Early-Life Socioeconomic Status and Mortality in Later Life: An Integration of Four Life-Course Mechanisms

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Objectives. Using data from the Wisconsin Longitudinal Study, we examine (a) how socioeconomic status (SES) at age 18 affects all-cause mortality at ages 54–72, and (b) whether the effect of early-life SES is consistent with the critical period, accumulation of risks, social mobility, and pathway models. We also explore gender differences in the effect of early-life SES and life-course mechanisms.

Method. Participants (N = 6,547) were surveyed in 1957, 1975, and 1993, with vital status established until 2011. We combine discrete-time survival analysis with structural equation modeling. SES and health behaviors are modeled as latent factors.

Results. Early-life SES affects mortality indirectly via status attainment and health behaviors in adulthood and midlife. This finding is contrary to the critical period and consistent with the pathway model. Persistent disadvantage at three life stages is a strong risk factor for mortality, thus, supporting the accumulation of risks. Moreover, the mortality risk of individuals who experienced downward socioeconomic mobility is comparable to their peers with persistent disadvantage.

Discussion. This study highlights the complexity of interrelated life-course processes underlying the effect of early-life SES on mortality in later life.

Key Words: Gender—Health behaviors—Life course—Mortality—Socioeconomic status.

RESEARCH consistently documents striking socioeconomic disparities in mortality that persist regardless of technological and medical advances (Phelan, Link, & Tehranifar, 2010). Biological, social, behavioral, and psychological factors implicated in chronic diseases of aging operate over the life course and across generations (Ben-Shlomo & Kuh, 2002). Therefore, understanding how the processes of social inequality affect mortality in old age requires a life-course perspective, starting with early-life exposures.

Studies exploring early-life origins of mortality disparities revealed the importance of socioeconomic status (SES) of the family of origin (Beebe-Dimmer et al., 2004; Galobardes, Lynch, & Davey Smith, 2004) and documented that all-cause mortality was higher among adults who experienced poorer socioeconomic conditions during childhood (Beebe-Dimmer et al., 2004; Galobardes et al., 2004). Yet, existing research on early-life SES and mortality in later life was characterized by several limitations. In most studies, parents’ SES was assessed with one measure, typically father’s occupation (Beebe-Dimmer et al., 2004; Smith, Hart, Blane, & Hole, 1998). Further, most studies were based on retrospective reports of early-life SES that may be subject to recall bias and lead to the underestimation of the true effect (Galobardes et al., 2004). Moreover, few studies have explicitly examined gender differences in the effect of early-life SES on mortality and in the life-course mechanisms underlying this effect. Finally, researchers overwhelmingly predicted mortality using regression models that cannot explicitly incorporate measurement error, test direct and indirect effects, or examine complex chains of mediators.

We use the 1957–2011 data from 6,547 participants in the Wisconsin Longitudinal Study (WLS) to examine how SES measured at age 18 (in 1957) affects all-cause mortality in later life between 1993 (age 54) and 2011 (age 72). Using SES in adulthood and midlife and health behaviors in midlife, we evaluate life-course mechanisms mediating this effect. We also explore whether and how the focal relationship and life-course mechanisms differ for men and women. A methodological contribution of our study is the implementation of discrete-time survival structural equation models that incorporate measurement error and decompose the association between early-life SES and mortality into direct and indirect effects. Treating SES and healthy lifestyle as latent factors allows for a comprehensive measure that captures real-life multidimensionality of these constructs.

Mechanisms Linking Early-Life SES and Mortality

Our theoretical framework is based on a life-course perspective (Ben-Shlomo & Kuh, 2002). Within the life-course framework, four major conceptual mechanisms...
were proposed to explain the relationship between early-life socioeconomic circumstances and late-life health: the critical period model, the accumulation of risks model, the pathway model, and the social mobility model (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004; Stringhini et al., 2011). Importantly, prior research demonstrates that the distinction between these four models is more conceptual than empirical. These mechanisms are closely interrelated in real life; thus, it is difficult and even unnecessary to separate them statistically (Hallqvist et al., 2004; Rosvall, Chaix, Lynch, Lindström, & Merlo, 2006). Rather, different models should be combined to provide a more complete picture of the effect of early-life SES on mortality (Rosvall et al., 2006). Therefore, our purpose is to explore how each mechanism conveys the effect of early-life SES on later life mortality while also emphasizing the interconnectedness of the four models.

