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Cumulative Childhood Adversity, Educational Attainment, and Active Life Expectancy Among U.S. Adults

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Abstract

Studies of the early-life origins of adult physical functioning and mortality have found that childhood health and socioeconomic context are important predictors, often irrespective of adult experiences. However, these studies have generally assessed functioning and mortality as distinct processes and used cross-sectional prevalence estimates that neglect the interplay of disability incidence, recovery, and mortality. Here, we examine whether early-life disadvantages both shorten lives *and* increase the number and fraction of years lived with functional impairment. We also examine the degree to which educational attainment mediates and moderates the health consequences of early-life disadvantages. Using the 1998–2008 Health and Retirement Study, we examine these questions for non-Hispanic whites and blacks aged 50–100 years using multistate life tables. Within levels of educational attainment, adults from disadvantaged childhoods lived fewer total and active years, and spent a greater portion of life impaired compared with adults from advantaged childhoods. Higher levels of education did not ameliorate the health consequences of disadvantaged childhoods. However, because education had a larger impact on health than did childhood socioeconomic context, adults from disadvantaged childhoods who achieved high education levels often had total and active life expectancies that were similar to or better than those of adults from advantaged childhoods who achieved low education levels.

Keywords

Active life expectancy; Mortality; Childhood conditions; Education; Life tables

Introduction

Mounting evidence documents that adult physical functioning and mortality risk, like many health conditions, reflect the accumulation of experiences throughout the life span (see Kuh

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and Ben-Shlomo 2004). Studies focusing on the early-life origins of functioning and mortality have found that childhood health and socioeconomic context (SES) have an especially "long reach" in their health effects, often irrespective of adult experiences. Here, we investigate the extent to which these early-life experiences reverberate across the life course to shape the *intersection* of functional ability and mortality risk—that is, the years of life lived with and without functional impairment, or active life expectancy.

This study is motivated by two main concerns. The first concern is whether early-life experiences similarly impact active and total life expectancies. Adverse early-life experiences may slightly shorten lives but substantially increase the number and fraction of those years lived with functional impairment. In contrast, adverse early-life experiences may substantially shorten lives but slightly increase the number of those years impaired. In other words, we are interested in how childhood experiences impact both the quality and length of life, which resonates with the debate about whether recent gains in life expectancy reflect more years lived in better health (Fries 1980) or in poor health (Gruenberg 1977).

The second concern is the extent to which educational attainment mediates and moderates the link between early-life experiences and active life expectancy. Addressing this concern helps illuminate the life course origins of active life expectancy (e.g., whether it mainly reflects childhood or adulthood or both) and redirect policy efforts toward the portion(s) of the life course at which intervention may be the most effective. We focus on education as the key marker of adult context for several reasons. Education is highly correlated with numerous dimensions of adult life—such as health-related behaviors, social ties, and psychosocial well-being—and it is causally prior to other dimensions of SES (Mirowsky and Ross 2003). Education is also a potentially powerful policy lever. For instance, public expenditures on education in the United States have the most substantial impact on state-level mortality rates compared with other public services, such as environment and housing, welfare, and health and hospitals (Dunn et al. 2005).

Our questions are straightforward. How many years of total life and active life do individuals who experienced adverse early-life contexts live compared with their counterparts who experienced salubrious early-life contexts? Do adverse early-life contexts shorten active life more than they do total life and thus expand the fraction of life lived impaired, or do they shorten total life more than they do active life? How do early-life experiences combine with educational attainment to predict total and active life? We address these questions for non-Hispanic white and black men and women 50 to 100 years of age in the 1998–2008 Health and Retirement Study using a multivariate, multistate life table approach.

Our study reveals that compared with adults from advantaged childhoods, adults from disadvantaged childhoods lived fewer total years of life, lived fewer years of active life, and spent a greater portion of life functionally impaired, even when we adjusted for educational attainment. Higher levels of educational attainment did not ameliorate the health consequences of disadvantaged childhoods. However, because education had a larger impact on health than did childhood SES, adults from disadvantaged childhoods who achieved high levels of education generally had total and active life expectancies that were similar to or

better than those of adults from advantaged childhoods who achieved low levels of education, although this was more apparent for men than for women.

Active Life Expectancy

Active life expectancy is a powerful means to gauge inequalities in health because it directly reflects the combined implications of age-specific functioning and mortality risks on the health experiences of individuals over their projected life cycle. In comparison, cross-sectional estimates of population health reflected in prevalence rates (e.g., the prevalence of functional impairment) inherently mask the underlying dynamics of disability incidence, recovery, and mortality selection. Although recent efforts to model health trajectories have yielded valuable information on gross types of trajectories (e.g., Nusselder et al. 2006; Warner and Brown 2011), the timing and nature of the underlying transitions in functioning —as well as mortality selection and the number of years lived with health conditions—are often obscured. Active life expectancy, in contrast, takes into account the timing and nature of disability changes, as well as mortality that is hinged to disability, to estimate the years of life expected to be disabled—and not disabled—for persons of a given age. For example, for individuals aged 65, it estimates how many additional years they can expect to live and how many of those years will be lived with and without functional impairment.

In this study, active life expectancy is defined as the total number of years that individuals can expect to live without functional limitations (e.g., difficulty climbing stairs), difficulty with instrumental activities of daily living (e.g., shopping), or difficulty with basic activities of daily living (e.g., bathing). Substantial disparities in active life expectancy exist between demographic subgroups. Gaps between education subgroups are particularly striking, supporting the theory that education is a powerful resource through which adults garner the material, cognitive, social, and psychological means for creating healthy lives (Mirowsky and Ross 2003). To illustrate, Crimmins and Saito (2001) reported that in the United States in 1990, the gap in total life expectancy between white men aged 30 with 0–8 years of education and their peers with 13 or more years of education was 6.7 years, compared with a 10.8-year gap in healthy life expectancy. For black men, the education gap in total life expectancy was 11.8 years compared with a 16.2-year gap in healthy life expectancy. The patterns were even more accentuated among women.

Although the association between active life expectancy and educational attainment is well established, a life course epidemiological perspective impels us to consider the possibility that active life expectancy reflects experiences throughout the life course. Indeed, many childhood and adult experiences independently predict the prevalence of functional limitations (Alvarado et al. 2007; Guralnik et al. 2006; Haas 2008; Haas and Rohlfsen 2010; Luo and Waite 2005; Turrell et al. 2007) and disability (Bowen and Gonzalez 2010; Freedman et al. 2008; Haas 2008) as well as mortality risk (Barker 1997; Davey Smith et al. 1998; Finch and Crimmins 2004; Hayward and Gorman 2004; Kuh et al. 2002; Montez and Hayward 2011; Turrell et al. 2007; Warner and Hayward 2006). The next step, which we address here, is to assess how early-life experiences shape transitions in functional ability across the disablement process (Verbrugge and Jette 1994) and thereby shape disparities in active life expectancy.

