor, C.G., & Boldsen, J. (1992). Birth order and the health status in a British national sample. *Journal of Biosocial Science*, *24*, 25-33.
Keister, L.A. (2003). Sharing the wealth: The effect of siblings on adults' wealth ownership. *Demography, 40*(3), 521-542.
Keith, T. Z. (2006). *Multiple regression and beyond*. Boston: Allyn & Bacon.
Lewis, S.A., & Britton, J.R. (1998). Measles infection, measles vaccination and the effect of birth order in the aetiology of hay fever. *Clinical and Experimental Allergy, 28,* 1493-1500.
Li, J., & Taylor, B. (1993). Factors affecting uptake of measles, mumps, and rubella immunization. *British Medical Journal, 307*, 168-171.
Lorig, K., Stewart, A., Ritter, P., Gonzalez, V., Laurent, D., & Lynch, J. (1996). *Outcome measures for health education and other health care interventions*. Thousand Oaks CA: Sage Publications.
Macintyre, S. (1986). Health and illness. In R.G. Burgess (Ed.). *Key variables in social investigations*. London: Routledge & Kegan Paul.
Magnus, P., Berg, K., & Bjerkedal, T. (1985). The association of parity and birth weight: Testing the sensitization hypothesis. *Early Human Development, 12,* 49-54.
Marmot, M. (2004). *Status syndrome*. London: Bloomsbury.
Mirowsky, J., & Ross, C.E. (2003). *Education, social status, and health*. New York: Aldine de Gruyter.
Modin, B. (2002). Birth order and mortality: A life-long follow-up of 14, 200 boys and

girls born in early 20th century Sweden. *Social Science & Medicine*, *54*, 1051-1064.

O'Toole, B.J. (1990). Intelligence and behavior and motor vehicle accident mortality. *Accident Analysis and Prevention, 22*, 211-221.

Perls, T.T., Wilmoth, J., Levenson, R., Drinkwater, M., Cohen, M., Bogan, H., Joyce, R., Brewster, S., Kunkel, L., & Puca, A. (2002). Life-long sustained mortality advantage of siblings of centenarians. *Proceedings of the National Academy of Sciences of the United States of America*. *99* (12), 8442-8447.

Rodgers, J.L. (2001). What causes birth order-intelligence patterns? The admixture hypothesis revived. *American Psychologist, 56*(6/7), 505-510.

Rodgers, J.L., Cleveland, H.H., van den Oord, E., Rowe, D.C. (2000). Revolving the debate over birth order, family size, and intelligence. *American Psychologist, 55*(6), 599-612.

Ross, C.E. & Mirowsky, J. (1999). Refining the association between education and health: The effects of quantity, credential, and selectivity. *Demography, 36* (4), 445-460.

Ross, C.E., & Wu, C.L. (1996). Education, age, and the cumulative advantage in health *Journal of Health and Social Behavior, 37*, 104-120.

Sewell, W.H., Hauser, R.M., Springer, K.W., & Hauser, T.S. (2004). As we age: A review of the Wisconsin longitudinal study, 1957-2001. In K. Leicht (Ed.). *Research in Social Stratification and Mobility*, 20, pp. 3-111. Oxford: Elsevier.

Shenkin, S.D., Starr, J.M., Pattie, A., Rush, M.A., Whalley, L.J., & Deary, I.J. (2001). Birth weight and cognitive function at age 11 years: The Scottish Mental Survey 1932. *Archives of Diseases of Childhood, 85*, 189-196.

Taylor, M.D., Hart, C.L., Davey Smith, G., Starr, J.M., Hole, D.J., Whalley, L.J., Wilson, V., & Deary, I.J. (2003). Childhood mental ability and smoking cessation in adulthood. *Journal of Epidemiology and Community Health, 57*, 464-465.

