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EDUCATIONAL DIFFERENTIALS IN U.S. ADULT MORTALITY: AN EXAMINATION OF MEDIATING FACTORS

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Abstract

We use human capital theory to develop hypotheses regarding the extent to which the association between educational attainment and U.S. adult mortality is mediated by such economic and social resources as family income and social support; such health behaviors as inactivity, smoking, and excessive drinking; and such physiological measures as obesity, inflammation, and cardiovascular risk factors. We employ the NHANES Linked Mortality File, a large nationally representative prospective data set that includes an extensive number of factors thought to be important in mediating the education-mortality association. We find that educational differences in mortality for the total population and for specific causes of death are most prominently explained by family income and health behaviors. However, there are age-related differences in the effects of the mediating factors. Higher education enables individuals to effectively coalesce and leverage their diverse and substantial resources to reduce their mortality and increase their longevity.

Keywords

education; mortality; longevity; NHANES

1. Introduction

Educational differences in U.S. adult mortality have been extensively studied but remain a national priority: the recently released *Healthy People 2020* public health initiative includes as a central focus the social determinants of health and as an overarching goal the

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elimination of health disparities (U.S. Dept. of Health and Human Services [DHHS] 2010). Several recent studies have shown that educational differences in mortality widened over the last two decades (Jemal et al. 2008; Meara, Richards, and Cutler 2008; Masters et al. 2012; Miech et al. 2011; Montez et al. 2011). However, understanding the factors that are responsible for, or mediate, the education-mortality relationship remains underdeveloped (Cutler and Lleras-Muney 2006; Hummer and Lariscy 2011). We use a large nationally representative prospective data set and multivariate hazards models to test three hypotheses based on human capital theory regarding the factors that potentially mediate the education-mortality relationship. More specifically, we seek to determine: (1) to what extent sets of economic and social resources, health behaviors, and physiological measures independently mediate the relationship between educational attainment and all-cause adult mortality; (2) to what extent economic and social resources, health behaviors, and physiological indicators combine to mediate the educational attainment and mortality relationship; and (3) to what extent these mediating factors are moderated by age.

1.1 Background and significance

Research consistently finds that in the United States higher education is associated with better health outcomes and a lower risk of death (Christenson and Johnson 1995; House et al. 1994; Lynch 2006; Miech et al. 2011; Montez et al. 2012). U.S. adults with less than a high-school education have nearly twice the risk of death in a five-year follow-up period compared to adults with 17 or more years of education (Rogers et al. 2000). This difference in risk translates into a 5–7 year gap in life expectancy at age 25 across educational groups, depending on sex and racial/ethnic group (Lin et al. 2003; Molla et al. 2004). Alternatively, each additional year of education, on average, reduces the odds of adult mortality by about 5% yearly (Elo and Preston 1996; Zajacova and Hummer 2009). When multiple indicators of socioeconomic status (SES)—such as education, income, and occupational status—are considered, education often has the strongest effect on mortality and health outcomes, particularly when both direct and indirect effects are considered (Hummer and Lariscy 2011; Mirowsky and Ross 2003; Williams 1990). The strength and consistency of the inverse relation between education and mortality over time, across places, and across demographic groups suggests that education is a “fundamental cause” of health and longevity (Link and Phelan 1995; Phelan et al. 2004).

The study of mortality is particularly important because it is salient to everyone, does not possess the problems associated with self-reported measures of health, can be assessed reliably over time and across subpopulations, and is not subject to reverse causality (those who die cannot return to better health). In other words, “mortality is by far the most readily and reliably measured index of health” (Vaupel 2010: 536). More specifically, educational differences in mortality are important not only to those who are disadvantaged but also to policymakers who are interested in reducing social inequality and improving population health for all Americans (U.S. DHHS 2010). Thus, understanding how educational attainment works to influence mortality risk through various sets of mediating factors is an essential step toward trying to close the survival gaps that currently characterize the U.S. population.

1.2 Education and adult mortality: theory and hypotheses

Human capital theory was originally developed to explain labor market success and was later used to explain differences in health and longevity. It refers to the investments people make in gaining information, knowledge, and skills. One of the most important investments in human capital is education (Becker 1994, 2005), which improves reading comprehension and contributes to more resourceful, effective, and efficient critical thinking, problem solving, and decision making (Cutler and Lleras-Muney 2006; Mirowsky and Ross 2003). Education facilitates increased earnings and beneficial social connections across the life course. And it helps people lead healthier lifestyles and avoid health problems through its impact on more effective human agency (Mirowsky and Ross 1998, 2003). Increased education helps individuals acquire knowledge; develop higher self-esteem; and exhibit a greater sense of mastery, self-efficacy, personal control, and confidence. More effective agency may be especially important for individuals in understanding how to improve overall health behavior, understand and act upon health promotion and disease prevention messages, and follow effective medical treatment regimens. Importantly, the development of more effective human agency with a higher level of education persists past the completion of formal schooling. The benefits of education accumulate because with time individuals can build upon their educational base to acquire new information and knowledge, learn from past mistakes, and understand and incorporate new scientific findings into their lifestyle (Mirowsky and Ross 2008). Such accumulation protects individuals against social, economic, and personal traumas and shocks, and provides resources to most appropriately deal with new personal situations and setbacks they may encounter. Thus, we expect to find a strong link between higher levels of education and lower risk of mortality for U.S. adults; moreover, because of the broad range of human capital resources that education helps develop, we expect that this relationship will be strong across a range of causes of death.