Early-Life Socioeconomic Context and Health

This study focuses on two early-life experiences—childhood SES and health—that have a particularly long reach in shaping adult health (Blackwell et al. 2001; Hayward and Gorman 2004). Childhood SES is multifaceted and multidimensional, reflecting the education, economic well-being, psychosocial resources, and sociocultural capital of key caregivers (e.g., parents) and the community (e.g., schools, neighborhoods), and thus shapes the "social ecology" (Bronfenbrenner 1977) or "environtype" (Sameroff 2000) of early life. It tends to materialize into clusters of salubrious or deleterious exposures. For instance, compared with children raised in advantaged socioeconomic contexts, children raised in adverse contexts consume less-nutritious diets; engage in less physical activity; experience more family conflict and disruption; have parents who are less responsive and more authoritarian; attend lower-quality schools; live in housing structures that are crowded and noisy; and are more likely to be exposed to secondhand smoke and toxins, such as lead-based paint, within their homes (Cohen et al. 2010; Evans 2004). It may be the accumulation of these experiences—rather than any single experience—that makes childhood SES a powerful predictor of adult health outcomes (Bronfenbrenner 1977; Evans 2004; Sameroff 2000).

Childhood health also reflects a wide range of experiences—for example, disadvantaged SES (Luo and Waite 2005), low birth weight (Haas 2007), infectious disease exposure (Haas 2008), abuse and neglect (Green et al. 2010), and exposure to secondhand smoke. Although childhood health partly reflects childhood SES, the two factors may independently influence adult health (Blackwell et al. 2001; Haas 2007; Luo and Waite 2005). This pattern implies that once childhood health is compromised, it can "take on a life of its own" in shaping adult health. Another reason we focus on childhood health is that it may capture important early-life exposures (e.g., accidental poisoning) that were not assessed by the survey and may not strongly correlate with the assessed exposures.

Conceptually, early-life health and socioeconomic context may shape adult functioning, mortality risk, and active life expectancy through "direct" and "indirect" processes. They may directly influence these outcomes through biological imprint processes. For instance, the link between birth weight and metabolic and cardiovascular diseases (Barker 1997) and lean muscle and bones mass (Gale et al. 2001) suggests that prenatal nutrition may impart an enduring imprint on organs, tissues, and bone. Other correlates of childhood SES, such as chronic exposure to pathogens (Finch and Crimmins 2004) and psychosocial stressors (McEwen 1998), may permanently alter regulatory set points that can damage health.

Early-life SES and health may also *indirectly* shape functioning and mortality by setting in motion adult circumstances that, in turn, affect these outcomes. One well-established and important indirect mechanism is educational attainment. Children who experienced poor health and adverse SES in childhood tend to achieve lower levels of education, which can lead to poor adult health. Other indirect mechanisms such as health behaviors exist, although we do not examine them in this study. For instance, in a study of U.S. adults, their parents' education was related to the adults' physical functioning partly through educational attainment and health behaviors (Mirowsky and Ross 1998).

Educational Attainment

Because early-life conditions set in motion adult achievement processes and exposures largely through the critical life experience of education, it is important to understand how early-life conditions and education combine to influence active life expectancy. We are interested in two issues. First, when education is statistically controlled, what are the net consequences of early-life conditions? Second, to the extent that both early-life conditions and education influence later life functioning and mortality, how do these factors combine to influence the range of potential health outcomes? For example, in life table terms, how many years of life and active life are gained by attaining a high school credential among individuals from disadvantaged childhoods?

Our focus on educational attainment reflects several considerations. Education is a key mechanism through which social class is reproduced across generations, providing economic, social, and cultural capital, and indelibly influencing dispositions and behaviors (Bourdieu and Wacquant 1992). The inverse association between education and health has been documented for more than 50 years within the United States (Hummer and Lariscy 2010; Kitagawa and Hauser 1973), and its persistence across time and place suggests that education is a flexible and powerful resource that allows individuals to avoid health risks and adopt healthy lifestyles regardless of time or place, such that the association persists even if the mechanisms change (Link 2008). For example, education provides favorable economic and material conditions, greater psychosocial resources such as salubrious social ties and a sense of control, and healthier lifestyles (Ross and Wu 1995)—factors that in turn promote health. Indeed, several recent studies suggest a causal role of education on health (Baker et al. 2011; Glymour et al. 2008; Lleras-Muney 2005), although health may also influence educational attainment to some degree (Haas et al. 2011). Although other socioeconomic resources (such as income and occupation) also shape health, education is causally prior (Mirowsky and Ross 2003), and its association with health often remains after adjusting for these resources (Baker et al. 2011). Education data are also available for all adults regardless of employment or age, and education is relatively stable across the adult life course.

Our focus on educational attainment also reflects policy considerations. Some scholars have advocated a focus on education policy as a solution to the growing health divide within the United States (House et al. 2008; Low et al. 2005). The logic is as follows. The inequitable distribution of socioeconomic resources within a population is a “fundamental cause” of health disparities (Link and Phelan 1995), and these disparities exist across the range of socioeconomic statuses (i.e., not just between the poor and nonpoor). However, determining which socioeconomic resource(s) to target for social policy is crucial. Some solutions, such as universal health care, are unlikely to be wholly affective; others, such as income redistribution, are politically contentious (House et al. 2008; Low et al. 2005). Furthermore, solutions like the previous two target only a segment of the population. In contrast, ensuring access to high-quality education for the entire population could “lift all boats” and reduce inequities in downstream socioeconomic resources (e.g., income). Indeed, public expenditures on education in the United States have the most substantial impact on state-level mortality rates compared with other public services (Dunn et al. 2005). We contribute

to this debate by assessing the extent to which education mediates and moderates the health consequences of early-life adversities in our study.

Data and Methods

The data come from the Health and Retirement Study (HRS), which is a household panel survey designed to study retirement processes, economic well-being, and health among U.S. adults 50 years of age and older (HRS 2008). The present study uses the RAND HRS Version L Data File, which is a cleaned and consolidated file of all 1992– 2010 survey waves developed by the RAND Center for the Study of Aging and supported by the National Institute on Aging and the Social Security Administration (RAND 2011). The file contains 30,671 adults who are representative of cohorts born between 1890 and 1953 and their spouses. The present study begins with the 1998 wave because it was the first to collect detailed data on early-life contexts, and it ends with the 2008 wave because it was the last year for which deaths were linked to the National Death Index (described later in the article). The analytic sample for the present study includes U.S.-born, non-Hispanic whites and non-Hispanic blacks aged 50–100.

Mortality and Functioning Status

The multistate life table approach in modeling active life expectancy requires two types of information: mortality incidence and the incidence of functioning changes. The HRS provides vital status information to determine mortality incidence from two sources. One source is the HRS tracker file, which reports vital status at each interview wave based on information gathered during the interview process (e.g., a spouse may report that a study member died since the last interview). Month and year of death are then ascertained through an exit interview with the spouse or other knowledgeable individual. The second source is the National Death Index (NDI), which is a computerized database of all certified deaths in the United States since 1979. The HRS provides information to the NDI on adults whose vital status was unconfirmed or presumed dead, and then vital status and date of death are ascertained by the NDI through a probabilistic matching algorithm (Lochner et al. 2008; National Center for Health Statistics NCHD 2009). For the present study, an adult is considered deceased if either source classified the adult as deceased.[1] Note that deaths are recorded for all HRS respondents who died, even those who were institutionalized or had left the sample prior to death.