Appendix A

Documentation regarding the health information collected by telephone in 2004 can be found at:
www.ssc.wisc.edu/wlsresearch/documentation/waves/?wave=grad2k&module=ghealth

Appendix B

Documentation regarding the health information collected by mail in 2004 can be found at:
www.ssc.wisc.edu/wlsresearch/documentation/waves/?wave=grad2k&module=gmail_healthb

The indicator of 14 diagnosed illnesses used in this study was a composite of the following illnesses: allergies, asthma, chronic bronchitis/emphysema, chronic sinus problems, circulation problems, fibromyalgia, high cholesterol, irritable bowel syndrome, kidney/bladder problems, multiple sclerosis, osteoporosis, back trouble, ulcers, prostate problems.

The indicator of 25 physical symptoms used in this study was a composite of the following symptoms experienced in the past six months: aching muscles, back pain, bone pains, chest pains, constipation, coughing, diarrhea, painful sex, dizziness, excessive sweating, exhaustion, headaches, lack of energy, neck/should pain, numbness, pain in hands/wrists, pain in ankles/knees, palpitations, ringing in ears, shortness of breath, skin problems, stiff/swollen joints, trouble sleeping, upset stomach, urination problems.

Appendix C

For more information about the total household income indicator, go to:
www.ssc.wisc.edu/wlsresearch/documentation/waves/?wave=grad2k&module=goinc

Appendix D

For more information about the net worth indicator, go to:
www.ssc.wisc.edu/wlsresearch/documentation/waves/?wave=grad2k&module=gassets

Appendix E

For more information about the indicators of parental SES, go to the following location:
www.ssc.wisc.edu/wlsresearch/documentation/waves/?wave=wls5764&module=apar

Appendix F

For more information about the IQ scores, select Appendix G (see memo 121, memo 124, cor652) at the following location:
www.ssc.wisc.edu/wlsresearch/documentation/appendices

Table 1

Latent Variables and their Indices: Range and H Coefficients

Latent Variables (coefficient *H*)	Observed Variables	Range Low	Range High
Sibling Effects			
(*H* = .778)	Sibship Size	0	26
	Birth Order	1	15
Health			
(*H* = .676)	Self-rating (R)	1	5
	Doctor's Report (R)	0	5
	Physical Symptoms (R)	0	25
	Diagnosed Illnesses (R)	0	14
Educational Attainment			
(*H* = .978)	Years of Education, 1975	12	20
	Years of Education, 1992	12	21
Wealth			
(*H* = .750)	Total Household Income, 2004	0	710,000
	Net Worth, 2004	-15,000	12,000,000

Table 1 (continued)

Composition and Coding of Latent and Observed Variables

Latent Variable (*H* coefficient)	Observed Variables	Range Low	Range High
Parental Socioeconomic Status			
(*H* = .691)	Mothers' Years of School	7	18
	Fathers' Years of School	7	18
	Parental Income in 1957	1	998
Adolescent Aptitude			
(*H* = .785)	High School IQ	61	145
	High School Rank	0	99

Note. *N* = 3,968. The latent variable names and their WLS variable names follow. Sibling Effects: birth order (bor), sibship size (sibstt). Health: self-rating (gx201re), doctor's report, which combined responses to eight variables (gx341re, gx342re, gx346re, gx348re, gx351re, gx356re, gx360re, gx361re), physical symptoms (ix082rec), and diagnosed illnesses (ix117rec). All four health indices were reversed (R) coded, so that higher scores represent better health. Educational Attainment: years of education, 1975

(edeqyr), years of education, 1992 (rb003red). Wealth: total household income 2004 (gp260hec), net worth 2004 (gr100rpc). Parental SES: mothers' years of school (edmo57q), fathers' years of school (edfa57q), parental income in 1957 (bmpin1). Adolescent Aptitude: high school IQ (gwiiq_bm), high school rank (hsrankq).