Structurally, increased education can improve health and increase longevity through the accumulation of economic resources. And higher family income is associated with lower mortality risk among adults of all ages, both sexes, and majority and minority populations (Krueger et al. 2003; Lantz, Goldberstein, House, and Morenoff 2010). Human capital theory strongly suggests that higher income does not simply and automatically lead to improved health and lower mortality; that is, highly educated people are also more likely to use their higher income to invest in their health and longevity (Mirowsky and Ross 2003). For example, higher income can be used to purchase nutritious food, high-quality housing in safe neighborhoods, health insurance (which can encourage prevention as well as treatment of a host of conditions, including hypertension, hypercholesterolemia, and obesity), and memberships in fitness clubs and smoking cessation and weight loss programs.¹ Furthermore, individuals may be able to stretch their money farther through innovative ways to obtain needed resources at lower costs. Highly educated people may also be more likely to save income for future health-related needs through effective planning (Smeeding and Weinberg 2001).

¹Some researchers raise issues of reverse causality: rather than low incomes contributing to poor health, it is possible that individuals in poor health earn low incomes (see Cutler and Lleras-Muney 2010).

Social resources—including marriage, friendship, kinship, and ties to the community—can contribute to health and longevity through emotional, instrumental, and informational advice and support (House et al. 1988); by encouraging healthy and discouraging unhealthy behavior; and by providing health information, thereby reducing the risk of death (Berkman and Glass 2000; Cutler and Lleras-Muney 2010; Pampel, Krueger, and Denney 2010). Working through improved human agency, education contributes to the building of stable forms of social support, including higher rates of marriage and lower rates of divorce (Mirowsky and Ross 2003). Even beyond marriage, highly educated individuals also have access to other highly educated coworkers, friends, and neighbors who they can call upon for health-related advice and support: “Given that high-SES persons adopt healthy behaviors and associate with other high-SES persons, their networks of social support, influence, and engagement promote health and widen disparities” (Pampel, Krueger, and Denney 2010: 360-61). More highly educated individuals also are more likely to seek out health-enhancing networks and activity in their communities; and individuals who belong to clubs and other social organizations receive additional sources of social support through such belonging (Berkman and Glass 2000; Burr, Caro, and Moorhead 2002; Hummer et al. 2004).

A rigid test of the human capital theory as applied to the education-health relationship, though, goes beyond income and social resources (Mirowsky and Ross 1998). More highly educated individuals are also better able to use their more effective human agency and merge various habits together into a coherent healthy lifestyle. Compared to other sociodemographic characteristics, “only education correlates positively and consistently with positive health behaviors” (Mirowsky and Ross 1998: 419). In an earlier study on the education-mortality association, for example, Lantz et al. (1998) found that the odds ratio of mortality for the lowest to the highest education group fell by 14% with controls for health behavior.

More specifically, highly educated people are more likely than the less educated to frequently exercise, and to refrain from smoking and heavy drinking (Cutler and Lleras-Muney 2006; Lantz et al. 2010; Pampel, Krueger, and Denney 2010). Increased physical activity reduces obesity, improves health, and increases life expectancy (Fried et al. 1998; Paffenbarger et al. 1986). Alcohol consumption has a J-shaped relationship with adult mortality risk: light to moderate drinkers experience lower mortality than either abstainers or heavy drinkers (Klatsky and Udaltsova 2007). Heavy drinking can increase the risk of death from some cancers, cirrhosis of the liver, and external causes (Corrao et al. 2004). While there is a positive relationship between education and frequency of drinking, there is a negative relationship between education and the quantity of alcohol consumed per drinking episode (Mirowsky and Ross 2003; Ross and Wu 1995). Even more important, less educated people are more likely to begin smoking, less likely to seek out and follow antismoking advice, and less likely to quit (Rogers et al. 2005). Denney et al. (2010) showed that smoking may account for up to 44% of the educational gap in mortality among working-aged U.S. men.

Physiological indicators of health provide an assessment of long-term socioeconomic vulnerability and the accumulation of health behaviors over the life course untapped by standard survey reports of income, smoking, alcohol use, and exercise. Ross and Wu (1995:

738) conclude that part of their unexplained effect of education on health could be due to unmeasured “physiological consequences of education.” For example, hypertension is influenced by the long-term effects of smoking, poverty, and social strife; inflammation embodies the long-term effects of chronic work and life stressors, and limited preventative medical care; and obesity results from the long-term effects of inactivity and poor diet.

Compared to their less educated counterparts, highly educated persons are more likely to maintain healthy weight through improved nutrition and increased exercise (Denney et al. 2004). Evidence on the effects of body mass on mortality is mixed: some studies suggest that overweight and obesity are second only to smoking as a major cause of preventable mortality, but some studies claim much more modest influence, especially among older adults (see Al Snih et al. 2007; Lantz et al. 2010). While improved medical intervention and drugs may have lessened the effects of obesity on mortality (Gregg et al. 2005), obesity clearly increases disability and functional limitations (Al Snih et al. 2007), which have direct effects on mortality.

Less educated individuals are also more likely to suffer from accumulated assaults on the body through long-term bouts with hypertension, hypercholesterolemia, and a weakened immune system (Kooiker and Christiansen 1995; Muennig et al. 2007; Pampel and Rogers 2004; Ross and Wu 1995). Cardiovascular disease mortality, the leading cause of death in the United States, is associated with high levels of cholesterol, glycosylated hemoglobin, and blood pressure. Hypertension, or high blood pressure, is associated with disability, a number of chronic conditions, including circulatory diseases, and overall mortality (Fields et al. 2004). And there is a strong inverse association between education and blood pressure (Colhoun, Hemingway, and Poulter 1998). Total cholesterol has a U-shaped relationship with mortality; while high cholesterol is a more common problem, low cholesterol levels may indicate a preexisting condition and therefore also increase the risk of death (Crimmins and Vasunilashorn 2011). Recently, better diets and greater use of cholesterol-lowering drugs have improved cholesterol levels for the adult population (Gregg et al. 2005). Glycosylated hemoglobin is associated with glucose control, cardiovascular diseases, and mortality (Crimmins and Vasunilashorn 2011; Selvin et al. 2004).

