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J Health Soc Behav. 2006 September ; 47(3): 209–226.**Early-Life Origins of the Race Gap in Men's Mortality*****DAVID F. WARNER** and

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Abstract

Using a life course framework, we examine the early life origins of the race gap in men's all-cause mortality. Using the National Longitudinal Survey of Older Men (1966–1990), we evaluate major social pathways by which early life conditions differentiate the mortality experiences of blacks and whites. Our findings indicate that early life socioeconomic conditions, particularly parental occupation and family structure, explain part of the race gap in mortality. Black men's higher rates of death are associated with lower socioeconomic standing in early life and living in homes lacking both biological parents. However, these effects operate indirectly through adult socioeconomic achievement processes, as education, family income, wealth, and occupational complexity statistically account for the race gap in men's mortality. Our findings suggest that policy interventions to eliminate race disparities in mortality and health should address both childhood and adult socioeconomic conditions.

The race gap in men's mortality is a national tragedy.¹ In 2002, black males at birth had a life expectancy of 68.8 years, compared to 75.1 years for white males (Kochanek et al. 2004). The gap narrows with age, yet at age 45 white men in 2002 could expect to live almost 4.5 years longer than could black men (32.9 years vs. 28.5 years).

Much of the mortality gap reflects differences in chronic conditions. Middle-aged black men, for example, have higher prevalence and incidence rates of hypertension, diabetes, and stroke (Hayward et al. 2000; Smith and Kington 1997). Blacks are more likely to have multiple fatal conditions and more functional limitations than are whites (Hayward et al. 2000; Hayward and Heron 1999; Smith and Kington 1997). As a result, black men live fewer years *and* live more years with a chronic condition (Crimmins and Saito 2001; Hayward, Friedman, and Chen 1996; Hayward and Heron 1999). These disparities underscore the differential ability of blacks and whites to invest in careers and family relationships and to enjoy returns on these investments.

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¹Throughout this paper, we refer to black-white differences as "race" differences. While such a comparison does not represent all racial and ethnic heterogeneity [see Hayward and Heron (1999)], the health of blacks and whites represents the extreme comparison in many cases. Moreover, the data we use are limited to these two groups.

Here, we use a life course framework (Elder 1999) to investigate how early life conditions combine with conditions in adulthood to give rise to the race gap in mortality (Elo and Preston 1992; Kuh and Ben-Shlomo 1997; Preston, Hill, and Drevenstedt 1998). This approach is appealing given the predominance of fatal conditions that begin to emerge in midlife, after years of subclinical development (Hertzman, Frank, and Evans 1994). Although previous research has examined the impact of early-life disease (Blackwell, Hayward, and Crimmins 2001; Elo and Preston 1992) and socioeconomic conditions on adult health (Gunnell et al. 1996; Hayward and Gorman 2004; Power and Peckham 1990; Preston et al. 1998), none to our knowledge has explicitly examined the role of early life in generating race differences. A life course framework (Kuh and Ben-Shlomo 1997) may be particularly important for understanding the race gap in mortality, as early life circumstances initiate “social chains of risk” (Kuh et al. 1997). We expect that early life conditions matter for differences in later-life health, insofar as racially based social stratification differentially allocates the early life resources necessary for the production and maintenance of good health (O’Rand 2001).

PREVIOUS RESEARCH

Adult Socioeconomic Status and Mortality

Disparities in socioeconomic status, particularly education and income, account for much of the race gap in health and mortality (Hayward et al. 2000; Preston and Taubman 1994; Williams and Collins 1995; Williams and Wilson 2001). Education is important partly because blacks’ lower educational attainment limits access to resources that protect health (Link and Phelan 1995). For example, black men’s lower levels of education increase their chances of working in blue-collar jobs with lower cognitive demands and greater physical demands (Farkas et al. 1997), characteristics associated with negative health outcomes (Marmot and Shipley 1996; Marmot et al. 1997a; Moore and Hayward 1990). Blacks’ lower education also constrains their ability to generate income throughout the work career. Higher incomes allow for the long-term purchase of better health through choice of residential community, health care, and the ability to avoid exposure to environmental toxins (Hayward, Pienta, and McLaughlin 1997; Williams and Collins 1995). Blacks’ lower income trajectories also constrain wealth accumulation, which provides resources to meet future health care demands, especially among those no longer working (House et al. 1994; Rogers, Hummer, and Nam 2000; Smith and Kington 1997; Williams and Collins 1995).

Health behaviors are an additional mechanism by which blacks’ lower socioeconomic status may lead to poor health outcomes. Smoking, alcohol abuse, and obesity consistently raise the risk of morbidity and death (National Center for Health Statistics 2001; Rogers et al. 2000; Winkleby et al. 1992). Health behaviors appear to mediate a portion of the association between adult socioeconomic status and health (Hayward and Gorman 2004; Mulatu and Schooler 2002). Overall, people with higher educational attainment are less apt to smoke, more likely to drink alcohol in moderation, and less likely to be underweight or obese (Rogers et al. 2000; Ross and Wu 1996). Although blacks are disadvantaged relative to whites in socioeconomic status, blacks do not appear to have uniformly worse health

practices than whites. For example, while blacks are more likely to be obese (National Center for Health Statistics 2001), they are less likely to smoke (Williams and Collins 1995).

Prior research demonstrates that adult social and economic characteristics affect health outcomes and explain blacks' higher mortality. However useful for explaining race differences in mortality, the adult socioeconomic attainment model rests on a static, and often ill-defined, conceptualization of socioeconomic status (Williams and Collins 1995). To understand better the social origins of the race gap in men's mortality, we examine ways in which early life conditions combine with adult conditions to generate mortality risks.

Early-Life Origins of the Social and Economic Gradients in Adult Mortality

From a biomedical standpoint, interruptions in early-life development may affect adult mortality because early life is a sensitive period for the development of organ systems and regulatory processes. For example, major infections in early life are associated with a range of adult health outcomes (Blackwell et al. 2001), e.g., increased risk of heart disease (Buck and Simpson 1982; Mathews, Whittingham, and Mackay 1974), poorer autoimmune function (Paunio et al. 2000), and lung problems (Barker 1998). Other negative adult health outcomes may result from environmental toxin exposure and nutritional deficits during early life (Gunnell et al. 1996; Holland et al. 2000; Perry 1997) and in utero (Barker 1998; Barker 1999; Martyn, Barker, and Osmond 1996).