Table 2

Background Demographic Characteristics of Calibration and Validation Sub-samples: Percentages

Latent Variable Indicator	Calibration Sub-sample (n = 1,984)	Validation Sub-sample (n = 1,984)
Sex		
Men	44.7	44.9
Sibling Effects		
Birth Order		
Only & First	38.4	40.6
Middle	39.4	37.4
Last	22.2	22.0
Sibship Size		
0 & 1	26.9	28.4
2	23.3	21.0
3	17.0	17.7
4	11.3	11.6
5 & 6	12.5	11.3
7 – 26	9.1	10.1

Table 2 (continued)

Background Demographic Characteristics of Calibration and Validation Sub-samples: Percentages

Latent Variable Indicator	Calibration Sub-sample (n = 1,984)	Validation Sub-sample (n = 1,984)
Parental Socio-economic Status		
Mothers' Years of School		
7	29.9	29.5
10	18.2	20.0
12	28.4	28.2
13	7.2	6.3
14	5.5	5.2
16 -18	10.7	10.8
Fathers' Years of School		
7	38.3	38.9
10	16.6	17.4
12	19.2	18.3
13	9.9	8.6
14	6.5	6.6
16 – 18	9.7	10.2

Note. Some levels of the indicators of birth order, sibship size, and parents' years of school are grouped together here to simplify presentation.

Table 3

Correlations, Means, and Standard Deviations

Observed Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Means	Standard Deviations
1.Birth Order	□	.63**	-.21**	-.24**	-.15**	-.12**	-.06**	-.16**	-.17**	-.03	-.04	-.06**	-.05*	-.03	.01	2.36	1.78
2.Sibship Size	.63**	□	-.20**	-.22**	-.15**	-.12**	-.10*	-.19**	-.19**	-.06**	-.08**	-.07**	-.02	-.01	.04	3.12	2.51
3.Mothers' Education	-.22**	-.19**	□	.49**	.24**	.22**	.16**	.30**	.30**	.15**	.16**	.11**	.04	.04	-.01	10.77	2.98
4.Fathers' Education	-.22**	-.20**	.49**	□	.33**	.22**	.15**	.35**	.35**	.16**	.15**	.09**	.05*	.02	-.05*	10.43	3.20
5.Parental Income	-.16**	-.15**	.24**	.35**	□	.17**	.07**	.25*	.24**	.15**	.20**	.07**	.03	.05*	-.01	64.51	54.78
6.High School IQ	-.13**	-.14**	.25**	.27**	.16**	□	.58**	.42**	.43**	.24**	.17**	.15**	.08**	.01	.00	103.58	14.39
7.High School Rank	-.09**	-.09**	.16**	.15**	.09**	.59**	□	.37**	.38**	.15**	.10**	.17**	.07**	-.01	.00	56.62	27.73
8.Education, 1975	-.15**	-.17**	.30**	.33**	.24**	.45**	.38**	□	.95**	.31**	.25**	.21**	.09**	.09**	.01	13.69	2.32
9.Education, 1992	-.15**	-.16**	.29**	.34**	.24**	.46**	.38**	.96**	□	.31**	.25**	.21**	.08**	.07**	-.01	13.88	2.39
10.Income, 2004	-.07**	-.09**	.12**	.13**	.16**	.18**	.11**	.32**	.32**	□	.61**	.14**	.01	.05*	-.01	68809.23	83136.24
11.Net Worth, 2004	-.05*	-.06**	.12**	.14**	.15**	.14**	.10**	.26**	.25**	.55**	□	.14**	.05*	.09**	-.00	705253.99	1245802.43
12.Self-Rated Health	-.05*	-.03	.07**	.10**	.06**	.13**	.16**	.18**	.18**	.10**	.12**	□	.38**	.38**	.26**	3.9	.91
13 Doctor Reports	.03	-.00	.03	.00	.04	.02	.09**	.08**	.06**	.01	.02	.38**	□	.30**	.28**	3.81	.95
14.Physical Symptoms	-.03	-.03	.04	.05*	.02	-.00	.04	.09**	.08**	.00	.04	.36**	.33**	□	.42**	16.58	4.84
15.Diagnosed Illnesses	-.01	.02	.01	.04	-.00	.00	.03	.05*	.04	.00	.00	.33**	.32**	.42**	□	12.26	1.64
Means	2.36	3.11	10.74	10.38	69.42	102.91	56.87	13.71	13.92	71350.20	760897.79	3.91	3.83	16.62	12.21		
Standard Deviations	1.77	2.41	2.95	3.21	70.66	14.51	27.68	2.30	2.38	90098.47	414799.59	.92	4.88	1.75	.97		