Long-term inflammation—measured through C-reactive protein (CRP) and albumin—contributes to poor health, functional limitations, coronary heart disease, and shorter lives (Crimmins et al. 2003; Kiecolt-Glaser et al. 2002). Smoking, physical inactivity, obesity, diabetes, high blood pressure, heart attack, stroke, and mortality are associated with high levels of CRP (Crimmins and Vasunilashorn 2011; Ridker 2003), which reduce the body's ability to fight viruses. Further, low albumin concentration is associated with functional decline and mortality (Goldwasser and Feldman 1997; Visser et al. 2005).

We include physiological indicators—weight-for-height, two measures of inflammation, and three cardiovascular risk factors—because of their associations with mortality and because individuals with high levels of education may be able to use their more effective human agency over the life course to reduce their chances of acquiring risky levels of these indicators. Thus, similar to our more direct indicators of SES, social resources, and health

behavior, we expect that levels of these physiological indicators will be more favorable for highly educated individuals.

Based on previous literature (Denney et al. 2010; Lantz et al. 1998; Mirowsky and Ross 2003, 2008), we expect to find that sizable portions of the education-mortality association will be mediated by the separate contributions of income, social resources and activity, health behaviors, and physiological indicators (hypothesis 1 [H1]). The analysis that tests this hypothesis allows us to determine which set of factors has the strongest independent mediating effect on the education-mortality relationship. Furthermore, we hypothesize that more highly educated individuals will be better able to rally their formidable resources and strategically position themselves to most effectively live a long life and reduce their yearly mortality risk (H2); thus, the ‘coalescent’ hypothesis suggests that, in addition to single sets of factors, the *combined* impact of economic and social resources, health behaviors, and physiological indicators will work to explain educational differences in mortality to a greater degree than any single set of mediating factors alone.

To determine if age moderates the education-mortality relationship, as previous studies have suggested (Cutler and Lleras-Muney 2010; Hummer and Lariscy 2011; Zajacova and Hummer 2009), we disaggregate our analyses by broad age group. For example, cigarette smoking has become increasingly concentrated among the less educated in recent birth cohorts (Meara et al. 2008) and, thus, we hypothesize that health behaviors will be stronger mediators of the education-mortality relationship for younger than for older adults (H3). More generally, it will be important to reveal whether the mediating effects of income, social resources, health behaviors, and physiological indicators are more pronounced in one age group than another.

To provide more specific tests of the three hypotheses we derived from human capital theory, we examine both all-cause and cause-specific mortality risk for U.S. adults aged 25 and over. Although we do not derive specific hypotheses regarding how our proposed mediators will work to differentially explain the education-mortality relationship by cause of death, there are reasons to expect that their mediating influence will vary by cause. For instance, educational differences in causes of death that are more strongly related to health behavior and medical treatment, such as cardiovascular diseases (Cutler and Lleras-Muney 2006), may be mediated by health behaviors and physiological measures to a greater degree than causes that have a weaker association with health behavior and medical treatment, such as external causes. And external causes of death may reveal a large education gap because they epitomize problems with low levels of education: individuals with low education may miscalculate risks based on faulty reasoning and thus exhibit higher mortality risk than more highly educated individuals.

2. Data and methods

2.1 Data

We use the National Health and Nutritional Examination Survey (NHANES III) Linked Mortality File (LMF) to test our hypotheses. The NHANES-LMF is based on a multistage design that drew a nationally representative sample from the non-institutionalized U.S.

population (National Center for Health Statistics [NCHS] 1994, 2009, 2010). Over six years (1988–1994), NCHS collected and assembled detailed cross-sectional information from interviews, physical examinations, and laboratory tests for this sample. NHANES III-LMF is the best choice for our analyses because the data are nationally representative; include a long follow-up for survival status for each individual (through December 31, 2006); include a detailed array of socioeconomic, social, behavioral, and physiological measures; and include a large number of deaths that allows for age- and cause-specific mortality modeling.

Although the NHANES III is linked to the mortality files beginning at age 17, we limit our sample to individuals aged 25 and above at the time of the interview to increase the probability that they had completed or nearly completed their education. We also limit our sample to those who identify as non-Hispanic white, non-Hispanic black, or Mexican American, and were eligible to be matched to mortality records ($n=15,988$; 5,137 deaths). Because of missing information on variables included in the analysis, we drop an additional 297 individuals (1.86%), 152 of whom died during follow-up, resulting in a final sample size of 15,691 individuals and 4,985 deaths. Our sample includes 1,875 persons who did not give blood and/or urine and therefore lack information on albumin, CRP, total cholesterol, and glycosylated hemoglobin. Supplementary analyses not shown revealed that including these respondents do not bias our results; thus we include “missing” indicators for these variables to avoid dropping the cases.

2.2 Variables and Measurement

Educational attainment is the key variable of interest. While a continuous measure would best assess the day-to-day and week-to-week accumulation of human capital through education, the limited number of deaths among people with very low or very high levels of education makes categorization of educational attainment a preferred strategy for this analysis. Further, NHANES III collected educational data in years of schooling rather than as a combination of years and degrees, making it impossible for us to fully understand how individuals' time spent in school converts to the development of human capital.² To best overcome these data limitations, we coded educational attainment into 11 or fewer years, 12 years, 13 to 15 years, and 16 or more years of education (referent). Each of these categories contains a reasonably large number of individuals and follow-up deaths and, together, they approximate cut-points that have been and continue to be important in the U.S. educational system. These categories are also similar to those most recently shown to best capture the functional form of the education-mortality relationship in the United States (Montez et al. 2012), although we also include a category of 16 and higher to approximate the college-educated group of American adults.