Social and economic conditions in early life may also influence later-life mortality, although there is debate over whether early-life factors affect adult health directly (albeit via biomedical pathways) or indirectly through adult achievement processes and health behaviors. Physical and psychosocial environmental conditions in socially disadvantaged homes place children at greater risk of poverty, inadequate health care, infections and other environmental hazards, poorer nutrition, stress, and family conflict (Ben-Shlomo and Kuh 2002). Some studies, for example, report persistent effects of early-life socioeconomic conditions net of—and along with—the effects of adult socioeconomic achievement (Claussen, Smith, and Thelle 2003; Kuh et al. 2002; Mheen et al. 1998; Rahkonen, Lahelma, and Huuhka 1997). Other studies report that early-life socioeconomic conditions are related to adult health outcomes, but primarily through adult socioeconomic status (Hayward and Gorman 2004; Lynch et al. 1994). These studies point to early life as the period when individual life chances are determined, and these life chances, in turn, indirectly influence adult health outcomes.

Early-life family relationships may also influence adult health, as parental divorce and family conflict are associated with higher risks of illness and mortality (Lundberg 1993; Schwartz et al. 1995). Although family effects are often robust in the presence of controls for other early-life and adult characteristics, there are few studies focusing on family effects, indicating the need for additional research.

Black Americans' persistent social, economic, and family disadvantages from early life into old age point to the importance of assessing the early-life origins of the race gap in mortality. Early life shapes access to physical and psychosocial resources, creating a situation where American blacks confront health-compromising conditions across the life

course. As noted above, however, the life course mechanisms giving rise to the race gap in adult health outcomes are far from clear. Differences in study designs, measures, outcomes, and sample populations all cloud current understanding of how life course social and economic conditions combine to stratify adult health outcomes—and by extension give rise to the race gap in these outcomes. Although we are unable to resolve these issues in the current study, we are nonetheless able to assess how major life course mechanisms account for the race gap in American men's later-life mortality. We evaluate the role of direct and indirect effects of early life conditions, as well as possible race differences in the pathways giving rise to the race gap in mortality. Overall, we expect that blacks experience social chains of risk that lead to higher mortality rates compared to whites.

METHODS

Data and Sample

The National Longitudinal Survey of Older Men (NLS), although not originally conceptualized for this purpose, largely meets the requirements to enable us to test our life course model because it contains biographical information about early life conditions, adult socioeconomic status, health behaviors, and mortality. The NLS is a nationally representative sample of American men ages 45–59 in 1966, followed through 1990. Respondents provided biographical information about changes in many domains of adult life, including socioeconomic circumstances, marriage, and health behaviors. Of the approximately 5,000 respondents in 1966, 2,693 deaths were recorded through 1990 via death certificates from states' vital statistics offices or widow proxy reports. The effective sample size is 2,346 deaths (87%) after we delete about 13 percent of the sample due to missing data on adult income, wealth, and occupation. The patterns of missing data for adult indicators do not indicate any systematic bias (Hayward and Gorman 2004).

Although information is available on major causes of death, we focus on all-cause mortality for several reasons. First, there are many fewer deaths for causes other than cardiovascular disease (about 54% of all deaths), and we encounter statistical problems associated with small numbers of deaths for most causes within the black subsample. Second, cancer deaths (the second-leading cause) are not differentiated by organ site, a factor important in cancer etiology and assessing early life influences (Leon and Ben-Shlomo 1997). Third, there is uncertainty about the quality of cause-of-death reports.

With respect to early-life social conditions implicated in a life course approach to adult health, the NLS offers information about socioeconomic, family, and residential conditions when the respondent was age 15. Although information on early-life disease exposure, central to theories of life course epidemiology (Kuh and Ben-Shlomo 1997), is unavailable, the information on both early life and adult circumstances allows us to test alternative *social pathways* associated with the black-white mortality gap (Kuh et al. 1997). Information on early-life disease exposure (Blackwell et al. 2001; Kuh and Ben-Shlomo 1997) would allow analyses of the biomedical mechanisms by which social conditions in early life have an effect. However, information on infectious disease exposure is unlikely to lead to substantively different conclusions about the *social pathways* between early life and later-life mortality, given our results. Exposure to infectious diseases is not random in the

population and is linked to early-life social conditions (Coggon et al. 1993; Dedman et al. 2001; Preston and Taubman 1994).

The sample eligibility-age of the NLS means that our results are *conditional on surviving to age 45*. Thus, the NLS allows us to model men's mortality from age 45 until the last observed age at death at age 83. This period of life is when socioeconomic conditions strongly influence mortality (Hertzman et al. 1994; House et al. 1994), as the effects of long-term exposure to poverty, social stressors, and environmental toxins, as well as the cumulative effects of health behaviors, begin to manifest through chronic disease. Beginning in midlife, mortality is overwhelmingly the end result of at least one chronic disease process (Hertzman et al. 1994; National Center for Health Statistics 2001). Nonetheless, selectivity is a potential issue. The health experiences of blacks and whites differed in early life for the NLS birth cohorts, with higher infant and early-life mortality rates among blacks (Preston and Haines 1991). We suspect that those black men included in the NLS are those with better early-life circumstances or who are unusually robust. Such selectivity should minimize observed race differences, providing a conservative test of our hypotheses. However, prior research provides little guidance on the age-specificity of early-life effects or life course mechanisms.

We estimated race-specific life expectancies for the panel to evaluate the representativeness of the NLS men's mortality experiences. We calculated the expectancies based on the results of a Gompertz hazard model regressing the log risk of death on age and race.² We used the parameter estimates from the statistical model to calculate predicted life-table death rates, and we then calculated race-specific life tables.³ Table 1 presents these results, as well as period life expectancies for the U.S. population in 1980 and 1990 for whites (Anderson 1999) and blacks (Elo 2001); 1980 is roughly the center of the period when the sample was at risk.⁴

It is difficult to directly compare the expectancies, given differences in methodology (the NLS expectancies are based on a statistical model, while the Vital Statistics expectancies are calculated using an occurrence/exposure approach) and population coverage (the NLS excludes the institutionalized population), as well as the fact that the NLS expectancies are based on a real cohort's experiences, compared to the period-based vital statistics expectancies. Nonetheless, the NLS expectancies for both blacks and whites are relatively close to the vital statistics estimates, especially those for 1990. In addition, the race gap in men's mortality in the NLS mirrors that reported by the offices of vital statistics.

²We also estimated the hazard model including a term for the interaction between race and age to determine whether black and white men have different hazards of death in any given age-interval (Ferraro and Farmer 1996). The interaction was not significant, so we deleted it from the model. Likewise, we do not differentiate mortality experiences by birth cohort because the effect was not significant in preliminary models.

³Although we only observed death up to age 83, and thus our model-based life tables predict out-of-age-range death rates, we use the predicted rates to simulate the complete mortality experience of the NLS cohort.

⁴We use Elo's (2001) expectancies rather than those calculated by the National Center for Health Statistics because Elo adjusts for age misreporting and underenumeration among blacks.