Note: The matrix for the calibration sub-sample is shown below the diagonal; the validation matrix is shown above the diagonal. **p < .01 * p < .05

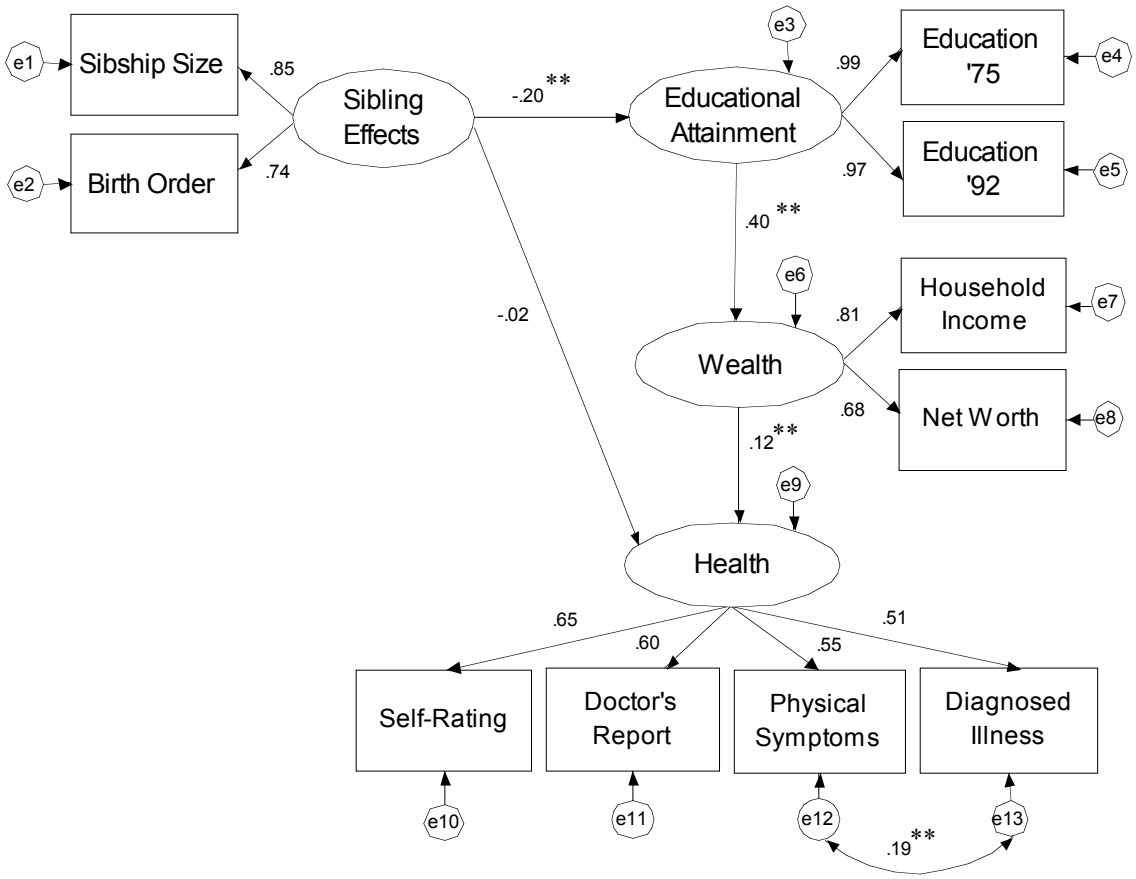
Table 4

Final Model: Standardized Regression Weights for Men and Women

Seven Paths	Men (n = 1,779)	Women (n = 2,189)
Sibling Status -> Educational Attainment	-.02	.01
Parental SES -> Educational Attainment	.26**	.38**
Educational Attainment -> Health	.10*	.12**
Educational Attainment -> Wealth	.36**	.34**
Adolescent Aptitude -> Health	.14*	.08*
Adolescent Aptitude -> Educational Attainment	.57**	.37**
Wealth -> Health	.02	.11*

Note. These standardized regression weights are derived from the fitting of the final model to data from the calibration sub-sample without constraints. *$p < .05$. ** $p < .001$.