Adults who were alive at an interview wave are classified here into one of four mutually exclusive and exhaustive functioning states (Crimmins et al. 1994, 1996) based on a series of questions about physical functioning. Distinguishing four states provides a fairly fine-grained assessment of the stages within the disablement process where early-life experiences shape functional ability and thus a potentially better understanding of the etiology of impairment.

[1]Comparisons of the deaths uncovered via the HRS tracker files and deaths uncovered via the NDI matching algorithm in the HRS found that the algorithm fails to capture some deaths and depresses mortality rates, especially among minority status women at older ages.

Adults were asked whether they had difficulty with certain activities because of a health or memory problem, excluding difficulties that they expected to last less than three months. Adults who reported difficulty with (including inability to do) at least one of six activities of daily living (ADL)—walking across a room, dressing, bathing, eating, getting in and out of bed, and toileting—are classified as having an ADL at that wave. Adults who did not experience any difficulties with ADL but did report some difficulty with (or inability to do) at least one of five *instrumental* activities of daily living (IADL)—using a telephone, managing money, taking medications, shopping for groceries, and preparing meals—are classified as having an IADL at that wave. Adults who did not experience difficulty with any of the 11 ADL/IADL activities but reported some difficulty with at least 1 of 11 functions—walking one block, walking several blocks, sitting for two hours, getting up from a chair after sitting for long periods, climbing several flights of stairs without resting, climbing one flight of stairs without resting, stooping/kneeling/crouching, lifting or carrying weights more than 10 pounds, picking up a dime from a table, reaching arms above shoulder level, and pushing or pulling large objects—are classified as having a functional limitation (FL) at that wave. Adults reporting no functional difficulties of any kind are classified as healthy at that wave. Between each wave, adults could transition between any two functioning states, and all states could transition to death.

Early-Life Experiences and Education

We include two early-life experiences: childhood health and SES. Adults were asked to rate their health while growing up, from birth to age 16, as excellent, very good, good, fair, or poor. We dichotomized the measure into excellent, very good, or good versus fair or poor.

We created an indicator of cumulative socioeconomic adversity in early life. Because socioeconomic exposures tend to cluster, the indicator allows us to estimate potential dose–response and threshold effects. This is particularly beneficial if it is the quantity of exposures, rather than the quality of any single exposure, that strongly predicts health outcomes, as many mental health studies have shown (Sameroff 2000). Similarly, Cohen and colleagues (2010) advocated quantitative measures of childhood SES that can detect graded associations, particularly because gradation would provide provocative evidence of causality. An index may also provide a better view of the early “social ecology” (Bronfenbrenner 1977) and avoid overestimating the effects of any single exposure (Green et al. 2010). The index has a few drawbacks, however. For example, it assumes the exposures have equal weight and are additive.

The indicator sums dichotomized information on parents’ education, occupation, income, and family structure. The items included mother's education (1 = less than 8 years); father's education (1 = less than 8 years); respondent's perception of whether their childhood family was pretty well-off financially, average, or poor (1 = poor); whether before age 16 financial difficulties caused their childhood family to move to a different place (1 = yes); whether there was a time when their childhood family received help from relatives because of financial difficulties (1 = yes); whether they never lived with their father (1 = yes); and their father's occupation when they were age 16 (1 = blue collar, which includes protective services, farming/fishing/forestry, service, construction and production, and don't know).

Very few respondents experienced six or seven adversities, so we collapsed the index to range from zero to five or more. It is modeled as five indicator variables (0 (the omitted reference), 1, 2, 3, 4, and 5 or more) to explore nonlinearities. In preliminary analyses, we analyzed each of the seven individual survey items. Each predicted functioning, although mothers' and fathers' education were the most significant predictors.

The few cases with missing values were excluded, except for cases missing data only on parent's education. Missing data for mother's and father's education were imputed as less than eight years, consistent with other studies that found adults in the HRS missing data on parents' education were similar on other economic and health variables to adults who reported that their parents had less than eight years of education (Luo and Waite 2005; Montez and Hayward 2011).

Educational attainment is measured with four binary variables indicating 0–11 years, a high school diploma or GED (omitted reference), some college, or a bachelor's degree or higher. Table 1 shows the distribution of early-life experiences and educational attainment.[2]

Age, Race, and Gender

Age is a time-varying, continuous variable from 50 to 100 years used to define the hazard functions for disability and mortality incidence models. All models are adjusted for gender (1 = male; 0 = female) and race/ethnicity (1 = non-Hispanic white (hereafter, white); 0 = non-Hispanic black (hereafter, black)).

Analytic Strategy

The analysis is based on a person-year file in which individuals are aged by one year beginning with their first interview until their year of death or 2008 if they survived the follow-up period. Because the HRS data are collected in two-year intervals, our file imputes a person-year record for the skipped year.[3] The pooling of person-year records across HRS waves assumes a Markov process and no period effects. The total numbers of person-year records for each of the 16 potential transitions (and nontransitions) are shown in Table 2.

The analysis is conducted in two steps. First, health state transition rates are estimated from 16 hazard models reflecting the 16 potential health state transitions. The transition rate is defined by Eq. (1), where P_{ij} is the probability that a transition from state i (e.g., IADL) to state j (e.g., ADL) occurs in the age interval x to $x + n$ (for this study, n = 1 year), given that the adult was in state i at age x.

[2]Although the three experiences—childhood health, childhood SES, and educational attainment—are correlated, the magnitude did not cause collinearity concerns. Kendall Tau's correlation using the person-year file was .09 ($p < .0001$) between childhood health and SES, –.06 ($p < .0001$) between childhood health and educational attainment, and –.32 ($p < .0001$) between childhood SES and educational attainment.