Measures

Early life conditions—Respondents provided retrospective information about their family when they were age 15. Socioeconomic information included the household head's occupational status (dummy-coded professional, managerial, clerical, sales, crafts, foremen and operatives, household service, service, farmer, farm laborer, military, and laborer, the last of which is the reference) and educational attainment (dummy-coded 13 or more years, 9–12 years, or 7–8 years, with less than 6 years as the reference). Respondents also reported on family structure (living with both biological parents, father and stepmother, mother and stepfather, mother only, father only, a male relative, living on his own, or in another arrangement, dummy-coded where living with both biological parents serves as the reference) and whether the respondent's mother worked outside of the home (dummy-coded). As men who did not live with their mothers could not report on their mother's work status, we include these men in the analysis by dummy-coding them as “missing” on mother's work. Early-life community size (dummy-coded rural farm, rural nonfarm, a town with fewer than 25,000 residents, a suburban area, a smaller city with between 25,000 and 100,000 residents, and a large city with 100,000 or more residents, which is the reference category) allows us to explore differences by size of place. We also control for nativity (coded 1 if foreign-born, 0 otherwise) because immigration rates were very high during the early twentieth century.⁵ We retain all cases by including dummy variables indicating whether the respondent was missing for applicable variables, given that the distribution of missing data for the early-life indicators was not random.

It is important to consider whether these measures of early life conditions reflect unhealthy conditions that might be linked to adverse adult health and mortality. Prior work generally supports this idea. Households in the early twentieth century had lower rates of infant mortality when the father worked as a farmer or salesman and when both parents were literate. Infant-mortality risks were also lower when an adult male wage earner was present, although living with both biological parents was most protective. Growing up in a rural area, especially on a farm, was also associated with lower mortality (Preston and Haines 1991; Preston et al. 1998). Having a father from a lower social class in early life is associated with high blood pressure and elevated serum cholesterol levels (Blane et al. 1996; Coggon et al. 1993), which are risk factors for cardiovascular disease.

A potential concern regarding the NLS early-life measures is their reference to age 15. A life course perspective sensitizes us to the importance of timing in lives (Elder 1999). However, we must assume that there was relative stability in respondents' experiences during early life and that dramatic social mobility was an exception.⁶ As a result, our analysis deals with

⁵We also attempted to account for early-life region of residence in our preliminary models. Unfortunately, we did not have a measure of region of residence during early life and used residence in the South at the time of the first interview as a proxy. When residence in the South was included in the models, the coefficients for occupation of the household head were reduced significantly, corresponding with the geographic distribution of occupations, particularly farming, in the early twentieth century. We excluded the measure from further analyses, however, because it did not significantly improve our model fit; also, occupation of the household head, in addition to being a more precise measure, is more consistent with our theoretical suppositions (see also note 13).

⁶As some of the men in our sample born after 1914 were age 15 or younger during the Great Depression, this raises some concern as to the assertion of such stability. However, cohort parameters are not statistically significant and are unaffected by the inclusion of childhood socioeconomic conditions, regardless of model specification. Likewise, we did not detect any cohort differences in recall of childhood living conditions. Together, these suggest that there are no cohort differences in the degree of early-life social instability faced by the NLS men.

exposure to early-life circumstances but not the timing or duration of that exposure. Measuring family structure and socioeconomic status at a fixed age in early life is consistent with prior research (Dedman et al. 2001; Lynch et al. 1994; Lynch, Kaplan, and Salonen 1997; Marmot et al. 1997b; Rahkonen et al. 1997).

Adult characteristics—We specify education as a measure of adult socioeconomic attainment, although we are aware that some men, particularly in the cohorts studied here, may have ceased education before age 15. However, education is often conceptualized as the anchor point of occupational and earnings trajectories in adulthood. Thus, treating education as an adult characteristic allows us to estimate a traditional adult model of mortality (Hertzman et al. 1994; Kitagawa and Hauser 1973; Preston and Taubman 1994) and evaluate whether early life conditions operate through more proximate circumstances, including education. We measured education with a set of three dummy variables: “9–12 years,” “13 years or more,” and “8 years or less” (omitted reference).

We include time-varying measures of family income and net asset wealth (in 1983 dollars) as measures of adult financial resources. Given the skewed distribution of these measures, we logarithmically transformed them after adding a constant to eliminate negative and zero values. We also include information about the longest occupation held over a man’s career, to capture exposure to physical demands and stressful conditions, which in turn are associated with mortality. We use four conditions of the work environment associated with adult health (Hayward, Friedman, and Chen 1998; Marmot et al. 1997a): substantive complexity (comprising the level of creativity, cognitive skills, and autonomy required), physical and environmental demands, social skills, and manipulative skill demands. Work characteristics listed in the fourth edition of the *Dictionary of Occupational Titles* (Miller et al. 1980) were matched to respondent reports of the longest occupation held [see Moore and Hayward (1990)].

The two final time-varying measures of adult social conditions are residence (three dummy variables for “urban,” “suburban,” and “rural” [reference]) and marital status (four dummy variables for “married,” “never married,” “divorced,” and “widowed” [reference]). Urban men have higher age-adjusted mortality rates than men living in rural areas, despite the historically lower access to and quality of health care services in rural areas (Hayward et al. 1997). Married men tend to have better health outcomes and lower mortality risks than men who are either divorced or widowed (Lillard and Waite 1995; Pienta, Hayward, and Jenkins 2000).

Health behaviors—Respondents (or their proxies) provided information in 1990 about lifetime smoking and alcohol use. We code smoking using a series of dummy variables for whether a man “never smoked,” was a “former smoker,” or was “currently smoking” (reference). Alcohol consumption is a measure of respondents’ usual daily consumption, captured with a set of four dummy variables for “1–2 drinks,” “3–4 drinks,” “5 or more drinks,” and never drinking alcohol (reference). The mortality risks associated with alcohol use generally follow a U shape, with moderate alcohol use more protective than either abstinence or heavy consumption. Our smoking and alcohol measures reference *lifetime* typical usage rather than only current behavior, minimizing endogeneity problems due to

cessation in response to declining health (Rogers et al. 2000).⁷ We also calculate a body-mass index (BMI) based on self-reported height and body weight in 1973. We categorize the index into five groups defined by quintiles, assigning labels to each group for the ease of interpretation and using each as the criterion for a dummy variable in a set of variables to measure the concept. The body mass categories are “extreme underweight” (BMI < 20), “underweight” (BMI 20–23), “normal weight” (BMI 23.1–25), “overweight” (BMI 25.1–27.5), and “obese” (BMI 27.6–52.1). Underweight serves as the reference group. These category designations allow us to model BMI’s U-shaped association with mortality, similar to alcohol use.⁸ Each health behavior measure also includes a separate dummy variable (coded 1) to retain respondents with missing data; such cases with missing data were coded as 0 on the corresponding health behavior dummy variables.