Figure 1. Simple model of sibling effects on health in later life. Numbers in the figure are standardized regression weights derived from the SEM analysis of the data from the calibration sub-sample. All the weights associated with indicators of the latent variables are significant. The significance of the weights associated with structural paths is indicated. * $p < .01$. ** $p < .001$.



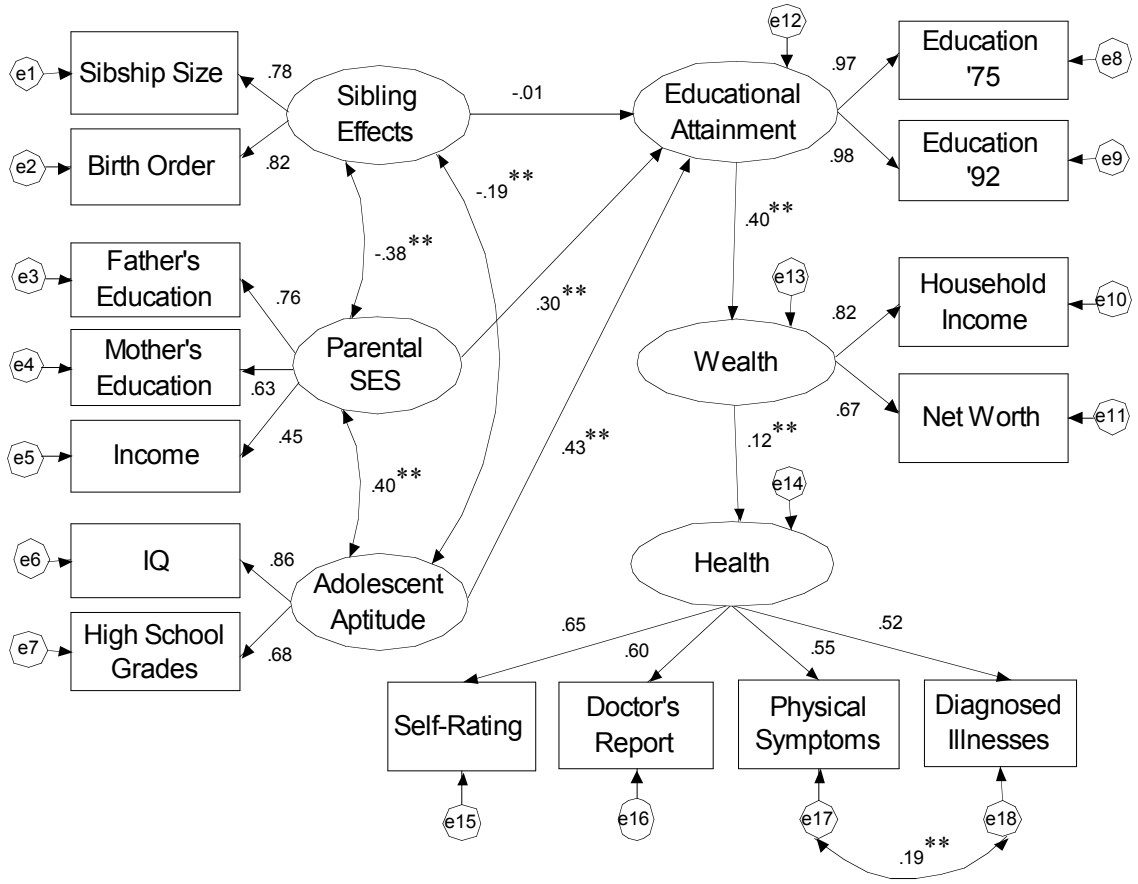


Figure 2. Alternate model of sibling, parental socioeconomic status, adolescent aptitude, educational attainment, and wealth effects on health in later life. Numbers in the figure are standardized regression weights derived from the SEM analyses of the data from the calibration sub-sample. All the weights associated with indicators of the latent variables are significant. The significance of weights associated with the structural paths is indicated. * $p < .01$. ** $p < .001$.