The Critical Period Model

The critical period model reflects a biological imprinting mechanism and suggests that early-life SES has long-lasting and potentially irreversible effects on biological systems (Ben-Shlomo & Kuh, 2002). Consistent with the critical period model, research indicates that SES of the family of origin affects vulnerabilities in childhood that predispose individuals to heart disease in later life (Hamill-Luker & O’Rand, 2007). Moreover, early-life SES can be more influential for cardiovascular risk factors than SES in adulthood (Murray et al., 2011). Thus, we hypothesize that parents’ SES has an enduring impact on later life mortality that is not explained by status attainment and health behaviors in adulthood. An even stronger support for the critical period model will be observed if the effect of early-life SES on mortality risk is greater than the effect of one’s own SES in adulthood.

The Accumulation of Risks Model

The accumulation of risks model suggests that deleterious exposures at different life-course stages inflict a cumulative damage on biological systems and thus have compounding effects on later life mortality (Ben-Shlomo & Kuh, 2002). It is the overall burden of low SES across the life course that contributes to mortality rather than low SES at a particular life-course stage. Persistent disadvantage at multiple stages is particularly deleterious for health (Kahn & Pearlin, 2006).

Statistically, the accumulation of risks mechanism can be modeled as additive effects and interactive effects. In the case of additive effects, low SES in childhood, adulthood, and later life each contributes to increased risk of mortality “independent” of other periods (Galobardes et al., 2004; Wamala, Lynch, & Kaplan, 2011). In the case of interactive effects, SES at multiple life stages affects health synergistically such that the effect of SES at an earlier stage depends on SES at later stages. The adverse effect of earlier disadvantage is magnified with one or more periods of later disadvantage. In the additive model, early-life disadvantage and later life disadvantage are related to health independently, whereas in the interactive model, these effects are conditional on each other and surpass their mere sum.

The Pathway Model

The pathway model suggests that early-life environment is consequential mainly because it shapes life-course trajectories of beneficial or harmful exposures and experiences (Ben-Shlomo & Kuh, 2002). Of particular interest is not the direct effect of early-life SES on mortality, but the pathways connecting family background to health in later life. Because the pathway model emphasizes that the effects of earlier exposures are mediated by later exposures, it is distinct from the accumulation of risks model, which posits the joint and concurrent effects of early-life and later life socioeconomic resources.

An important mediator of the association between parents’ SES and offspring’s adult mortality is SES in adulthood (Galobardes et al., 2004; Lawlor, Sterne, Tynelius, Davey Smith, & Rasmussen, 2006). Parents’ SES is positively associated with individuals’ own SES (Sewell & Hauser, 1975), and socioeconomic conditions in adulthood are strongly and negatively related to mortality (Montez, Hayward, Brown, & Hummer, 2009). Another important pathway linking socioeconomic family background and later life mortality is health behaviors. Higher childhood SES is related to reduced risk of smoking, obesity, higher likelihood of moderate drinking, and regular physical inactivity in adulthood (Blane et al., 1996; Kuh & Cooper, 1992). In turn, low-risk lifestyle (not smoking, regular exercise, healthy diet, and healthy weight) decreases the risk of mortality (Ford, Zhao, Tsai, & Li, 2011). Moreover, because healthy or unhealthy behaviors often co-occur within individuals, it is important to examine synergistic effects of interconnected behaviors aggregating into a lifestyle (Ford et al., 2011). Based on the pathway model, we hypothesize that early-life SES affects mortality indirectly by shaping socioeconomic achievement and health behaviors in adulthood and midlife.