Analytic Strategy

We estimate nested discrete-time hazard models to evaluate the ways that early life conditions contribute to the race gap in men’s mortality. We assess the role of these conditions by comparing changes in the coefficient for black race across models, and we conduct the analyses in two stages. First, we regress the risk of death on each early-life condition separately. Second, we estimate a series of nested models that regress the risk of death on the full set of early-life characteristics and then adult socioeconomic characteristics and health behaviors. By comparing changes in the coefficients across models, we can assess the life course pathways accounting for the race gap in mortality.

In preliminary analyses, we stratified our models by race and tested for the equality of coefficients across models to determine whether the parameter effects were similar for whites and blacks (Clogg, Petkova, and Haritou 1995).⁹ The test of equality revealed no significant differences between the parameters for whites and blacks; in fact, the size and direction of the coefficients were remarkably similar for both groups. The basic life course mechanisms connecting early life and mortality appear to operate similarly for black and white men. However, as we document below, white men were far more likely than black men to occupy advantageous positions in early life.

FINDINGS

Race Differences in Early Life Conditions

We present weighted sample means in Table 2 to examine the degree to which early-life experiences differ between blacks and whites.¹⁰ Black men, in general, report growing up in more disadvantaged social and economic environments than do white men. Compared to

⁷Given that proxies provided information about smoking and alcohol use for decedents in 1990, there is some concern about the accuracy of this information. However, our findings show the expected associations between health behaviors and mortality risk and thus suggest that any reporting bias by proxies is minimal.

⁸Although we are unable to model body mass as a time-varying characteristic, our analysis shows the expected associations between extremes in body mass and mortality risk. While multiple measures would be preferable, we are convinced that the single measure of body mass accurately captures the health risks associated with being either underweight or overweight.

⁹This is akin to estimating a single saturated model, where a race interaction is specified for every covariate.

¹⁰We also present weighted sample means for adult characteristics in Table 2, which largely correspond to what we know about race differences in adult socioeconomic resources and health behaviors (Cherlin 1992; Smith 1997; Williams and Collins 1995). However, given space limitations, we leave these to the reader to examine in detail.

whites, at age 15 black men lived in households where the head was more likely to have six or fewer years of education (31.9% vs. 24.3%, respectively) and less likely to have completed any schooling beyond high school (7.2% vs. 1.9%, respectively). Most men in these cohorts, however, did not know the educational attainment of the household head.¹¹ Black men were also more likely to have lived in households where the head worked in a lower-status occupation, especially farming. Black men were far less likely to live with both biological parents (56.2%) than were whites (78.3%). Among men residing with their mother at age 15, more than twice as many blacks as whites had a mother who worked for pay. Having an employed mother largely reflects economic disadvantage for these cohorts (Elder 1999), as indicated by the low educational and occupational attainment of the household head, and this is particularly the case for blacks who are twice as likely as whites to have resided in mother-only households.

The communities in which the NLS men lived reflect the occupational distribution of the household head. Although all men were more likely to have lived in rural areas than elsewhere, black men were much more likely to have lived on a farm than were whites (50.1% vs. 34.6%, respectively). Whites were more likely to have grown up in large cities.

Race Differences in the Effect of Early Conditions on Mortality

We present weighted hazard ratios for the effect of early life conditions on the race gap in men's mortality in Table 3. Model 1 is a baseline estimate of mortality as a function of age, race, cohort, and nativity. The risk of death for black men is approximately 1.33 times the risk for white men at every age. This is the overall race gap in mortality risk that we attempt to account for statistically in the remaining models. Models 2–5 estimate the mortality risk associated with our five blocks of early life conditions separately. Model 6 presents the net effects on mortality when all of the early life conditions are included simultaneously.

We find little evidence that the household head's education has an influence on mortality among the men in these cohorts, although it does slightly reduce the race gap (model 2). We see clearer evidence, however, that the occupation of the household head has an influence on the race gap in mortality, although not as one might expect (model 3). The relative risk for black men is about 2 percent larger in model 3 than in model 1 [$((1.351 - 1.326) / 1.326) \times 100 = 1.89$], suggesting that had black men lived in households with the same occupational mix as whites, the race gap in mortality would be slightly *larger*. The mortality "advantage" for black men results from the greater likelihood of having grown up with a household head that worked as a farmer. Sons of farmers have a risk of mortality 0.73 times that of sons of laborers, about 27 percent lower.

Incorporating family structure and mother's work status in model 4 reduces the relative risk of mortality for black men by slightly more than 5 percent [$((1.326 - 1.256) / 1.326) \times 100 = 5.30$]. This reduction occurs because living apart from one or both biological parents and having a mother who worked for pay increases the risk of mortality, and both of these

¹¹This distribution is consistent with overall patterns of education in the general population in the early part of the twentieth century (U.S. Bureau of the Census 2000). Furthermore, additional analyses did not detect any systematic bias in the report of household head's education, other than residing in a father-absent household at age 15 (not shown).

conditions are more common among blacks (see Table 2). For example, black men are twice as likely as whites are to have been living on their own at age 15 and therefore to be missing the mother's work status. This is associated with a mortality risk about 1.39 times the risk of living with a biological father and a mother who did not work for pay.¹² In fact, once we account for the effect of mother's work status, all of the nonparental living arrangements are associated with a higher risk of mortality than living with two biological parents where the mother did not work for pay. These results coincide with a family socioeconomic investment model; two-biological parent households are best able to invest in offspring (Amato and Booth 1997). Our results also show that it is not simply access to male wages that reduces mortality risk, as men who lived with an at-home biological mother and a stepfather at age 15 have a mortality risk that is 1.34 times the risk of those who lived with both biological parents.

As expected, growing up on a farm is associated with lower mortality compared to men raised in a large city (model 5) (Preston et al. 1998). Men who lived on a farm in their early years face a mortality risk only 0.84 times the risk of men who lived in a large city. Recall that black men were more likely to have lived on farms, and had they not been living on farms, their relative risk of mortality would be marginally higher (about 3%).

When all of the early life conditions are included in the model simultaneously (model 6), we find that the relative risk of mortality for black men is reduced by about 4.5 percent [$((1.326 - 1.267) / 1.326) \times 100 = 4.45$]. It is important to note that this reduction is a combination of offsetting effects, i.e., controlling for protective conditions (e.g., farm residence) as well as detrimental ones (e.g., family structure). We now examine the role of adult socioeconomic and health behaviors in accounting for the race gap in mortality.

A Life Course Model of Race Differences in Mortality

The life course perspective suggests that differential access to social and economic advantages across the life course may lead to health disparities between blacks and whites. As noted above, some of the race gap appears to originate in early life, yet it is clear that other factors—perhaps in adulthood—come into play as well. The basic issue is how early life and adult conditions combine to account for the race gap in mortality. Is the race gap due to a combination of early life and adult influences? Or, is the race gap reflective of early-life factors that operate indirectly through adult conditions? We test these alternatives using a series of nested models that add adult socioeconomic factors sequentially to observe changes in both the parameter estimates for race and those for the early life conditions (see Table 4). To facilitate comparisons, we have continued the model numbering sequence and included the full early-life model (model 6) from Table 3.