[3]Our data structure assumes that all transitions—functional and deaths—occur at the end of each year, except when death censors a functional transition. In that case, death is assumed to occur midyear. For example, suppose the transition of interest is from healthy to functional limitations between the 1998 and 2000 waves. Among respondents who were healthy in 1998 but reported limitations in 2000, we randomly assign one-half to transition at the end of 1998 (exposure = 1 and transition = 1 in 1998, and exposure = 0 and transition = 0 in 1999) and the other half to transition at the end of 1999 (exposure = 1 and transition = 0 in 1998, and exposure = 1 and transition = 1 in 1999). Respondents who were healthy in 1998 but died in 1999 were assigned exposure = 1 and transition = 0 in 1998, and exposure = 0.5 and transition = 0 in 1999. If they died in 1998, they were assigned exposure = 0.5 and transition = 0 in 1998.

$$\mu_{ij}(x) = lim \frac{P_{ij}(x,n)}{n} \quad \text{as} \quad n \to 0. \quad (1)$$

The transition rates are estimated from multivariate hazard models using PROC LIFEREG in SAS 9.3, which assume that the variation in transition times between states *within* an exposure interval can be described by an exponential survival distribution (i.e., the risk was constant within the age interval). The general form of the hazard models is provided by the series of models that follow, where *ELH* is early-life health, *ELSES* is early-life socioeconomic context, and *ED* is educational attainment. All models include gender and race as covariates, and were adjusted by the sample weights. These models allow us to assess the total effects of early-life health and SES, as well as the degree to which educational attainment mediates the effects.[4]

$$ln\mu_{ij}(x) = \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(ELH) \quad (2.1)$$

$$ln\mu_{ij}(x) = \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(ELSES) \quad (2.2)$$

$$ln\mu_{ij}(x) = \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(ED) \quad (2.3)$$

$$ln\mu_{ij}(x) = \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(ELH) + \beta_{ij3}(ELSES) \quad (2.4)$$

$$ln\mu_{ij}(x) = \beta_{ij0} + \beta_{ij1}(age) + \beta_{ij2}(ELH) + \beta_{ij3}(ELSES) + \beta_{ij4}(ED). \quad (2.5)$$

In analyses not shown, we assessed whether education moderated the association between early-life SES and the transition rates. We did this by adding the interaction between *ELSES* and *ED* to Eq. (2.5), but we used a continuous measure of *ELSES* so that the models estimated 3 rather than 15 interaction terms. For men, 1 of the 16 transitions contained a statistically significant interaction at $p < .05$, but it did not improve the model fit according to the Akaike information criterion. For women, two of the five transitions that contained a significant interaction (FL to IADL, and ADL to Healthy) improved the model. The interactions indicated that higher levels of education had more health benefits for women from socioeconomically advantaged childhoods.

We use the parameter estimates from Eqs. (2.4) and (2.5) for combinations of covariates to calculate predicted hazard rates, which are the inputs into the multistate life tables of active life expectancy. Specifically, we generate matrices of transition rates from Eq. (2.4) and then again from Eq. (2.5), for men and women separately (for women, we include an *ELSES* × *ED* interaction in the transitions for FL to IADL and from ADL to healthy), to estimate total, active, and inactive life expectancy using population-based, multistate life tables. These tables distribute the radix population according to the observed prevalence in each health

[4]We tested a variety of functional forms of the association between age and the risk of disability incidence and mortality. For all transitions, a (ln)linear specification was the best fitting functional form.

state at age 50 in the HRS sample, and then estimate the expectancies in each health state. Detailed descriptions of the procedures for estimating multistate life tables are available elsewhere (e.g., Crimmins et al. 1994).

Results

Before addressing our main research aims, we examine how early-life health and SES predict the 16 transition rates. Figures 1 and 2 summarize the key results from Eqs. (2.1) through (2.5) (full results available from authors). The dark gray bars in Fig. 1 represent antilogs of the hazard model coefficients estimated from models that contain only early-life health (Eq. (2.1)) or SES (Eq. (2.2)) for the six transitions involving functional deterioration. Figure 2 shows the six transitions involving functional improvement. In both figures, the dark gray bars reveal that fair/poor early-life health often predicted unfavorable transitions. For example, the age-specific risk of declining from FL to IADL was [100(1.442 – 1.000)], or 44.2 %, greater for adults who reported fair/poor childhood health than for adults with better childhood health. The risk of improving from FL to healthy was [100(0.724 – 1.000)], or 27.6 %, smaller for adults with fair/poor childhood health. The dark gray bars also show that multiple SES adversities predicted unfavorable transitions—particularly functional deterioration. SES adversities and deterioration exhibited a dose–response relationship (ancillary analyses did not find significant nonlinearities), with three or more adversities consistently predicting an elevated risk of decline ($p < .05$) compared with zero adversities. Lastly, neither early-life health nor SES strongly predicted transitions from the four functioning states to death (results available from authors).

How do early-life health and SES predict transitions net of each other? The light gray bars in Figs. 1 and 2 represent antilogs of the transition rates estimated from Eq. (2.4). The bars reveal a negligible attenuation of early-life health and SES when they are mutually adjusted for each other, which suggests that these experiences fairly independently shape health transitions for the cohorts included in the analysis.

To what extent does educational attainment mediate the association between the two early-life experiences and health transitions? The white bars in Figs. 1 and 2 represent antilogs of transition rates from Eq. (2.5) (models provided in Table 3). There was a negligible mediation of early-life health and some mediation of SES. The mediation was more pronounced among adults who experienced multiple SES adversities because their transition rates were the most elevated.

We now address our main research questions. How many years of total life and active life do individuals who experienced adverse childhood contexts live compared with their counterparts who experienced salubrious contexts, and do they spend a greater or smaller proportion of life functionally impaired? Using the transition rates estimated from Eq. (2.4) within multistate life tables, we estimated total, active (healthy), and inactive (FL or IADL or ADL) life expectancy at age 50 for adults who experienced the most disadvantaged childhoods (fair/poor health and five or more SES adversities) and the most advantaged childhoods (good or better health and no SES adversities). The results in Table 4 reveal that at age 50, adults from disadvantaged childhoods could expect to live roughly three fewer

years than their advantaged peers (3.5 years for white men, 2.9 for black men and women) although the gap was larger (4.5 years) for white women. Gaps in active life expectancy were even more pronounced: 6.5 years for white men (almost double the gap in total life), 6.6 for black men, 5.2 for black women, and 6.3 for white women. In addition, adults raised in disadvantaged childhoods spent a greater percentage of total years of life functionally impaired. For instance, white men from disadvantaged childhoods could expect to live 73 % of their remaining years with some degree of impairment, compared with 55 % for their peers from advantaged childhoods.

How do early-life experiences combine with educational attainment to predict total and active life? Table 5 contains life expectancies for adults from the most- and least-advantaged circumstances. Several findings are noteworthy and apply across race-gender groups. First, the extremes are striking. For example, active life expectancy at age 50 among white men ranged from 4.6 years for the most disadvantaged to 15.3 years for the most advantaged. Second, more years of education predicted more years of total and active life regardless of childhood context, but the gains were somewhat larger for adults from advantaged childhoods. The gains in total life from a college degree (compared with 0–11 years of education) were roughly one year greater for men, and two years greater for women, from advantaged childhoods.[5] However, recall that the moderating effect of education on early-life SES met our "significant and meaningful" threshold for women only. Third, upwardly mobile adults (i.e., disadvantaged childhoods but a college degree) could expect to live 3–4 years longer than downwardly mobile adults (i.e., advantaged childhoods but 0–11 years of education). For example, at age 50, upwardly mobile black women could expect to live an additional 30.1 years compared with 26.4 years among their downwardly mobile peers. In addition, upwardly mobile men could expect to live more active years than downwardly mobile men, but the reverse was found among women. Upward mobility was not as beneficial for women because their active life expectancy[6] was more tightly anchored to early-life experiences than was men's, which concurs with our finding that education provided more health benefits for women from advantaged than disadvantaged childhoods.