The Social Mobility Model

Contrary to the critical period model, the social mobility model predicts that early-life effects are modified by later circumstances. The burden of early disadvantage can be counterbalanced by improved conditions in adulthood because it is possible to mitigate or even reverse the deleterious impact of early hardships (Ferraro & Shippee, 2009). Upwardly mobile individuals typically have better health outcomes in later life than people who experience socioeconomic disadvantage throughout the life course (Hallqvist et al., 2004; Langenberg, Hardy, Kuh, Brunner, et al., 2011).
In contrast, later disadvantage can outweigh the health benefits of an auspicious start in life because downward mobility is related to increased health risks compared with stable advantage (Hallqvist et al., 2004). Therefore, we hypothesize that upward and downward social mobility modify the effects of initial early-life socioeconomic circumstances. Statistically, the social mobility and the accumulation of risks mechanisms are modeled similarly by estimating interactive effects of SES at different life stages. Yet, whereas the accumulation of risks model evaluates the synergistic effects of persistent disadvantage, the social mobility model emphasizes the transitions from social advantage to disadvantage, and vice versa. The social mobility model is also distinct from the pathway model. The latter analyzes the continuity in SES reflecting the fact that low-SES children are more likely to become low-SES adults than their peers from advantaged background. In contrast, the social mobility model explores life-course discontinuity in SES reflecting improvement or deterioration of SES in adulthood relative to childhood.

Gender Differences
Each of these mechanisms can operate differently for men and women. The distribution of SES, health behaviors, and mortality differs markedly by gender (Montez et al., 2009; Rogers, Everett, Onge, & Krueger, 2010). There is also evidence that early-life socioeconomic disadvantage may be a stronger predictor of chronic disease and mortality in later life among women than men (Claussen, Davey Smith, & Thelle, 2003; Hamil-Luker & O’Rand, 2007). Yet, existing research did not evaluate gender differences in the mechanisms linking parental socioeconomic resources and offspring’s mortality in later life. This study investigates whether and how the effects of early-life SES and hypothesized mechanisms differ by gender.

Design and Methods
The WLS is a long-term cohort study of 10,317 men and women who graduated from Wisconsin high schools in 1957. Every third high school senior was randomly selected, and all of them participated in the baseline study. Participants were interviewed at ages 18 (in 1957), 36 (in 1975), 54 (in 1993), and 65 (in 2004). Deceased participants were matched to the National Death Index to ascertain cause of death and age at death. This study’s analytic sample contains 3,031 men (456 deaths) and 3,516 women (392 deaths) who participated in the 1957, 1975, and 1993 interviews. About 82% of the 1957 sample participated in 1993. A detailed analysis of sample attrition reveals that those who dropped out of the study were more likely to be men, to have lower SES, and to be unmarried. To adjust for differential attrition due to death and nonparticipation, a selection instrument was created using the Heckman selection model and included in all analyses.

Measures

Mortality.—We created a binary indicator of mortality for each year between 1993 and 2011 coded 1 for those who died in this year and coded 0 for those who were alive at the end of this year. These 19 binary variables are used as indicators of the morality factor in all models (Figure 1).

SES in 1957.—Socioeconomic characteristics of the family of origin include “father’s and mother’s education” measured in years, “family income” measured in $100s, “father’s occupation” represented with five categories (unskilled worker, farmer, skilled worker, white-collar worker, and professional/executive), “father’s occupational education” reflecting the percentage of persons in the 1970 Census in a given occupation who completed one or more years of college, and “father’s occupational income” representing the percentage of persons in the 1970 Census in a given occupation who earned at least $10,000.

SES in 1975 and 1993.—Education was assessed as the total completed years of schooling. Occupation of the current job or last job for participants who were not working for pay at the time of the interview is represented with the following categories: professional/managerial, clerical/sales/service, and crafts/operatives/laborers. We include natural logs of household income (1975 and 1993) and household wealth (1993). Occupational education reflects the percentage of persons in the 1970 Census (1975) and in the 1990 Census (1993) in a given occupation who completed 1 year of college or more. Occupational income in 1975 represents the percentage of persons in the 1970 Census, who earned $10,000 in 1969. Occupational income in 1993 represents the percentage of persons in the 1990 Census, who earned $14.30/hr in 1989.

Health behaviors in 1993.—Physical activity in 1993 was assessed with two measures: the frequency of “light exercise” and the frequency of “vigorous exercise” (1 = less than once per month, 2 = one to three times per month, 3 = once or twice per week, 4 = three or more times per week). Body mass index (BMI) is measured as weight in kilograms divided by height in meters squared. Moderate drinking is coded based on the 2010 Federal Dietary Guidelines as 1 drink/day for women and 1–2 drinks/day for men. “Smoking” is coded 1 for persons who never smoked and 0 for current and former smokers.