¹²In preliminary analyses (not shown), we did not find a statistically significant interaction between family living arrangements and *biological* mother's work status. However, it makes little theoretical sense to interpret the effects separately. Thus, to calculate the combined hazard ratios for various family living arrangements, multiply the ratios for family structure and the appropriate ratio for mother's work status. Recall that we code men who did not reside with their mothers as "missing" on this measure. To estimate the effect of living with a biological father and stepmother, multiply the hazard ratio for family structure (0.546) by the hazard ratio for missing mother's work status (2.063), for a hazard ratio of 1.126. Thus, men who lived with their father and stepmothers have a risk of death that is about 13% higher than the risk for men who live with both biological parents and had mothers who did not work for pay (the reference category).

Model 7 estimates the risk of mortality as a function of early-life characteristics and adult education. The results are consistent with the idea that the effect of early life conditions on the race gap in mortality operates indirectly, in part through educational attainment because of the reductions in the effect of household head occupation. However, we also see that education has an effect on the race gap, net of the early life measures, with the relative risk of mortality for black men diminishing about 4 percent between models 6 and 7 [$((1.267 - 1.218) / 1.267) \times 100 = 3.87$]. We do not observe the complete mediation of the effect of household head's occupational attainment on mortality via offspring education, consistent with prior research (Lynch et al. 1994; Lynch et al. 1997; Power, Manor, and Matthews 1999; Rahkonen et al. 1997).

Next, we add net assets and total family income to the model. Financial resources explain part of the link between mortality and education, reducing the relative risk associated with education by between 3 percent and 14 percent when added to the model (see model 8). Thus, part of the race gap in men's mortality appears to arise indirectly: Differences in the occupation of the household head between blacks and whites lead to differentials in educational attainment and then in adult financial status, which, in turn, lead to different risks of death. It is also clear, however, that adult socioeconomic status independently contributes to the race gap in mortality, as reflected in the change in the effect size for race. After controlling for family income and wealth, the age-specific risk of death for black men is only 1.11 times the risk for white men, and the difference is no longer statistically significant.

Although we have statistically accounted for the race gap in men's mortality, we continue to add adult characteristics to the model to investigate the potential multiple pathways by which race influences mortality risks. In models 9 and 10, respectively, we find that occupational complexity and marital status behave in the expected ways and, combined, almost eliminate the (statistically nonsignificant) race gap in mortality. We also find the expected relationships between health behaviors and mortality: Extremes in body mass, not drinking, usually having more than three drinks daily, and currently smoking all increase the risk of mortality (see model 11). However, health behaviors explain *none* of the race gap in men's mortality once the socioeconomic pathway, particularly education (Ross and Wu 1996), is incorporated into the model. In fact, in a model including only early-life education and health behaviors, the parameter for black is almost identical to that shown in model 11 (model not shown). However, the introduction of health behaviors does substantially reduce the effects of family structure and mother's work status. There is some evidence, therefore, that part of the race gap in mortality operates through early-life family structure as it influences adult health behaviors (e.g., black men's greater chances of being reared in a single-parent family increases the risk of obesity, which then increases the risk of death). Nonetheless, our models demonstrate that adult socioeconomic achievement is the primary mechanism accounting for the race gap in mortality.

Given our finding that early life conditions affect the risk of mortality indirectly via adult socioeconomic status, it is important to consider whether investigations into the origins of the race gap in mortality have overestimated the role of adult social and economic factors by not incorporating early life conditions explicitly. Model 12 presents the estimates of the

traditional adult socioeconomic attainment model. Education, income, wealth, occupational characteristics, area of residence, and marital status eliminate the race gap in mortality, reducing the hazard ratio for race from 1.326 in the baseline model (model 1, Table 3) to a nonsignificant 1.008. Our results do not imply any statistical bias when early life conditions are absent from the model. On the contrary, we witness remarkable consistency in the estimated effects of adult characteristics between models 10 and 12 in Table 4.¹³

Although most of the race gap in mortality appears to stem from differences in adult social and economic circumstances, recall that entering early life conditions alone into the model reduced the baseline relative risk of mortality for black men by 4.5 percent—an effect similar in magnitude to that of education. While the absence of early-life social and economic conditions does not bias the effect of adult socioeconomic indicators in models of mortality, failing to include these measures ignores the fact that adult socioeconomic status partly reflects achievement trajectories initiated by early-life socioeconomic status. The adult socioeconomic indicators that explain the race gap in men’s mortality are themselves outcomes of disparate early life experiences. For example, in additional analyses (not shown), we regressed the adult socioeconomic status indicators on the full set of early-life indicators, controlling for race, age and cohort. The findings indicate that our early-life social and economic indicators explain approximately 33 percent of the variance in educational attainment, 15 percent of the variance in income, and 10 percent of the variance in assets among the NLS men. Thus, failure to examine early life conditions underemphasizes their indirect role in generating the race gap in mortality, and this has important implications for policy interventions aimed at eliminating the race gap in mortality and in health more generally. We return to this point below.

DISCUSSION AND CONCLUSION

The purpose of this study was to explore the role of early life conditions in generating the race gap in older men’s mortality. We argued that early life shapes socioeconomic status in adulthood, given the differential allocation of opportunities for adult socioeconomic achievement between blacks and whites. Consequently, we hypothesized that early life conditions are an important, and often overlooked, source of the race gap in older men’s mortality.

Using the National Longitudinal Survey of Older Men, we find that the early-life experiences of black and white men born in the early twentieth century were dramatically different. Compared to whites, black men were more likely to grow up in households of lower socioeconomic standing, less likely to live with both biological parents, more likely to have a mother who worked for pay, and more likely to live in rural areas, especially on farms. This constellation of early-life social conditions, with some exceptions, disadvantages black men in terms of later-life mortality. The exceptions are the greater likelihood of living

¹³The one set of indicators that do change between the models are those for area of residence. When we exclude early life characteristics, we find a significantly higher risk of death associated with living in urban or suburban areas, relative to rural areas. A logistic model predicting area of residence demonstrates the increased odds that men who resided in rural areas or who lived in households where the head was a farmer at age 15 would live in rural areas as adults (model not shown). This suggests that area-of-residence effects are likely present in the adult-only model because of the tendency of these cohorts to reside in areas similar to those in which they lived at age 15.

in a household headed by a farmer, which affords black men mortality protection to some extent. Nevertheless, the dissimilar early-life experiences of blacks and whites account for a portion—but not the majority—of the elevated mortality risk black men face in later life.