Figure 3 shows active life expectancy for 24 combinations of early-life experiences and educational attainment among white men (data for other groups are shown in Online Resource 1). It illustrates that the educational gradient was steeper than the early-life SES gradient; the benefits of a college degree were pronounced. It also reveals the potential for education to extend active life expectancy. Bearing in mind that our tests for a moderating effect of education did not find that education could ameliorate childhood SES adversities, the impressive benefits of upward mobility meant that adults from disadvantaged childhoods who achieved high levels of education often had expectancies similar to or better than adults from advantaged childhoods who achieved low education levels. For example, the top panel

[5]For example, white women from disadvantaged childhoods could expect to live an additional 28.8 years if they had 0–11 years of education versus 32.9 years if they had a college degree, which is a gain of 4.1 years. The gain among white women from advantaged childhoods was 6.4 years. Thus, a college degree conferred 2.3 more years of life for white women from advantaged childhoods compared with their peers from disadvantaged childhoods. The gains were 2.1 for black women, 1.2 for white men, and 1.4 for black men. The differential gains for active life expectancy were 3.3 for white women, 3.5 for black women, 2.1 for white men, and 2.0 for black men.

[6]The total life expectancy gap between the upwardly and downwardly mobile was also slightly smaller for women (3.8 years for white men, 4.2 years for black men, 2.9 years for white women, and 3.7 years for black women).

of Fig. 3 shows that among white men with favorable childhood health, men who experienced *zero* childhood SES adversities but dropped out of high school had a similar active life expectancy as men who experienced *three* adversities but then achieved a high school credential (7.7 vs. 8.3 years) and shorter active life expectancy than men who experienced *five or more* adversities but then achieved a college degree (12.8 years). The patterns were similar for black men. The patterns were not as pronounced among women because, as noted earlier, education had fewer health benefits among women from disadvantaged childhoods.

Discussion

Numerous studies have found provocative evidence that early-life experiences—especially health and SES—exert an indelible influence on adult physical functioning and mortality risk. However, by and large, these studies examined functioning and mortality as distinct processes and assessed prevalence measures, which inherently mask the underlying dynamics of disability incidence, recovery, and mortality selection. Thus, we know little about how early-life experiences *jointly* impact functioning and mortality risk and therefore shape the health experiences of individuals over their projected life cycle—for example, the number of years they can expect to live with and without disability. This study addressed this gap by examining the extent to which total and active life expectancy were shaped by childhood health and SES, and the extent to which educational attainment mediated and moderated the consequences of those experiences among non-Hispanic U.S. adults aged 50–100.

The results revealed several insights into the life-course origins of active life expectancy. First, early-life health and SES independently predicted transitions across degrees of functional ability within the disablement process. The independence does not imply that early-life health and SES are unrelated. Rather, it indicates that after early health is compromised, it can take on a life of its own in shaping adult functional ability.

Second, early-life SES more strongly predicted functional decline than improvement. One hypothesis is that early experiences may leave an indelible stamp on multiple physiologic systems and create a susceptibility to accelerated aging, frailty, and reduced physiologic reserves, while functional improvement may be most amenable to proximal resources, such as income. For instance, one study found that education was more effective at staving off the onset of decline through preventive factors such as healthy lifestyles, while income was more effective at postponing further declines and death through factors like health insurance and medications (House et al. 2005). In addition, income and social ties facilitate emotional resilience to disability (Ubel 2006). Future studies should investigate the reasons for differential effect.

Third, the association between early-life SES and functional decline exhibited a dose–response pattern. This pattern is consistent with many studies of the etiology of mental illness that find it is the quantity of environmental exposures, rather than the quality of any single exposure, that strongly predicts mental illness (Sameroff 2000). By using a categorical measure of SES, we were able to detect that three or more adversities

consistently elevated the risk of decline compared with no adversities. However, this does not imply a threshold. Indeed, we found no statistical evidence of a threshold effect.

Fourth, educational attainment did not mediate the association between childhood health and functional transitions to a meaningful degree, but it partly mediated childhood SES. Although this study was not designed to adjudicate between biological imprint and social pathway processes, this finding is suggestive of imprint processes linking child health and adult functioning (because the association was not attenuated) as well as both social pathway and biological imprint processes linking childhood SES with adult functioning (because some of the association was attenuated). These hypotheses concur with other studies (e.g., Blackwell et al. 2001; Hayward and Gorman 2004; Luo and Waite 2005).

Fifth, total and active life expectancy reflected child and adult experiences for each race-gender group. At age 50, adults who experienced early-life disadvantages lived fewer years of total and active life, and they spent a greater portion of life functionally impaired. Early-life experiences remained important even after we adjusted for educational attainment. Further, more years of education predicted more years of total and active life regardless of childhood context. Although early-life experiences and educational attainment demarcated important gaps in total life expectancy, these factors played an even greater role in active life expectancy. Early-life disadvantage and low levels of education accelerated functional decline such that disadvantaged persons lived more years with functional problems in both absolute and relative terms.

Sixth, the health benefits of education were somewhat greater for adults from advantaged childhoods, although the differential in the underlying transitions rates met our “significant and meaningful” threshold only for women. As a result, women's active life expectancy was more heavily anchored to childhood circumstances than was men's. This finding concurs with a growing number of studies reporting that childhood circumstances appear more consequential for (some measures of) women's than men's health (e.g., Alvarado et al. 2007; Hamil-Luker and O'Rand 2007; Khlat et al. 2009; Maty et al. 2008). Several social and biological mechanisms have been hypothesized to explain the gender difference, but the reasons remain unclear.

Our focus on educational attainment as the marker of adult circumstances reflects several considerations outlined in the introduction. Our results support the view that enhancing access to quality education for all Americans may improve population health (e.g., Dunn et al. 2005; House et al. 2008), with benefits that compound with each generation. For instance, active life expectancy reflected educational attainment more than it did early-life SES, and upwardly mobile individuals generally had similar or better outcomes than downwardly mobile individuals. That being said, elevating education levels among all Americans without concomitantly enhancing broader social and economic conditions, or at least expanding opportunities for adults to translate their education into health and longevity, may dampen the overall benefits on population health.

Although education is a particularly strong and robust predictor of adult health, other dimensions of adult life—for example, occupation, income, health behaviors, and social ties

—also shape health. These dimensions may additionally mediate and moderate the link between childhood circumstances and active life expectancy. Thus, it is important to bear in mind that education is used in this study as an indicator of a wide range of adult circumstances (many of which are strongly tied to education). Future research may want to integrate other dimensions of adult life to glean additional insights into the life course origins of active life expectancy.