Because age has such a profound impact on the risk of death, rather than merely control for it, we use age and time since interview (in months) as the time scale in our mortality risk hazard models (see Kom, Graubard, and Midthune 1997). We further disaggregate some of our analyses into the age groups of 25–59 (with 882 deaths) and 60 and over (with 4,103 deaths) because the education-mortality relationship varies by age (Rogers et al. 2010a;

²Nevertheless, studies of the association between credentials and mortality (see, for example, Rogers et al. 2010b) remain a promising area for future research.

Zajacova and Hummer 2009). Sex is coded as 0 (female) or 1 (male). To ensure large numbers of minority groups in its sample, NHANES oversampled non-Hispanic blacks and Mexican Americans. We code race/ethnicity as non-Hispanic white (referent), non-Hispanic black, and Mexican American. We delete members of other ethnic groups because of small sample sizes. Because the United States includes a sizable proportion of immigrants, who differ from the native born in both average educational level and mortality risk, we control for nativity.

We measure family income with the income-to-poverty-level ratio, which assesses the ratio of family income to the poverty threshold defined by the Census Bureau. The ratio varies from 0 to 12, with higher values representing income levels that are farther from the poverty line. The NCHS (2001) provided these estimates and, when needed, replaced missing data with imputed values through multiple imputation methods.³

We include four measures of social resources. We code marital status as currently married (referent), never married, widowed, and divorced or separated. We code visits with friends and relatives as one or more times per week (referent) or none. We assess religious attendance as greater than once a week (referent), once a week, at least once over the last year but less than once a week, and no attendance over the last year. Finally, club involvement is measured as whether individuals belong to clubs and organizations (referent) or not.

Health behaviors include exercise, alcohol consumption, and smoking. We code individuals who have smoked fewer than 100 cigarettes in their life as never smokers, those who have smoked in the past but do not currently smoke as former smokers, and those who currently smoke as current smokers (see Krueger et al. 2004). We code nondrinkers as those who have consumed fewer than 12 drinks in their lifetime, former drinkers as those who have consumed fewer than 12 drinks in the last year but at least 12 drinks in their lifetime, light drinkers as those who drink 1-4 drinks per day on the days that they drink and who have had at least 12 drinks in the last year, and moderate to heavy drinkers as those who drink 5 or more drinks per day on the days that they drink and who have had at least 12 drinks in the last year. We code exercise as more than 7 hours per week (referent), 7 or fewer hours, and none. Although our three health behaviors are not exhaustive, they are central health behaviors identified in the adult mortality literature (e.g., Rogers et al. 2000), and should capture much of the effects of unhealthy and risky behaviors that the human capital perspective suggests are more common among individuals with less rather than more education.

Physiological indicators include hypertension, cholesterol, glycosylated hemoglobin, inflammation (measured by CRP and albumin), and obesity (assessed through the body mass index [BMI]). BMI is calculated as weight in kilograms divided by height in meters squared, and is coded as underweight (less than 18.5), normal or overweight (18.5 to less than 30), obese class 1 (30 to less than 35), and obese class 2 or greater (35 or more). We define the

³Because NHANES does not include birth weight or family background characteristics, we cannot directly test whether inherited health and social inequalities contributes to educational differences in adult mortality.

referent as normal or overweight because these categories include what is considered ideal (normal weight) and because overweight individuals have the same mortality risk as normal weight individuals (Al Snih et al. 2007). Following the American Heart Association and Centers for Disease Control and Prevention recommendations (Pearson et al. 2003), we code CRP as less than 0.3 mg/dL (the referent) compared to greater than or equal to 0.3 mg/dL, and albumin as greater than 4.5 g/dL (the referent), greater than 4.0 to less than or equal to 4.5 g/dL, greater than 3.5 to less than or equal to 4.0 g/dL, and less than or equal to 3.5 g/dL. We created a separate category, “missing because of infection,” to deal with individuals who were fighting an existing infection and who would therefore have artificially high CRP levels and low albumin.⁴

Following *JNC-7* (DHHS 2003), we average up to six blood pressure measurements taken during the examination and code hypertension as normal blood pressure (less than 80 diastolic blood pressure [DBP] and less than 120 systolic blood pressure [SBP]; the referent), pre-hypertensive (80-89 DBP or 120-139 SBP), hypertensive stage 1 (90-99 DBP or 140-159 SBP), and hypertensive stage 2 (currently taking antihypertensive medication, DBP of 100 or more, or SBP of 160 or more). Although medication reduces hypertension, compared to normotensive individuals, individuals treated for hypertension are still at an increased risk of death (Crimmins and Vasunilashorn 2011).

Because there is a U-shaped relationship between total cholesterol and mortality, we code total cholesterol into the following categories: less than 150, 150 to 249 (the referent), 250 to 349, and equal to or greater than 350.⁵ We code glycosylated hemoglobin as less than 6.4% (the referent) and 6.4% or more (see Osei et al. 2003).⁶ Even though the percentage of individuals with missing information is quite small, we include a category for missing values on the physiological measures to avoid dropping records, especially records with nonrandom missing values, such as those individuals who were too sick to complete the examination (NCHS 1994).

Our dependent variable is the hazard of death, with die/survive specified as the event of interest and age plus months since interview measuring duration. We right censor individuals who survive the entire follow-up period. We further specify several underlying causes of death in a portion of our analysis. The NHANES III LMF includes cause-of-death codes from the most recent classification scheme, the tenth revision of the *International Classification of Diseases* (ICD-10; World Health Organization 2007). We specify diseases of the heart (ICD10 I00-I09, I11, I13, and I20-I51), malignant neoplasms (ICD10 C00-C97), diseases of the respiratory system (ICD10 J00-J99), external causes (ICD10 U01-U03, V01-

⁴NHANES III collects extensive information on health behaviors, social resources, and physiological indicators—which are the focus of our paper—but not on psychosocial mediators.