Our analysis also shows that, for the most part, early-life social conditions *indirectly* affect the race gap in mortality. Introducing adult social and economic conditions into the model substantially attenuates the effects associated with the household head's occupation. Family-of-origin capital translates into better health via education, and, consequently, higher income and more wealth accumulation. The race gap in mortality also stems from early-life family structure, which in turn affects adult health behaviors, especially those that affect body mass.

Our results also provide clear evidence that most of the race gap in older men's mortality reflects adult achievement. Education, family income, wealth, and occupational complexity combine to eliminate the race gap in mortality, statistically speaking. The importance of these adult factors for understanding why black men live so many fewer years than white men is inescapable. Yet their origin in early life is equally undeniable.

Still, we urge caution when interpreting these results, because a number of analytic issues are unresolved. One concern is that the mortality experiences examined in this analysis refer to ages 45–83, with the bulk of the deaths occurring among men in their 50s, 60s, and 70s. We cannot consider the consequences of premature mortality for our analysis, which may be tied more strongly and directly to early life. Thus, the pattern of our results may be sensitive to the age group we are examining. A number of measurement issues also are unresolved. Do recall difficulties or systematic underreporting or overreporting bias reports of early-life status? How do we interpret the effects of family structure, particularly concerning the development of health behaviors, in the absence of direct measures of family relationships, nutrition, and the like? How would our results be different if we had information on the duration and timing of early-life social and economic conditions, rather than simply exposure to these conditions?

Previous research (Blackwell et al. 2001; Elo and Preston 1992; Leon and Ben-Shlomo 1997) also suggests that early life directly affects health in old age via disease exposure. Although we are unable to model such early-life disease exposure, we observe no residual early-life effects to which we could attribute such an exposure effect. Any early-life effects unaccounted for by adult economic well-being are related to health behaviors. It may be that the modeling of all-cause mortality is insensitive to different disease etiologies, the outcome of specific early-life exposure (Kuh and Ben-Shlomo 1997; Leon and Ben-Shlomo 1997). Unfortunately, we were unable to model cause-specific mortality given the reasons discussed above. We are confident, however, that socioeconomic disparities in early life contribute to the race gap in men's mortality, but we must be cautious in stating why such social pathways exist. Additional research needs to address these questions, some of which necessitate new data-collection efforts with an eye toward the importance of early life.

Although we can statistically eliminate the mortality disadvantage experienced by blacks in our sample, this does not mean that race per se does not matter. Early life conditions that arise from race-based stratification have long-lasting and pervasive, albeit indirect, effects on

adult mortality. Black Americans begin life in families that possess fewer resources and are less able to maximize those resources in adulthood given their limited opportunity structure, as evidenced by their lower education, lower income, and fewer assets. Moreover, additional insults accumulated in adulthood exacerbate the socioeconomic deficits that black men carry forward from early life. Blacks have dramatically fewer assets (Oliver and Shapiro 1997; Smith 1997) and are less likely to get and stay married than whites (Cherlin 1992). The unequal distribution of resources across race groups thus initiates a cascading series of events and circumstances—a social chain of risk—that systematically challenges the ability of black men to preserve their life chances. Although the nature of some of the mechanisms remains uncertain, inequality in early life persists across the life course (Ferraro and Farmer 1996; O’Rand and Henretta 1999).

Current policy directions toward ameliorating race disparities in health by providing health care to and facilitating health behavior changes among adults are effective in treating chronic health conditions or delaying their onset. However, such interventions must swim against a tide of countervailing forces rooted in the racial stratification of socioeconomic resources across the life course. At a minimum, the socioeconomic origins of the race gap in men’s mortality most likely undercut worthy public health policies to reduce health disparities. Policy interventions that aim to reduce the race gap in men’s mortality must include actions that unambiguously address lifetime—that is, both early-life and adult—socioeconomic disadvantage. Although federal health policy tends to be age-targeted and focused primarily on health care, this study adds to the mounting evidence that health is multifactorial, and policies that benefit children’s social and economic well-being will ultimately benefit their adult health (Elo and Preston 1992; Hayward and Gorman 2004; Kuh and Davey Smith 1997; Kuh et al. 1997). Thus, efforts to increase educational parity across communities and provide economic support to poor families will ultimately be *preventive* health policies. Besides improving the well-being of adults, such policies will benefit society as well. Improvements in the health capital of the population will reduce the fiscal burden of providing care for prolonged chronic diseases. Furthermore, improvements in population health will increase economic productivity by reducing or eliminating the premature disability and death of African Americans (Hayward et al. 1996; Hayward and Heron 1999). Recent federal initiatives to broaden health care coverage for children are an important first step in addressing early-life disadvantage. The challenge is to move beyond this narrow focus.

NOTES

¹Throughout this paper, we refer to black-white differences as “race” differences. While such a comparison does not represent all racial and ethnic heterogeneity [see Hayward and Heron (1999)], the health of blacks and whites represents the extreme comparison in many cases. Moreover, the data we use are limited to these two groups.

²We also estimated the hazard model including a term for the interaction between race and age to determine whether black and white men have different hazards of death in any given age-interval (Ferraro and Farmer 1996). The interaction was not significant, so we deleted it from the model. Likewise, we do not differentiate mortality experiences by birth cohort because the effect was not significant in preliminary models.

³Although we only observed death up to age 83, and thus our model-based life tables predict out-of-age-range death rates, we use the predicted rates to simulate the complete mortality experience of the NLS cohort.

⁴We use Elo's (2001) expectancies rather than those calculated by the National Center for Health Statistics because Elo adjusts for age misreporting and underenumeration among blacks.

⁵We also attempted to account for early-life region of residence in our preliminary models. Unfortunately, we did not have a measure of region of residence during early life and used residence in the South at the time of the first interview as a proxy. When residence in the South was included in the models, the coefficients for occupation of the household head were reduced significantly, corresponding with the geographic distribution of occupations, particularly farming, in the early twentieth century. We excluded the measure from further analyses, however, because it did not significantly improve our model fit; also, occupation of the household head, in addition to being a more precise measure, is more consistent with our theoretical suppositions (see also note 13).

⁶As some of the men in our sample born after 1914 were age 15 or younger during the Great Depression, this raises some concern as to the assertion of such stability. However, cohort parameters are not statistically significant and are unaffected by the inclusion of childhood socioeconomic conditions, regardless of model specification. Likewise, we did not detect any cohort differences in recall of childhood living conditions. Together, these suggest that there are no cohort differences in the degree of early-life social instability faced by the NLS men.

⁷Given that proxies provided information about smoking and alcohol use for decedents in 1990, there is some concern about the accuracy of this information. However, our findings show the expected associations between health behaviors and mortality risk and thus suggest that any reporting bias by proxies is minimal.

⁸Although we are unable to model body mass as a time-varying characteristic, our analysis shows the expected associations between extremes in body mass and mortality risk. While multiple measures would be preferable, we are convinced that the single measure of body mass accurately captures the health risks associated with being either underweight or overweight.