Control variables in 1993.—We control for marital status (coded 1 for the married and 0 for the unmarried) and the number of children. Although being married and having children is associated with lower mortality (Grundy & Kravdal, 2008; Zhu & Gu, 2010), in the WLS cohort individuals from higher SES background were less likely to be married and had fewer children than their lower SES peers.
Thus, models that do not include these variables can underestimate the protective effect of advantaged family background. Finally, we control for the average age at death of both parents measured 1993 as a proxy for unobserved factors that can jointly affect early-life socioeconomic indicators and later health.

**Statistical Analysis**

First, we obtain summary statistics for all study variables (available as Supplementary Table S1). Then we predict the hazard of mortality between 1993 and 2011 using a discrete-time survival structural equation model shown in Figure 1. The measurement part of this model is presented in Table 1. Factor loadings and fit indices suggest that the indicators measure each factor well.

We apply a multiple-group analysis to examine whether path coefficients differ significantly for men and women. We impose gender invariance constraints in the initial model and then relax an equality constraint for each path at a time and compare improvements in the model fit using Bayesian information criterion and Akaike information criterion. In addition to modeling SES and healthy lifestyle as latent factors, we also estimate discrete-time survival structural equation models with separate indicators of selected SES and health behavior variables. Finally, to explore the social mobility mechanism, we tested all possible two-way interactions and a three-way interaction among SES in 1957, 1975, and 1993. Because all latent variables are standardized with a mean of zero, the main effects in the models with interactions are at the zero values of the other components of the interactive terms. The analysis was conducted in Mplus 6.12. Variables had 2% missing values on average. Missing values were imputed in Mplus using multiple imputation based on Bayesian estimation.

**Results**

Table 2 presents estimates from the best-fitting model with latent factors. The upper panel indicates that only two factors—SES and health behaviors in 1993—have direct effects on the hazard of mortality, albeit with important gender differences. Individuals of higher SES in 1993 had a lower hazard of mortality in the subsequent 18 years than their lower SES peers, and this effect was significantly stronger among women ($\beta = -0.180, p < .001$) than men ($\beta = -0.131, p < .05$). Similarly, a healthier lifestyle was related to reduced mortality risk for all individuals, with the effect of health behaviors being stronger among women than men ($\beta_{women} = -0.204, p < .001; \beta_{men} = -0.128, p < .01$). The direct effect of SES in 1957 on health behaviors in 1993 is significant only among women ($\beta_{women} = 0.048, p < .01$) but not among men ($\beta_{men} = -0.020, p > .05$).

The lower panel indicates that early-life SES has an indirect effect on later life mortality via health behaviors in 1993 among women only ($\beta_{women} = -0.010, p < .01$). In addition, SES in 1957 affects mortality in later life indirectly by shaping men’s and women’s socioeconomic attainment in 1975 and 1993 ($\beta_{women} = -0.050, p < .001; \beta_{men} = -0.036, p < .01$). Socioeconomic advantage of the family of origin reduces the hazard of mortality in later life by increasing the chances of achieving higher SES in adulthood.
This pathway is significantly greater among women because SES in 1993 is related stronger to women’s mortality than men’s mortality. Moreover, among women only, SES in 1957 also affects mortality indirectly via SES in 1975 and health behaviors ($\beta = -0.008$, $p < .001$).

**Figure 2** illustrates predicted probabilities of mortality based on the significant three-way interaction among SES at three life stages: 1957, 1975, and 1993 (shown in Table 2). As indicated in Figure 2, women from higher SES family background, who experienced downward mobility either in young adulthood or midlife, had a significantly greater risk of mortality in later life than women with continuous socioeconomic advantage or upwardly mobile women. Moreover, persistent socioeconomic disadvantage across the life course increased the risk of mortality, yet the effect of downward mobility was just as detrimental as the effect of continuous hardship. In contrast, the two upwardly mobile groups (women who transitioned to higher SES either in young adulthood or in midlife) were similar to women with stably high SES in terms of mortality risk. Among men, the lowest risk of later life mortality was associated with stably high SES or the transition from socioeconomic disadvantage to higher SES between adolescence and young adulthood. Interestingly, the three groups of men were similar in terms of mortality risk: men exposed to persistent disadvantage, men who experienced downward mobility between mid-30s and mid-50s, and men who experienced upward mobility in the same period. The highest risk of morality was observed for downwardly mobile men who were born into socioeconomic advantage but transitioned to lower SES by mid-30s. Thus, men who experienced downward mobility, especially relatively early in life, had even higher mortality than men with persistent socioeconomic disadvantage. Yet, it is difficult to draw a causal inference from this finding. It is possible that downward social mobility affected the mortality risk. But it is also possible that both the downward social mobility and the increased mortality risk were driven by a common cause, such as deteriorating health.