⁵We do not include such additional cardiovascular risk measures as low density lipoproteins (LDLs) or triglycerides because these lipids are ascertainable only for fasting populations, and this requirement would severely reduce our sample sizes (Crimmins and Vasunilashorn 2011). Further, preliminary analyses suggested that these additional variables added very little insight into the association between education and mortality.

⁶In addition to the biomarkers discussed above, the data set includes whether a doctor had previously told the respondent that he or she had any of a variety of health conditions. We have not included these additional conditions because they may lead to model over-specification (for example, hypertension and hypercholesterolemia are markers for heart disease, and BMI will be closely related to diabetes); compared to clinical measures they are more likely to suffer from self-report biases; and several conditions (including diabetes and cancer) require additional coding that could distract readers from the paper's central focus. Furthermore, inclusion of these variables in our final model of all-cause mortality did not substantively alter the results.

Y09, Y85-Y86, Y87.0, and Y87.1), and a residual category (all other causes of death). Over the follow-up period, there were 2,287 deaths for cardiovascular diseases, 1,057 for cancer, 480 for respiratory diseases, 154 for external causes, and 1,007 for all other causes of death. Modest numbers of deaths prevent us from examining more detailed causes. In our cause-specific analyses, we right-censor individuals when they die from other causes or survive to the end of the follow-up period.

2.3 Methods

To examine the risk of death by educational attainment, we use Cox proportional hazards models (Allison 1984).⁷ We employ a progressive model building strategy (i.e., Mirowsky 1999) that starts with the baseline education-mortality model (net of demographic characteristics) and then successively add sets of covariates that are consistent with the development of our hypotheses outlined above. More specifically, we start with a baseline mode that includes education and basic demographic controls. To show the independent effect of each set of mediating factors on the education gap in mortality, we then separately add family income, social resources/activity, health behaviors, and then all three of these indicators together (see Baron and Kenny 1986). We next add physiological indicators to the baseline model and finally, to show the simultaneous effect of the combined factors, the full model includes all sets of mediating factors.

We begin our analysis with a focus on all-cause mortality for all adults aged 25 and above before turning to the more specific examination of age- and cause-specific education-mortality differences. We adjust all descriptive statistics and mortality models for the NHANES III sample weights and complex sampling frame using the “svy” commands in Stata 10.0 (StataCorp 2007).

3. Results

Table 1 reveals remarkable differences in the distributions of the covariates by educational attainment. Individuals with 12 years of education form the largest proportion of the adult population (34.1%), and those with 11 or fewer years form the second largest (24.5%). Over 40% of the sample has attained over 12 years of schooling, and 22.0% of the sample has obtained 16 or more. The most educated individuals exhibit virtually every attribute that is associated with better health and lower mortality risk. We conduct adjusted Wald tests to determine if means for respondents with 16 or more years of education are statistically different from the means for respondents with fewer years of education. Compared to individuals with less than 12 years of schooling, those with 16 or more years are statistically more likely to have a high income, be married, visit with friends and relatives, be involved in clubs/activities, abstain from smoking, drink moderately, exercise regularly, be normotensive, and have low cholesterol (below 250) levels.

⁷In our final model of Table 2, our main independent variables (less than or equal to 11 years of education, 12 years of education, and 13-15 years of education) all pass proportionality assumptions; however, several other variables fail this assumption and therefore the final model does not pass the global test. These violations motivate the disaggregation of our models by age in Table 3. When disaggregated, the results from the age group 25-59 pass the global proportionality tests, and our main variables of interest also pass for the age group 60 years and older.

The most highly educated persons also exhibit the greatest percentage of never smokers (55.6%) and the smallest percentage of current smokers (15.1%). Drinking varies with education in a nonlinear way. The group with 16 or more years of education has the greatest proportion of light drinkers (1-4 drinks per day, 55.7%) and the smallest proportion of heavy drinkers (5 or more drinks, 3.9%). The group with 11 or fewer years of education has the highest percentage of former drinkers (36.0%) and widowed individuals (17.4%), characteristics that may in part reflect their older age distribution. Indeed, these descriptive results are strongly influenced by age structure; to model all-cause mortality risk while taking age and other covariates into account, we turn to Table 2.

Table 2 presents a multivariate model of the association between educational attainment and all-cause mortality risk. Model 1 displays a strong, clear, graded relationship: compared to those with 16 or more years of schooling, those with 13-15 years experience a 38% increased risk (or a hazard ratio of 1.38), those with 12 years, a 59% increased risk, and those with 0-11 years, a 75% increased risk of death over the follow-up period, controlling for sex, race/ethnicity, and nativity.⁸

Because more educated people tend to be more affluent (see Table 1), controlling for income (Model 2) attenuates the association between education and mortality found in Model 1 for each education group, and reduces the mortality gap between the groups with the highest and lowest levels of education by 40% ($[\ln(1.75) - \ln(1.40)] / \ln(1.75) \times 100$). Controlling for social resources (Model 3) dampens the association between education and mortality found in Model 1, because more educated individuals are more likely to be married, visit with others, attend religious services, and be involved in clubs. This set of mediating factors reduces the gap in mortality between the groups with the highest and lowest levels of education by 22%.

Like previous researchers (e.g., Denney et al. 2010; Lantz et al. 2010), we find that the education gap in mortality is due in part to health behaviors. The mortality gap between those with 16 or more years of education and those with less than 12 years of education closes by 36% with controls for smoking, drinking, and exercise (compare Models 1 and 4). Model 5 controls for economic resources, social resources, and health behaviors and closes the gap between those with less than 12 years of education and those with 16 years or more by 72%. The gap closes by 13% with controls for physiological factors (compare Models 1 and 6). Simultaneously controlling for all covariates drastically reduces differences in mortality by educational attainment, and eliminates any statistical difference for those with less or more than 12 years of education (though not for those with exactly 12 years), compared to those with 16 or more (see Model 7). Indeed, simultaneously controlling for all variables reduces the education-mortality gap by 78%; however, this is not much stronger than the model (see Model 5) that controlled for economic resources, social resources, and health behaviors.⁹

⁸To test for health selection, we reran our analyses but excluded individuals who were in poor health at the time of the interview, based on their self-reported health status. This exclusion had no material effect on the results in Model 1, which suggests that health selection has a negligible impact on the education-mortality association.