Despite its strengths, the study has some limitations. One potential limitation is that functional ability was self-reported, although self-reports appear to be a fairly accurate reflection of actual ability for men and women (Merrill et al. 1997). Another limitation is that childhood conditions were assessed retrospectively. Retrospective measures of childhood SES appear to be reliable (Krieger et al. 1998) and accurate (Batty et al. 2005). In addition, studies of the reliability and validity of retrospective measures of childhood health support their careful use in population research (Elo 1998; Haas 2007; Haas and Bishop 2010; Smith 2009). For example, when asked to recall childhood health on the five-point ordinal scale during two different survey waves, 60 % of HRS respondents gave identical responses in both waves, and 33 % gave responses that were just one category apart, and the responses did not simply project current health status (Smith 2009). The measure also appears to be valid because it correlates with birth weight (Haas 2007). However, the measure does have some drawbacks and should be interpreted cautiously. For instance, the reliability of the measure is lower among blacks and less-educated adults (Haas 2007; Smith 2009). In addition, the interpretation of what it means to have experienced poor health as a child may vary by factors such as birth cohort and region. Future studies may want to explore whether the ordinal measure of childhood health provides additional insights. Future studies may also want to examine race differences, although studies using the HRS have not found black-white differences in how early-life SES predicts functioning (Haas and Rohlfsen 2010; Luo and Waite 2005). Lastly, extrapolating these findings to other ages, cohorts, or regions must be approached with caution because the results are likely moderated by contextual factors.

Conclusions

Among older U.S. adults, early-life circumstances and educational attainment jointly shaped total life expectancy, and they played an even greater role in active life expectancy. Adults who experienced good health and socioeconomic advantages in childhood lived more total years of life, lived more years of active life, and spent a smaller portion of life functionally impaired than adults who experienced poor health and socioeconomic adversities in childhood, net of educational attainment. Higher levels of educational attainment did not ameliorate the health consequences of disadvantaged childhoods. However, because education had a larger impact on health than did childhood SES, adults from socioeconomically disadvantaged childhoods who achieved high levels of education often had total and active life expectancies that were similar to or better than those of adults from advantaged childhoods who achieved low education levels.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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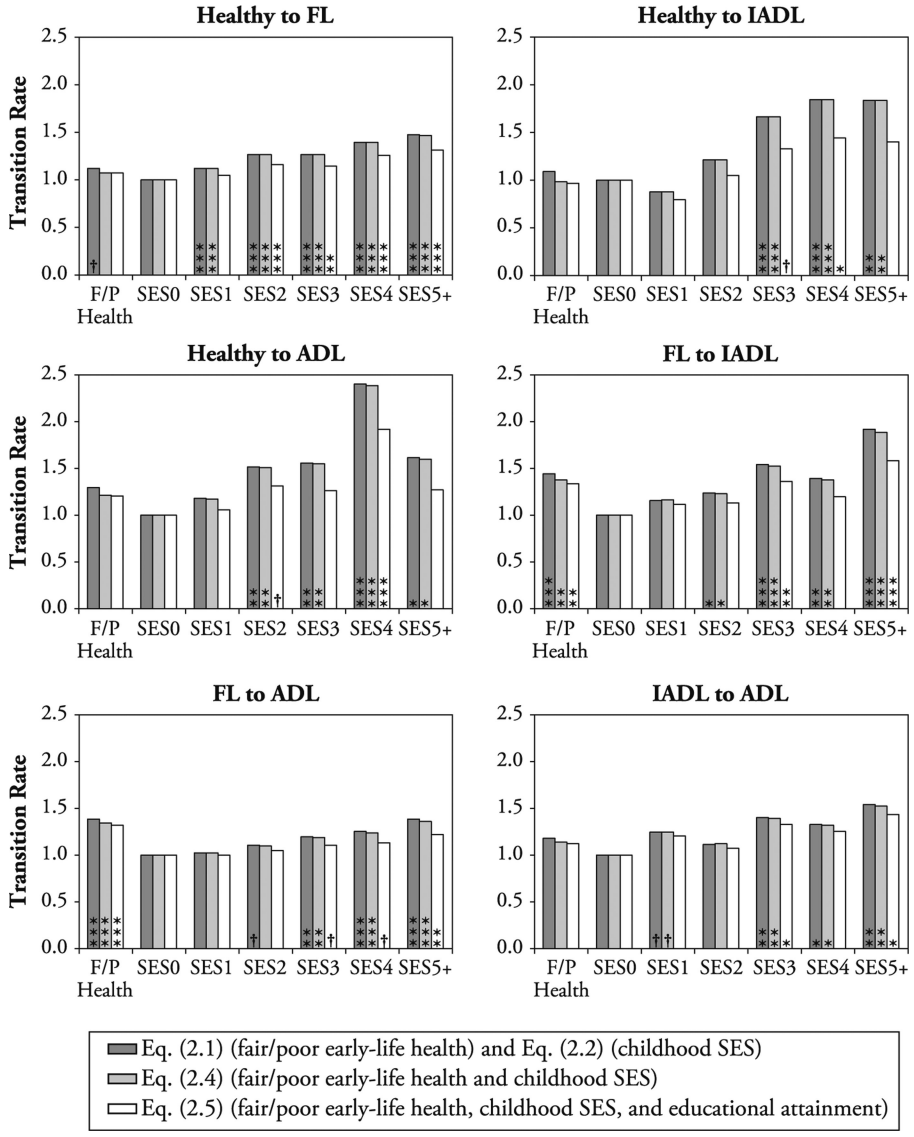

Fig. 1.

Antilogs of transition rate coefficients reflecting deterioration in functioning. FL = functional limitations; IADL = difficulty with instrumental activities of daily living; ADL = difficulty with basic activities of daily living; Healthy = no FL, IADL, or ADL; and SES = socioeconomic context. $^{\dagger}p < .10$; $*p < .05$; $**p < .01$; $***p < .001$