**Supplementary Analysis**

In a supplementary analysis, we modeled SES and health behaviors as separate variables to uncover the role of specific dimensions of SES and lifestyle in reducing or increasing mortality. Results indicate that consistent with the latent factor model, father’s education as a separate indicator of early-life SES does not affect later life mortality directly via SES in 1975 and health behaviors ($\beta = -0.008$, $p < .001$).

**Table 1.** The Measurement Part of the Discrete-Time Survival Model with Latent Variables Estimating the Effects of SES on Mortality: The Wisconsin Longitudinal Study, 1957–2011 ($N = 6,547$)

<table>
<thead>
<tr>
<th>Indicators</th>
<th>Factors</th>
<th>SES 1957</th>
<th>SES 1975</th>
<th>SES 1993</th>
<th>HB 1993</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father’s education</td>
<td></td>
<td>0.643 (0.598)</td>
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<tr>
<td>Mother’s education</td>
<td></td>
<td>0.475 (0.775)</td>
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<tr>
<td>Family income ($100s)</td>
<td></td>
<td>0.399 (0.841)</td>
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<tr>
<td>Father’s occupation</td>
<td></td>
<td>0.725 (0.474)</td>
<td></td>
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<tr>
<td>Father’s occupational education (ln)</td>
<td></td>
<td>0.857 (0.266)</td>
<td></td>
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<tr>
<td>Father’s occupational income (ln)</td>
<td></td>
<td>0.602 (0.638)</td>
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<tr>
<td>Model fit: $\chi^2$ (df) 171 (9), CFI 0.980, RMSEA 0.056</td>
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<tr>
<td>Education</td>
<td></td>
<td>0.644 (.586)</td>
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<tr>
<td>Household income (ln)</td>
<td></td>
<td>0.111 (.988)</td>
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<tr>
<td>Occupation</td>
<td></td>
<td>0.856 (.268)</td>
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<tr>
<td>Occupational education (ln)</td>
<td></td>
<td>0.854 (.271)</td>
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<tr>
<td>Occupational income (ln)</td>
<td></td>
<td>0.587 (.655)</td>
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<tr>
<td>Model fit: $\chi^2$ (df) 94 (5), CFI 0.991, RMSEA 0.052</td>
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<tr>
<td>Education</td>
<td></td>
<td>0.603 (0.636)</td>
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<tr>
<td>Occupation</td>
<td></td>
<td>0.839 (0.294)</td>
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<tr>
<td>Occupational education (ln)</td>
<td></td>
<td>0.832 (0.307)</td>
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<tr>
<td>Occupational income (ln)</td>
<td></td>
<td>0.706 (0.501)</td>
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<tr>
<td>Wealth (ln)</td>
<td></td>
<td>0.479 (0.782)</td>
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<tr>
<td>Model fit: $\chi^2$ (df) 60.634 (5), CFI 0.992, RMSEA 0.044</td>
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<tr>
<td>Healthy weight</td>
<td></td>
<td>0.377 (0.025)</td>
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<tr>
<td>Never smoked</td>
<td></td>
<td>0.155 (0.022)</td>
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<tr>
<td>Frequent light exercise</td>
<td></td>
<td>0.652 (0.034)</td>
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<tr>
<td>Frequent vigorous exercise</td>
<td></td>
<td>0.679 (0.035)</td>
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<tr>
<td>Model fit: $\chi^2$ (df) 23.989 (4), CFI 0.980, RMSEA 0.024</td>
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</tbody>
</table>

**Notes.** Each cell contains standardized factor loadings and residual variances (in parentheses). All factor loadings are significant at the 0.001 level. CFI = the comparative fit index; df = degrees of freedom; HB = health behaviors; RMSEA = the root mean square error of approximation; SES = socioeconomic status.
The indirect effect of father’s education via BMI is significantly greater among women ($\beta_{\text{indirect}}^{\text{BMI}} = -0.010, p < .001$) than men ($\beta_{\text{indirect}}^{\text{BMI}} = -0.003, p < .01$). These gender differences reflect the fact that father’s education has a direct effect on women’s BMI but not on men’s BMI in midlife.