⁹This is akin to estimating a single saturated model, where a race interaction is specified for every covariate.

¹⁰We also present weighted sample means for adult characteristics in Table 2, which largely correspond to what we know about race differences in adult socioeconomic resources and health behaviors (Cherlin 1992; Smith 1997; Williams and Collins 1995). However, given space limitations, we leave these to the reader to examine in detail.

¹¹This distribution is consistent with overall patterns of education in the general population in the early part of the twentieth century (U.S. Bureau of the Census 2000). Furthermore, additional analyses did not detect any systematic bias in the report of household head's education, other than residing in a father-absent household at age 15 (not shown).

¹²In preliminary analyses (not shown), we did not find a statistically significant interaction between family living arrangements and *biological* mother's work status. However, it makes little theoretical sense to interpret the effects separately. Thus, to calculate the combined hazard ratios for various family living arrangements, multiply the ratios for family structure

and the appropriate ratio for mother's work status. Recall that we code men who did not reside with their mothers as "missing" on this measure. To estimate the effect of living with a biological father and stepmother, multiply the hazard ratio for family structure (0.546) by the hazard ratio for missing mother's work status (2.063), for a hazard ratio of 1.126. Thus, men who lived with their father and stepmothers have a risk of death that is about 13% higher than the risk for men who live with both biological parents and had mothers who did not work for pay (the reference category).

¹³The one set of indicators that do change between the models are those for area of residence. When we exclude early life characteristics, we find a significantly higher risk of death associated with living in urban or suburban areas, relative to rural areas. A logistic model predicting area of residence demonstrates the increased odds that men who resided in rural areas or who lived in households where the head was a farmer at age 15 would live in rural areas as adults (model not shown). This suggests that area-of-residence effects are likely present in the adult-only model because of the tendency of these cohorts to reside in areas similar to those in which they lived at age 15.

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Life Expectancies for Black and White Males: National Longitudinal Survey (NLS) of Older Men, U.S. Vital Statistics, and Elo's Adjusted Estimates for Black Americans

TABLE 1

E(x)	NLS		1980		1990	
	Whites	Blacks	Whites	Blacks	Whites	Blacks
45	30.8	27.6	29.6	25.6	31.1	26.9
55	22.7	19.8	21.3	18.8	22.6	19.4
65	15.6	13.3	14.3	12.7	15.2	13.1
75	10.0	8.2	8.9	7.9	9.4	8.4

Notes: Vital statistics life expectancy values are presented for whites for 1980 and 1990 (Anderson 1999). Black life expectancies are adjusted for age misreporting and the under-registration of births and infant deaths [see Elo (2001) for a detailed discussion of the procedures]. The black expectancies shown for 1990 in this table are actually an average of life table values for 1985 and 1989–1991 and thus center on the late 1980s rather than 1990.

TABLE 2

Early Life and Adult Socioeconomic Characteristics of White and Black Men Ages 45–83, National Longitudinal Survey of Older Men (1966–1990), Weighted Means

<i>Demographic Characteristics</i>	White	Black
Died in interval	2.42	3.03
Age (years)	62.52	61.85
Cohort		
1906–1910	23.75	21.24
1911–1915	33.20	33.69
1916–1921	43.05	45.07
R foreign-born	6.41	3.50
<i>Early Life Characteristics (when R 15)</i>		
Education of head of household		
Less than 6 years	24.32	31.91
7–8 years	21.99	9.81
9–12 years	11.53	7.29
13 or more years	7.19	1.85
Missing	34.97	49.15
Occupation of head of household		
Professional	4.21	2.32
Manager	13.17	3.72
Clerical	2.06	.49
Sales	3.49	.20
Crafts	14.45	3.73
Operative	12.54	9.10
Private household worker	1.01	7.49
Service	3.65	4.20
Farmer	31.28	47.46
Farm laborer	.98	4.79
Laborer	5.88	8.87
Military	.10	.09
Missing	7.20	7.55
Family structure		
Mother and father	78.26	56.20
Father and stepmother	1.71	2.18
Mother and stepfather	1.94	2.33
Father only	3.13	4.16
Mother only	8.05	16.13
Male relative	2.47	5.93
Other arrangement	3.60	11.26
Living on own	.85	1.81

<i>Demographic Characteristics</i>	White	Black
Mother's paid work status		
Did not work	67.32	43.03
Worked	1.46	25.03
Missing	22.22	31.94
Community size		
Large city (100,000+)	2.53	13.81
Smaller city (25,000–100,000)	1.84	11.81
Suburb of a large city	2.48	1.02
Town less than 25,000	28.38	17.54
Rural, non-farm	3.15	5.74
Rural, farm	34.62	5.08
<i>Adult Characteristics</i>	White	Black
Education		
Less than 8 years	33.35	62.94
9–12 years	46.40	3.18
13 or more years	2.25	6.87
Financial resources ^d		
Assets	114,215.30	31,286.21
Family income	030,666.88	19,224.84
Occupational characteristics ^b		
Manipulative skill	–.06	–.02
Physical demands	.33	.70
Social skill	–.18	–.56
Substantive complexity	–.04	–.72
Residence area		
Urban	28.61	57.71
Suburban	4.83	13.53
Rural	3.55	28.75
Marital status		
Married	88.65	78.27
Divorced	3.90	1.62
Widowed	3.56	8.15
Never married	3.88	2.96
Body mass index		
< 20 (extreme underweight)	3.09	2.95
20–23 (underweight)	15.48	18.57
23.1–25 (normal weight)	2.03	16.28
25.1–27.5 (overweight)	29.15	21.62
27.6–52.1 (obese)	19.28	27.17
Missing	12.97	13.41
Daily alcohol consumption		
No drinks	52.59	5.45

<i>Demographic Characteristics</i>	White	Black
1–2 drinks	23.24	23.89
3–4 drinks	5.31	4.66
5 or more drinks	6.10	5.30
Missing	12.76	15.69
Smoking behavior		
Currently smoking	46.01	49.16
Never smoked	24.72	26.57
Quit smoking	17.82	1.77
Missing	11.45	13.50

Note: R = respondent. All variables are dummy-coded and may be interpreted as percents, unless otherwise indicated. Group differences are significant at $p < .05$.

^aMean, 1983 dollars.

^bMean value of scale. See text for details.