Figure 3

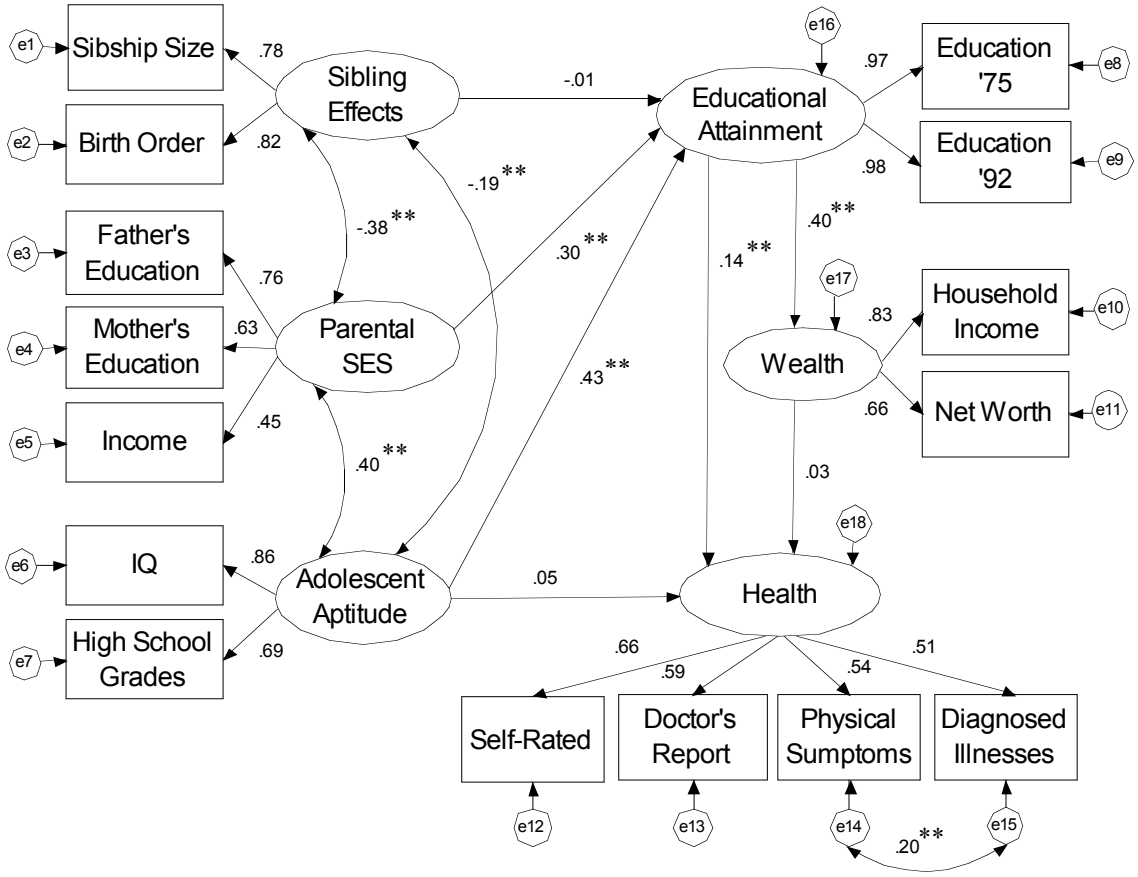


Figure 3. Final model of parental socioeconomic status, adolescent aptitude, educational attainment, and wealth effects on health in later life. Numbers in the figure are standardized regression weights derived from the fitting of the final model to data from the calibration sub-sample. All weights associated with indicators of latent variables are significant. The significance of weights associated with the structural paths is indicated. * $p < .01$. ** $p < .001$.