**Discussion**

Using the 1957–2011 data from the WLS, we combine discrete-time survival analysis with structural equation modeling to examine how SES at three life stages (adolescence, adulthood, and midlife) and lifestyle at midlife affect all-cause mortality in later life. We tested four life-course mechanisms linking early-life SES and mortality: the critical period model, the accumulation of risks model, the pathway model, and the social mobility model. We found support for all mechanisms, although to a different extent and in different contexts. These results emphasize the complexity of interrelated processes underlying the enduring effect of early-life SES on mortality over the life course.

**The Critical Period Model**

Whether early-life SES is measured as a multidimensional latent construct or father’s education, it does not affect later life mortality directly. Contrary to the critical period model, the effect of early-life SES on later life mortality in our study is indirect and reflects mechanisms through which family background shapes men’s and women’s SES in 1975 and 1993, as well as health behaviors in 1993. This conclusion is reinforced by findings that individuals who experienced early-life disadvantage fared well in terms of mortality if their circumstances improved in adulthood. Thus, our findings do not support irreversible damage associated with early hardship.
Previous research testing the critical period model with respect to early-life SES and mortality yielded mixed results. Some studies showed that the influence of childhood circumstances remained after adjustment for adult SES (Beebe-Dimmer et al., 2004; Kuh et al., 2002), whereas other studies revealed that the effect of early-life SES on mortality was mostly explained by socioeconomic achievement and health behaviors in adulthood (Hayward & Gorman, 2004; Stringhini et al., 2011). This inconsistency is not surprising given the vast differences across studies in countries of origin, birth cohorts, measures of early-life SES, years of follow-up, and ages at death. It is also important that most studies considered whether the effect of early-life SES persists net of adult SES but not healthy lifestyle, which is a critically important mechanism. Consistent with our findings, studies that included adult SES combined with health behaviors largely explained the effect of early-life SES (Hayward & Gorman, 2004; Warner & Hayward, 2006). The lack of direct effects of parents’ SES documented in our study does not negate the importance of family background as a root of later life mortality. We emphasize that family of origin is a powerful force propelling unequal life-course trajectories that ultimately affect health outcomes.

The Accumulation of Risks Model

The latent factor model does not support the accumulation of risks mechanism because there is no evidence of additive effects of SES at the three life stages. Only SES in 1993 is directly related to post-1993 mortality, whereas the effect of earlier stages is fully conveyed by socioeconomic resources at subsequent stages. Yet, the accumulation of risks mechanism is supported to some extent by the separate indicators model. Education in 1993 is a significant mediator of the effect of father’s education among men and women even net of education in 1975. This pattern reveals additive effects of socioeconomic disadvantage in young adulthood and midlife. Alternatively, this pattern may reflect intergenerational and intrapersonal social mobility—a change from origin to destination.

Further, support for the accumulations of risks model is provided by significant interactive effects indicating that persistent disadvantage (low SES in adolescence, adulthood, and midlife) is a strong risk factor for mortality.
especially relative to continuous advantage. These findings are consistent with research emphasizing the importance of joint effects of socioeconomic resources at different periods of the life course (Smith et al., 1998; Kahn & Pearlin, 2006). Thus, health implications of early-life SES are not fully absorbed by SES at subsequent stages; rather, the lingering effects of earlier socioeconomic disadvantage operate in concert with current low SES.

Pathway Model
Consistent with the pathway model, the effect of early-life SES on later life mortality is sequentially transmitted through SES at each subsequent life stage. Higher parents’ SES leads to higher offspring’s SES in young adulthood that is further related to higher SES at midlife. In turn, socioeconomic advantage in middle age is associated with reduced mortality in the next 18 years. Further, our findings show that parents’ SES is directly and positively related to women’s but not men’s health behaviors at midlife. Therefore, the indirect effect of early-life SES on mortality via health behaviors in 1993 is significant among women only. This finding also holds when obesity is included as a separate variable. The enduring effect of early-life SES on women’s body weight is consistent with the critical period model. Early-life social environment can launch changes in physiology that have long-term effects of on later life body weight. Stress in childhood resulting from low SES can lead to a chronic elevation of cortisol levels, which in turn is associated with metabolic irregularities promoting excess weight over the life course (Björntorp and Rosmond, 2000). Further, a heightened risk of obesity can be programmed during inadequate prenatal development or via postnatal biochemical disruptions (James, Fowler-Brown, Raghunathan, & Van Hoewyk, 2006). Girls may be more sensitive than boys to obesogenic environment because of biological sex differences in fat storage and metabolism (Power & Schulkin, 2008).