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TABLE 3
 Effect of Early Life Characteristics on the Race Gap in Men’s Mortality, National Longitudinal Survey of Older Men (1966–1990), Weighted Hazard Ratios

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	.000 ***	.000 ***	.000 ***	.000 ***	.000 ***	.000 ***
<i>Demographic Characteristics</i>						
Age	1.082 ***	1.082 ***	1.083 ***	1.084 ***	1.082 ***	1.085 ***
Black	1.326 ***	1.303 ***	1.351 ***	1.256 ***	1.363 ***	1.267 **
Cohort (1906–1910 reference)						
1911–1915	.974	.980	.965	.967	.978	.959
1916–1921	.946	.954	.927	.951	.937	.934
R foreign-born	.821 *	.823 *	.811 *	.803 *	.789 *	.779 **
<i>Early Life Characteristics (when R 15)</i>						
Education of head of household (6 years reference)						
7–8 years		.959				.916
9–12 years		1.045				1.014
13 or more years		.926				.969
Missing		1.069				1.001
Occupation of head of household (laborer reference)						
Professional			.700 **			.694 *
Manager			.760 **			.751 **
Clerical			.925			.929
Sales			.867			.855
Crafts			.872			.854
Operative			1.078			1.060
Private household worker			.882			.872
Service			.807			.813
Farmer			.733 ***			.805 *
Farm laborer			.837			.825
Military			.603			.700

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Missing			.880			.847
Family structure (mother and father reference)						
Father and stepmother				.546***		.534***
Mother and stepfather				1.336*		1.297
Father only				.633***		.623***
Mother only				.987		.971
Male relative				.700**		.719*
Other arrangement				.715**		.700**
Living on own				.679		.656
Mother's paid work status						
Worked				1.267***		1.215**
Missing				2.063***		2.004***
Community size (large city 100,000+ reference)						
Smaller city (25,000–100,000)					1.016	.996
Suburb of a large city					1.249	1.125
Town less than 25,000					1.003	1.006
Rural, non-farm					1.158	1.153
Rural, farm					.840**	.913
Log-likelihood	10216.6	10214.1	10195.1	10121.4	10205.1	10098.1

* $p < .05$;

** $p < .01$;

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

Notes: R = respondent. N of deaths = 2,346 (unweighted).

Effect of Early Life and Adult Characteristics on the Race Gap in Men's Mortality, National Longitudinal Survey of Older Men (1966–1990), Weighted Hazard Ratios

TABLE 4

	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
Intercept	.000***	.000***	8.093	6.215	6.726	4.953	2.230
<i>Demographic Characteristics</i>							
Age	1.085***	1.085***	1.084***	1.084***	1.083***	1.089***	1.081***
Black	1.267**	1.218**	1.111	1.034	.000	1.042	1.008
Cohort (1906–1910 reference)							
1911–1915	.959	.962	.976	.981	.979	.979	.993
1916–1921	.934	.964	.994	.996	.997	1.040	1.003
R foreign-born	.779**	.767**	.787*	.775**	.777**	.748**	.798**
<i>Early Life Characteristics (when R 15)</i>							
Education of head of household (6 years reference)							
7–8 years	.916	.967	.961	.968	.963	.941	
9–12 years	1.014	1.117	1.143	1.154	1.148	1.134	
13 or more years	.969	1.099	1.135	1.153	1.145	1.158	
Missing	.999	1.008	1.007	1.005	1.007	.957	
Occupation of head of household (laborer reference)							
Professional	.694*	.799	.820	.847	.842	.943	
Manager	.751**	.832	.842	.858	.857	.892	
Clerical	.929	.978	.999	1.049	1.035	1.179	
Sales	.854	.939	.952	.968	.944	1.083	
Crafts	.854	.897	.890	.913	.912	.987	
Operative	1.060	1.084	1.063	1.067	1.065	1.111	
Private household worker	.872	.882	.905	.905	.896	.962	
Service	.813	.865	.860	.873	.892	.940	
Farmer	.805*	.820	.817	.838	.845	1.151	
Farm laborer	1.019	.815	.815	.803	.811	.805	
Military	.700	.782	.780	.777	.700	.807	

	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
Missing	.847	.903	.888	.902	.902	.967	
Family structure (mother and father reference)							
Father and stepmother	.534***	.518***	.513***	.526***	.518***	.727	
Mother and stepfather	1.297	1.247	1.230	1.223	1.215	1.247	
Father only	.623***	.615***	.602***	.593***	.591***	.811	
Mother only	.971	.932	.911	.912	.908	.906	
Male relative	.719*	.687**	.672**	.668**	.670**	1.024	
Other arrangement	.700**	.670***	.654***	.649***	.648***	.850	
Living on own	.656	.697*	.591*	.595*	.583*	.731	
Mother's paid work status							
Worked	1.215**	1.210**	1.196*	1.184*	1.178*	1.123	
Missing	2.044***	2.018***	2.034***	2.034***	2.046***	1.150*	
<i>Community Size (large city 100,000+ reference)</i>							
Smaller city (25,000–100,000)	.996	.995	.998	1.016	1.004	1.049	
Suburb of a large city	1.207	1.168	1.163	1.178	1.157	1.267	
Town less than 25,000	1.006	.994	.996	1.013	1.010	1.030	
Rural, non-farm	1.153	1.099	1.100	1.114	1.108	1.166	
Rural, farm	.913	.855	.845	.869	.865	.916	
<i>Adult Characteristics</i>							
Education (8 years reference)							
9–12 years	.803***	.805***	.827***	.840***	.846**	.871**	.880**
13 or more years	.647***	.647***	.740***	.803**	.813*	.845*	.847*
Financial resources							
Net assets (Ln)	.880***	.880***	.880***	.880***	.895***	.939***	.886***
Total family income (Ln)	.451***	.451***	.451***	.451***	.457***	.438***	.513***
Occupational characteristics							
Manipulative skill				.973	.971	.954	1.011
Physical demands				1.029	1.031	1.022	.996
Social skill				1.043	1.042	1.015	1.045

	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12
Substantive complexity		.886 ***		.896 **		.917 *	.881 ***
Residence area (rural reference)							
Urban		1.070		1.075		1.040	1.158 **
Suburban		1.036		1.045		1.022	1.120 *
Marital status (widowed reference)							
Married				.737 ***		.712 ***	.733 ***
Divorced				1.065		.881	1.060
Never married				.649 ***		.590 ***	.745 **
<i>Health-Related Behaviors</i>							
Body mass index (< 20 reference)							
20–23 (underweight)						.852	
23.1–25 (normal weight)						.748 *	
25.1–27.5 (overweight)						.827	
27.6–52.1 (obese)						.960	
Missing						2.208 ***	
Daily alcohol consumption (no drinks reference)							
1–2 drinks						.787 ***	
3–4 drinks						.912	
5 or more drinks						1.120	
Missing						2.012 ***	
Smoking behavior (current smoker reference)							
Never smoked						.742 ***	
Former smoker						.708 ***	
Missing						.605 ***	
Log-likelihood	10098.1	10078.7	10052.9	10043.9	10028.9	9826.1	10143.7

* $p < .05$;

** $p < .01$;

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

Notes: R = respondent. N of deaths = 2,346 (unweighted). Ln = variables logarithmically transformed to account for skewed distribution.

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