Table 3 shows that the education-mortality relationship is much stronger for younger than for older adults, and that the effects of specific clusters of mediators vary by age. Among those aged 25-59, compared to those who have 16 or more years of education, those who have 11 or fewer years are almost three times as likely to die over the follow-up period (Model 1). This relationship is attenuated substantially with controls for economic resources (Model 2), social resources (Model 3), health behaviors (Model 4), all three of these factors combined (Model 5), and physiological risk factors (Model 6). Controlling for health behaviors has a greater impact on closing the education and mortality gap (which closes by 40%) than controlling for income (which closes the gap by 28%) or social resources (which closes the gap by 14%).

At older ages, the relative educational gap in mortality is smaller (cf. Hummer and Lariscy 2011). Among persons ages 60 and older, compared to those who have 16 or more years of education, those with 11 or fewer years are 44% more likely to die during the follow-up period. In this age group, controlling for economic and social resources, health behaviors, and physiological indicators eliminates any statistically significant educational difference in mortality (Model 7). Economic factors have the strongest effect and reduce the education gap in mortality by 55% (compare Models 1 and 2).¹⁰

Table 4 presents educational differences in cause-specific mortality. Compared to adults with 16 or more years of education, those with 11 or fewer years of education are 70% more likely to die from cardiovascular diseases during follow-up and more than twice as likely to die from respiratory diseases (Model 1). But the educational difference in respiratory disease deaths is attenuated as controls are added for income, social resources, and health behaviors (in Models 2, 3, 4, and 5). The patterns for cardiovascular diseases, the major cause of death in the United States, parallel those presented for all-cause mortality (Table 2). The education gap in cardiovascular disease mortality is reduced substantially by economic resources and health behaviors (by 35% each), and by social resources (by 29%). In combination, economic and social resources, health behaviors, and physiological indicators completely close the education gap in cardiovascular disease mortality (Model 5). The smallest educational gap for a specific cause of death is for cancer, which closes by 52% with the inclusion of health behaviors and by 41% with the inclusion of economic resources; it no longer displays significant differences once we control for physiological factors. The education gap in respiratory disease mortality closes by about one-third with controls for either economic resources or health behaviors. The largest educational gap for a specific cause of death is for external causes. Compared to those with 16 or more years of education, those with less than 12 years of education are 4.5 times as likely to die from external causes

⁹To ensure that select subpopulations with low levels of education are not biasing our results, we conducted supplementary analyses that excluded Mexican Americans and foreign-born individuals from our sample. The two sets of results were almost identical and therefore show no evidence of a material bias.

¹⁰We also ran sex-specific analyses. Because educational differentials in mortality by sex were quite similar, we do not present a separate table. Nevertheless, overall educational differences in mortality are modestly wider among men (for similar results, see Ross et al. 2012; Zajacova and Hummer 2009). Controlling for social resources substantially closed the education gap in mortality for both sexes and had a slightly larger influence for females. Controlling for economic resources and health lifestyles also attenuates the education-mortality relationship, especially among males. Compared to females, males are more likely to smoke, drink, exercise, and earn higher incomes. When these variables, especially health behaviors, are entered into the models, they have a greater dampening effect on the education gap in mortality for males than for females. The final models were able to eliminate any statistical educational differences in mortality for females but not for males.

(Model 1). This differential closes by 14% with controls for social resources and 9% for healthy behaviors.¹¹ There are no statistically significant educational differences in cardiovascular disease mortality, cancer, or other causes once we control for all mediating variables (Model 7).

4. Conclusion

This paper contributes to the literature by employing the human capital perspective to demonstrate how education is associated with overall, age-, and cause-specific mortality. A large part of the educational gap in mortality can be attributed to differing education-specific *distributions* of economic and social resources, health behaviors, and physiological indicators. Compared to individuals with less education, those with more can expect longer lives because they are more likely to be affluent; be married; engage in social relationships; be involved with clubs and organizations; abstain from smoking; avoid excessive drinking; exercise; and have better health conditions, including lower rates of obesity, inflammation, hypertension, and hypercholesterolemia. The strong mediating effects of each set of these variables support the first hypothesis of this paper.

A major economic resource, income, has a strong impact on the education-mortality relationship. Thus, social policies to reduce poverty and to increase education may both help close the educational gap in mortality, though raising people's educational level may impart lifelong benefits, whereas lifting individuals out of poverty may have more transitory effects. However, personal and structural obstacles may prevent some individuals from obtaining additional education. Some studies find that the effect of education on mortality is no longer significant once income is added to the model; Lantz et al. (2010: 156) claim that "education indirectly influences mortality through its strong association with income." In our models, income mediates nearly 40% of the education-mortality association but does not come close to fully accounting for it. In other words, the education and mortality relationship is also mediated by social resources, health behaviors, and physiological indicators, providing support for the coalescent hypothesis (H2).

Social resources provide a valuable source of support that reduces the risk of death. Spouses, friends, relatives, and community members can provide emotional, instrumental, and financial support to reduce overall, age-specific, and cause-specific mortality (see also Berkman and Glass 2000; House et al. 1988). More highly educated individuals are able to successfully use their human agency to create and maintain stable and supportive social relationships that can enhance their health and reduce their risk of death.