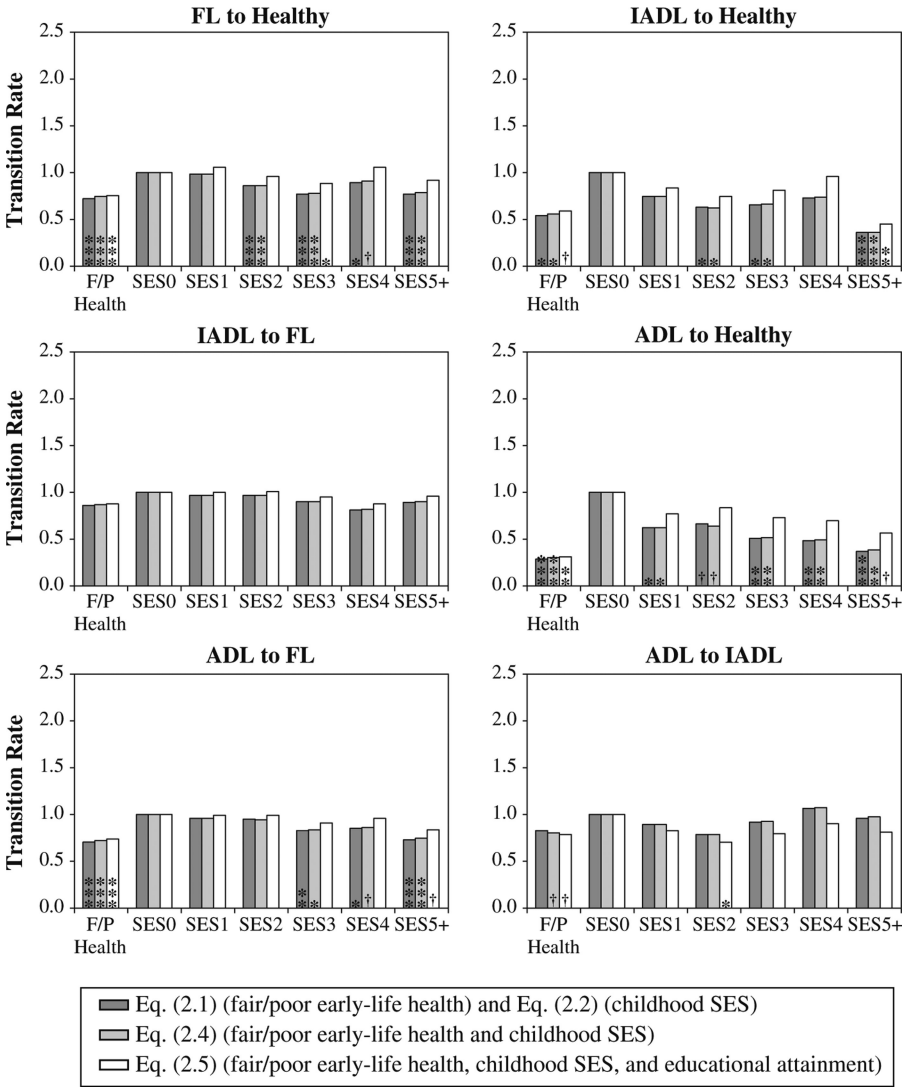

Fig. 2.

Antilogs of transition rate coefficients reflecting improvement in functioning. FL = functional limitations; IADL = difficulty with instrumental activities of daily living; ADL = difficulty with basic activities of daily living; Healthy = no FL, IADL, or ADL; and SES = socioeconomic context. $^{\dagger}p < .10$; $*p < .05$; $**p < .01$; $***p < .001$

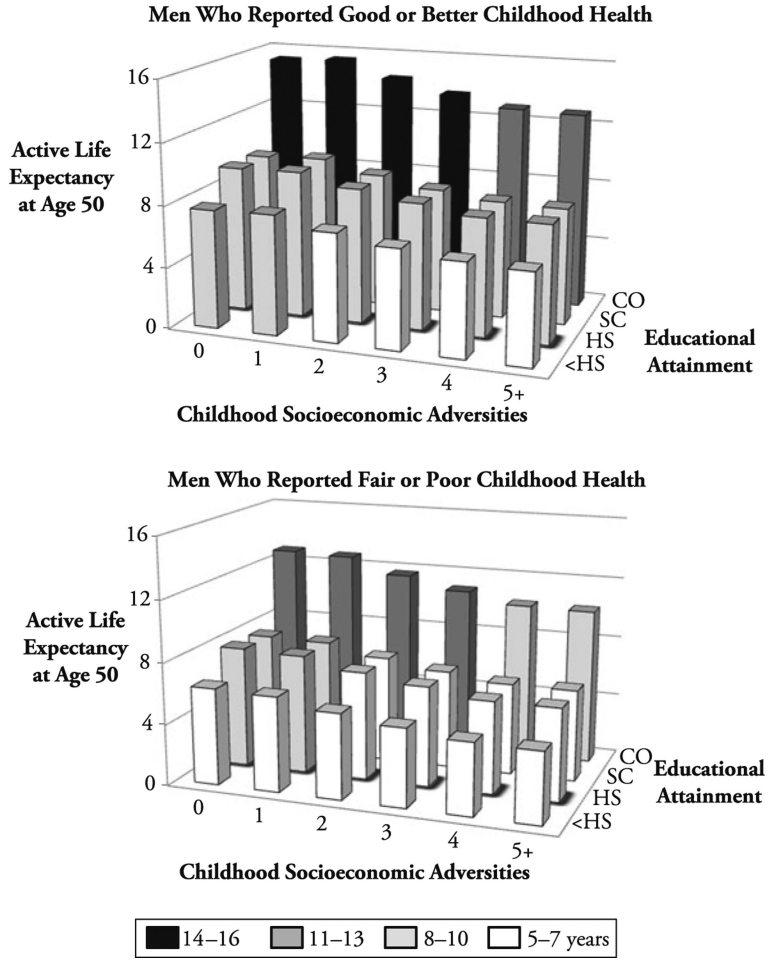

Fig. 3.

Active life expectancy at age 50 for white men across combinations of early-life health, early-life socioeconomic context, and educational attainment. <HS = less than high school; HS = high school; SC = some college; and CO = bachelor's degree or higher

Table 1

Weighted distribution of person-year records among analytic sample (percentages, except for age)

	Mean
Age (in years)	66.0
Male	43.9
Black	9.6
Father Had Less Than 8 Years of Education	35.2
Mother Had Less Than 8 Years of Education	25.6
Childhood Family Pretty Well-off	6.9
Childhood Family Average	64.9
Childhood Family Poor	28.2
Ever Moved for Financial Reasons	17.1
Ever Received Help From Relatives	13.0
Never Lived With Father	7.3
Father's Occupation[a]	
White collar	22.5
Blue collar	77.6
Number of Early-Life Socioeconomic Adversities	
0	14.6
1	30.0
2	19.3
3	18.3
4	11.0
5-7	6.9
Early-Life Health	
Excellent	53.2
Very good	25.1
Good	16.1
Fair or poor	5.7
Educational Attainment	
0-11 years	17.7
High school diploma or GED	36.9
Some college	22.7
Bachelor's degree or higher	22.7
Unweighted Number of Person-Year Records	148,232

Notes: Totals may not add to 100.0 because of rounding.

[a]White collar includes management (6.7), professional (5.4), sales (7.0), office and administrative (2.4), and military (1.0). Blue collar includes protective services (1.1); farming, fishing, and forestry (17.5); service (2.1); construction and production (36.0); and do not know (20.9).

Table 2

Unweighted distribution of person-year records by functioning state at the beginning and end of each person-year interval

	State at End of Interval					
State at Beginning of Interval	**Healthy**	**FL**	**IADL**	**ADL**	**Death**	**Total**
Healthy	37,048 (81.5)	6,986 (15.4)	495 (1.1)	581 (1.3)	324 (0.7)	45,434 (100)
FL	5,206 (7.4)	58,048 (83.0)	1,482 (2.1)	4,140 (5.9)	1,083 (1.5)	69,959 (100)
IADL	264 (3.7)	980 (13.6)	4,466 (62.1)	1,108 (15.4)	369 (5.1)	7,187 (100)
ADL	202 (0.8)	2,666 (10.4)	759 (3.0)	20,037 (78.1)	1,988 (7.7)	25,652 (100)
Total	42,720	68,680	7,202	25,866	3,764	148,232

Notes: FL = functional limitation; IADL = difficulty with instrumental activities of daily living; ADL = difficulty with basic activities of daily living; and Healthy = no FL, IADL, or ADL. In each cell, the top value is the number of person-year records, and the bottom value in parentheses is the row percentage.