Upward and Downward Social Mobility
Our findings reveal that both women and men who started with low SES but then experienced upward mobility, especially relatively early in life, are similar to their peers with stably high life-course SES. This pattern of no irreversible damage bolsters our lack of support for the critical period model. Further, individuals who experienced downward mobility either in young adulthood or in midlife had a significantly greater risk of death after 54 years than the continuously advantaged or upwardly mobile groups. The highest mortality risk was observed among downwardly mobile men who were born into socioeconomic advantage but then transitioned to low SES by their mid-30s. Our findings indicate that the adverse impact of downward mobility is comparable with or even worse than the impact of persistent disadvantage. This pattern suggests that socioeconomic advantage in early-life may not protect health against subsequent low SES. Facing hardship after prosperity can be a particularly pernicious stressor.

Finally, we find that among men being socioeconomically advantaged for most of the life course and then experiencing downward mobility in midlife is similar in its effect on mortality to spending most of the life course in low SES and then transitioning to higher SES in midlife. Thus, disadvantage accumulated over a considerable life-course segment undermines the positive effect of upward mobility, whereas accumulated advantage mitigates the unfavorable effect of later life downward mobility.

Limitations and Future Research
An important limitation of this study is that all participants graduated from high school and thus obtained higher levels of education than the average levels for this birth cohort. To speculate how this left selection of our sample with respect to own education might have affected our findings, we conducted a simulation (available upon request) adding the information on persons with less than high school education in the Health and Retirement Study to our original WLS sample. The results of this simulation suggest that if the WLS sample contained more participants with lower levels of education, the direction of the statistically significant effects would likely be the same, but the magnitude of the effects would increase slightly. In other words, the WLS provides conservative estimates of the effects of early-life socioeconomic characteristics on mortality. Our findings are further reinforced by considerable variability in our focal variables reflecting parents’ SES (Supplementary Table S1).

Because our sample comprises only White participants, we could not explore race and ethnic differences in the effect of early-life SES on mortality. This direction is important for future research because minority groups, especially African Americans, derive fewer health benefits from higher SES compared with White adults (Cummings & Jackson, 2008). Further, our study is based on one cohort born in 1939. An interesting avenue for future studies is to compare the life-course effects of SES on mortality in the WLS cohort to older and younger generations. Moreover, whereas all-cause mortality is a comprehensive and potent indicator of health disparities, attention to specific causes of death in future studies may help further elucidate pathways through which SES affects health. The WLS did not assess health behaviors in childhood, adolescence, and young adulthood, which may be important influences on mortality in addition to health behaviors at midlife included in our analysis. Future studies should include information about health behaviors assessed prospectively over the life course. In addition, the WLS does not contain information about intergenerational transmission of health behaviors, which may be an important mechanism through which parental SES affects later life mortality.
CONCLUSION

Despite these limitations, this study uncovers the mechanisms through which early-life SES operates across the life course to affect later life mortality. Because of enduring health implications of early-life disadvantage, policies addressing socioeconomic inequality among children and adolescents may be a critical route to alleviating socioeconomic health disparities in later life. Moreover, parents’ SES also shapes health behaviors that ultimately affect mortality. Thus, health behaviors should be viewed as a product of structural inequality. It is not sufficient to educate children and adults about optimal health behaviors if there are structural constraints that prevent low-SES individuals from adhering to a healthy lifestyle. Therefore, it is important to implement policies that are relatively inexpensive and easy to disseminate and thus allow to circumvent limited resources of low SES (Phelan et al., 2010). Yet these policies should also be designed from a life-course perspective because early life is a particularly malleable and fruitful period to prevent life-course processes of accumulation of socioeconomic and health disadvantages, which are already in full swing in adulthood and midlife.

SUPPLEMENTARY MATERIAL

Supplementary material can be found at: http://psychsocgerontology.oxfordjournals.org/

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