Health behaviors exhibited similar effects as economic resources among adults of all ages, accounting for 36% of the educational difference in mortality (compare Models 1 and 4 in Table 2), which is similar to what other researchers have found (e.g., Denney et al. 2010). We also found that the effects of healthy behaviors exert a more powerful impact on the education gap in mortality at younger than older ages (offering support for our third

¹¹The educational differential in external cause mortality widens with controls for income (compare Models 1 and 2), which might be due to small sample sizes or to greater risks of external causes among individuals with higher incomes.

hypothesis), perhaps because risky behavior has become more concentrated among more recent cohorts of less educated individuals (Meara et al. 2008). Reducing unhealthy lifestyles among the less educated, particularly cigarette smoking, could substantially reduce educational differences in mortality, especially among younger adults.

An intriguing result from our descriptive statistics is that compared to less educated individuals, highly educated persons are more likely to be light drinkers and less likely to be heavy drinkers (for similar results, see Liao et al. 2000; Mirowsky and Ross 2003), which is compatible with the notion that high-SES individuals live healthier lives.¹² Thus, ways to increase the length of life of individuals with less education include providing opportunities to obtain additional years of education, as well as becoming better informed about and engaging in healthy behaviors. Because heavy and binge drinking is detrimental to health and longevity, and is more common among individuals with lower levels of education, social policies that reduce heavy drinking could have a greater impact on subpopulations with higher prevalence rates of heavy drinking, including individuals with less education. And for many individuals, it might be easier to convert their drinking behaviors from heavy to light alcohol consumption than to eliminate drinking altogether. Additionally, as the benefits of exercise can be both immediate and long-lasting, it may be easier to entice individuals to exercise than to, say, quit smoking. Thus, promoting exercise might be a direct way to reduce stress, increase life expectancy, and reduce educational disparities in mortality in the United States.

This paper also included physiological indicators, which tap additional dimensions of health lifestyles, including long-term cumulative effects of risky behaviors expressed at the organ system level. While physiological risk factors are potentially amenable to intervention, they only modestly affect the relationship between education and mortality once we accounted for the economic, social, and behavioral mediating variables. Dowd and Goldman (2006) suggest that biomarker health status measures do not capture the primary pathways through which education affects mortality; our results are consistent with that notion. Still, they help tap into proximate risk factors, reflect real biological effects, and may capture additional effects of such long-term unhealthy lifestyles as smoking, excessive drinking, overeating, and inactivity that are not adequately measured by survey reports. Furthermore, many physiological risk factors can be minimized or eradicated through diet, exercise, medical intervention (including health screening and surgery), and prescription drugs (Colhoun et al. 1998; Gregg et al. 2005). Future work on education and mortality should include physiological measures to capture the expression of the long-term accumulation of risky behaviors, environmental insults, social disruption, and economic tumult.

Like previous researchers, we find greater relative educational differences in mortality for younger than older ages (see also Cutler and Lleras-Muney 2010; Hummer and Lariscy

¹²Like most studies, we rely on self-reported alcohol consumption, which could be underreported if respondents provide socially desirable responses. If more highly educated individuals are more likely to underreport their drinking, then the mediating effects of alcohol consumption on the association between education and mortality will be artificially reduced. But such a potential bias is unlikely to have a large effect because drinking status is but one of several health behavior measures, there is a strong reverse gradient between the level of education and heavy alcohol consumption (see Table 1), and most studies find that self-reports of drinking are generally reliable and valid (see the review article by Del Boca and Darkes 2003).

2011).¹³ Furthermore, age moderates the mediators. Educational differences at younger ages close substantially once we control for health behaviors, and close partly once we control for economic resources, but are not as substantially affected by social resources or physiological indicators. Still, even in the full model, we find substantial differences in mortality by educational attainment among the youngest age group. In contrast, educational differences in mortality at older ages display smaller relative gaps. We were able to statistically eliminate the complete education gap in mortality among older adults. In part, mortality selection of the least educated reduces relative gaps in the remaining population of older adults (Crimmins 2005). Among the older ages, the education gap in mortality is most responsive to economic resources, but is also responsive to social resources and health behaviors.

Similarly, our cause-specific analyses demonstrate that more highly educated individuals reduce their risk of death through a multi- rather than a single-pronged approach. More highly educated individuals use a combination of economic and social resources, and healthy behaviors to reduce the risk of cardiovascular and respiratory diseases; a greater reliance on healthy behaviors to reduce the risk of cancer mortality; and a heavier reliance on social resources to reduce the risk of death from external causes. Because cardiovascular disease is the leading cause of death among U.S. adults, reducing it would most likely have an exceptionally large impact on the overall educational gap in mortality. Respiratory disease mortality displays a sizable educational gap that is strongly influenced by unhealthy behavior, especially smoking. Both cardiovascular and respiratory diseases are more strongly related to prevention and treatment and have a greater educational gap than does cancer, which has less association with prevention (see Cutler and Lleras-Muney 2006). Still, cancer has some association with unhealthy behaviors, particularly cigarette consumption, excessive drinking, inactivity, and obesity. Increased education can increase knowledge; develop problem-solving, critical-thinking, and decision-making abilities; and can foster stable and supportive social relationships, which can ameliorate or eliminate life-endangering events (Cutler and Lleras-Muney 2006; Mirowsky and Ross 2003). Additional efforts to reduce the risk of death from external causes would benefit everyone, but especially those with less education.