Table 3

Antilogs of hazard model coefficients predicting transition rates between health states from early-life health, early-life socioeconomic context, and educational attainment (Eq. (2.5))

			Early-Life Socioeconomic Context					Educational Attainment		
State at Start of Interval	**State at End of Interval**	**Fair/Poor Early-Life Health**	**1**	**2**	**3**	**4**	**5-7**	**0-11 Years**	**Some College**	**Bachelor's Degree or Higher**
Improvement										
FL	Healthy	0.754***	1.057	0.963	0.887*	1.054	0.922	0.864**	1.045	1.365***
IADL	Healthy	0.587†	0.840	0.748	0.808	0.958	0.450**	0.779	0.966	1.543**
IADL	FL	0.877	0.996	1.009	0.951	0.878	0.955	0.917	1.048	1.075
ADL	Healthy	0.308**	0.767	0.835	0.726	0.698	0.568†	1.034	1.425†	2.197***
ADL	FL	0.736***	0.993	0.992	0.911	0.960	0.835†	0.824***	1.023	1.092
ADL	IADL	0.787†	0.826	0.703*	0.791	0.903	0.814	1.227*	0.955	0.787
Deterioration										
Healthy	FL	1.072	1.050	1.160***	1.146**	1.260***	1.315***	0.966	0.983	0.742***
Healthy	IADL	0.971	0.799	1.051	1.330†	1.446*	1.399	1.861***	1.062	0.767*
Healthy	ADL	1.205	1.057	1.308†	1.260	1.920***	1.274	1.323*	0.840	0.677***
FL	IADL	1.332**	1.111	1.131	1.358**	1.194	1.583***	1.483***	0.981	0.953
FL	ADL	1.316***	1.000	1.048	1.110†	1.134†	1.224**	1.269***	1.012	0.952
IADL	ADL	1.121	1.207	1.071	1.328*	1.253	1.434*	1.036	1.063	0.826†
Alive to Dead										
Healthy	Dead	0.507†	0.870	0.553**	0.493***	0.730	0.591†	1.262	0.933	0.468***
FL	Dead	1.158	0.869	0.885	1.017	0.783†	0.676*	1.222*	1.012	0.789**
IADL	Dead	0.980	0.826	0.732	0.809	0.973	0.895	1.091	1.115	0.801
ADL	Dead	1.104	1.038	1.056	0.956	0.992	0.932	1.063	0.974	0.976

Notes: All models control for age, gender, and race. The omitted reference for early-life socioeconomic context is 0, and the omitted reference for educational attainment is high school diploma or GED.

† $p < .10$

* $p < .05$

** $p < .01$

*** $p < .001$ (two-tailed tests)

Table 4

Total, active, and inactive life expectancy at age 50 by race and gender for combinations of early-life health and socioeconomic context

		Active	Inactive				
	Total	Healthy	FL	IADL	ADL	%of Years Impaired[a]	%of Race-Sex Group
White Men							
Fair/poor child health, 5+ SES adversities	26.69	7.11	10.90	1.69	6.99	73.4	0.5
Good or better child health, 0 SES adversities	30.17	13.64	12.45	0.86	3.22	54.8	15.8
Black Men							
Fair/poor child health, 5+ SES adversities	23.50	7.02	7.20	1.57	7.70	70.1	1.9
Good or better child health, 0 SES adversities	26.36	13.65	8.26	0.83	3.63	48.3	1.9
White Women							
Fair/poor child health, 5+ SES adversities	30.23	4.33	13.39	1.68	10.83	85.7	0.9
Good or better child health, 0 SES adversities	34.77	10.64	16.79	1.49	5.85	69.4	14.9
Black Women							
Fair/poor child health, 5+ SES adversities	27.14	3.26	9.90	1.70	12.28	88.0	2.4
Good or better child health, 0 SES adversities	30.03	8.50	13.16	1.50	6.87	71.7	2.5

Notes: SES = socioeconomic context.

[a]The number of years spent with functional limitations (FL), difficulty with instrumental activities of daily living (IADL), or difficulty with basic activities of daily living (ADL) out of the total expected years of life.

Table 5

Total, active, and inactive life expectancy at age 50 by race and gender for combinations of early-life health, early-life socioeconomic context, and educational attainment

		Active	Inactive				
	Total	Healthy	FL	IADL	ADL	% of Years Impaired[a]	% of Race-Sex Group
White Men							
Fair/poor child health, 5+ SES adversities, <HS	25.22	4.63	10.34	2.06	8.18	81.6	0.20
Fair/poor child health, 5+ SES adversities, CO	30.08	10.17	11.83	1.45	6.63	66.2	0.07
Good or better child health, 0 SES adversities, <HS	26.33	7.72	12.64	1.44	4.54	70.7	0.31
Good or better child health, 0 SES adversities, CO	32.36	15.32	12.73	0.87	3.44	52.7	9.22
Black Men							
Fair/poor child health, 5+ SES adversities, <HS	22.70	5.25	7.36	1.69	8.40	76.9	1.33
Fair/poor child health, 5+ SES adversities, CO	27.51	11.44	8.21	1.15	6.71	58.4	0.00
Good or better child health, 0 SES adversities, <HS	23.31	8.66	8.81	1.16	4.69	62.9	0.18
Good or better child health, 0 SES adversities, CO	29.48	16.81	8.57	0.67	3.44	43.0	0.39
White Women							
Fair/poor child health, 5+ SES adversities, <HS	28.81	3.89	12.13	1.59	11.20	86.5	0.35
Fair/poor child health, 5+ SES adversities, CO	32.92	6.15	14.54	1.74	10.50	81.3	0.05
Good or better child health, 0 SES adversities, <HS	29.99	7.49	13.64	2.21	6.65	75.0	0.35
Good or better child health, 0 SES adversities, CO	36.39	13.04	16.29	1.55	5.51	64.2	6.38
Black Women							
Fair/poor child health, 5+ SES adversities, <HS	26.28	2.89	9.15	1.62	12.62	89.0	1.16
Fair/poor child health, 5+ SES adversities, CO	30.11	4.78	11.48	1.69	12.16	84.1	0.25
Good or better child health, 0 SES adversities, <HS	26.39	5.70	10.80	2.19	7.70	78.4	0.08
Good or better child health, 0 SES adversities, CO	32.33	11.05	13.37	1.49	6.42	65.8	0.83

Notes: SES = socioeconomic context; <HS = 0-11 years; and CO = bachelor's degree or higher.

[a]The number of years spent with functional limitations (FL), difficulty with instrumental activities of daily living (IADL), or difficulty with basic activities of daily living (ADL) out of the total expected years of life.