Social policies should fully exploit the power of education. Policymakers should ensure that their public health messages resonate with individuals of all education levels. The first adopters of healthier practices are generally individuals with higher levels of education. But public health officials should actively and creatively explore ways to appeal to individuals with the greatest needs and with less education by, say, ensuring that public health messages are salient, and that steps to better health are clear and achievable. Education policies might more directly tout the health-enhancing benefits of education. For example, educators might devote more time to teaching students how to critically read, critique, and better understand the health and medical literatures. And social policies could do more to remove or reduce structural barriers to educational opportunities. Because differential educational attainment can contribute to inequality across generations (Palloni et al. 2009), removing barriers to and increasing opportunities for additional education, particularly among those from

¹³The age differences may partly reflect cohort effects, since recent birth cohorts have witnessed a widening in the education-mortality gap (Masters et al. 2012).

disadvantaged family backgrounds, may also reduce intergenerational inequality. Providing affordable education, increased access to educational opportunities (including additional courses on the weekends and at night), targeting underserved populations (including individuals with disabilities), innovative use of new technologies (including distance and on-line learning), and additional specialized learning environments (including workshops, short courses, and summer courses) that are available to a wide array of individuals can provide multiple social, economic, and health benefits, including the strong possibility of increased life expectancies.

While we used the NHANES because it includes a rich and diverse set of covariates, we acknowledge that the contribution of each group of mediators reflects the true strength of the association in addition to the quantity and quality of the selected mediators. The strong mediating effect of health behaviors could have been strengthened further with the inclusion of additional variables (such as diet and sleep patterns). Consistent with the human capital perspective, we could have included risky behaviors (such as seat belt use and risky driving) and psychosocial resources (to capture additional dimensions of human agency). And some variables may change over time. Thus, future data collection efforts are warranted that include additional covariates, including time-varying covariates, and further research is called for that can test and identify important additional mediators in the relationship between education and mortality.

Our results show that education continues to be strongly associated with mortality risk in the United States. Our results also reinforce the notion that education is a fundamental cause of mortality because it operates through a variety of social and economic resources, healthy lifestyles, and health conditions (Link and Phelan 1995). In support of the coalescent hypothesis, education enables individuals to assemble their vast and complex array of social and economic resources and health behaviors into a coherent lifestyle that preserves life. Rather than adhere to simple, singular, formulaic solutions, individuals may need to implement complex, ever-changing, fluid solutions to optimize their longevity, tasks that favor those with higher levels of education. Education enables individuals to effectively coalesce and leverage their diverse and extensive resources to increase their longevity.

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Highlights

- We analyze how the education-mortality association is mediated by specific sets of factors.
- We use the NHANES-LMF and multivariate hazards models to test three hypotheses.
- We find that family income and health behaviors are the strongest mediators, but that their effects differ by age.
- Higher levels of education enable individuals to leverage resources to increase their longevity.

Table 1

Descriptive Statistics of Covariates by Education, U.S. Adults Aged 25 and Over, 1988-1994

	All	Education Category			
		16 yrs	13-15 yrs ^a	12 yrs ^a	0-11 yrs ^a
Education					
11 years	24.47				
12 years	34.14				
13-15 years	19.37				
16 years	22.02				
Demographic Factors					
Male	47.36	54.47	46.57 ^{**}	42.94 ^{***}	47.80 ^{**}
Female	52.64	45.53	53.43 ^{**}	57.06 ^{***}	52.20 ^{**}
Age					
25 to 39	39.04	42.46	47.64 [*]	41.10	26.27 ^{***}
40 to 54	28.04	35.24	29.68 ^{**}	27.57 ^{***}	20.92 ^{***}
55 to 69	19.76	14.73	14.17	21.71 ^{***}	26.02 ^{***}
70 to 85	11.57	6.93	7.41	8.74	22.96 ^{***}
85+	1.59	0.64	1.10	0.88 [†]	3.83 ^{***}
Race/ethnicity					
Non-Hispanic white	83.99	92.77	86.29 ^{***}	84.95 ^{***}	72.93 ^{***}
Non-Hispanic black	11.45	5.92	10.84 ^{***}	12.14 ^{***}	15.96 ^{***}
Mexican American	4.56	1.31	2.87 ^{***}	2.91 ^{***}	11.11 ^{***}
Foreign-born	6.38	6.59	5.14 [†]	4.13 [*]	10.29 [*]
US-born	93.62	93.41	94.86 [†]	95.87 [*]	89.71 [*]
Economic Resource					
Income-to-poverty ratio (mean)	3.70	5.07	4.07 ^{***}	3.47 ^{***}	2.48 ^{***}
Social Resources					
Marital status					
Married	69.12	73.06	68.66	71.39	62.76 ^{***}
Divorced/separated	11.51	8.64	14.06 ^{**}	11.76 [*]	11.74 [*]
Never married	10.74	15.10	11.80 [†]	9.22 ^{***}	8.08 ^{***}
Widowed	8.63	3.20	5.48 ^{**}	7.63 ^{***}	17.42 ^{***}
Visit with friends, relatives < 1 visit per week	4.41	2.22	2.45	4.07 ^{**}	8.46 ^{***}
Visit with friends, relatives > 1 visit per week	95.59	97.78	97.55	95.93 ^{**}	91.54 ^{***}
Religious attendance					
No attendance	38.42	30.81	34.69	40.46 ^{***}	45.37 ^{***}

	All	Education Category			
		16 yrs	13-15 yrs ^a	12 yrs ^a	0-11 yrs ^a
< once a week	25.60	30.92	27.98	25.23 ^{**}	20.89 ^{***}
Once a week	26.74	28.81	27.75	25.65	25.61
> once a week	9.24	9.46	9.58	8.66	8.13
Club involvement	40.51	60.13	48.25 ^{***}	35.67 ^{***}	23.46 ^{***}
No club involvement	59.49	39.87	51.75 ^{***}	64.33 ^{***}	76.54
Health Lifestyle					
Smoking status					
Former	27.82	29.31	38.88	25.74 [†]	28.53
Current	28.20	15.14	24.50 ^{***}	33.93 ^{***}	34.88 ^{***}
Never	43.98	55.55	36.62 ^{***}	40.33 ^{***}	36.59 ^{***}
Drinking status					
Never	9.89	6.91	5.94	9.89 [*]	15.69 ^{***}
Former	29.83	20.80	27.48 ^{**}	32.58 ^{***}	36.00 ^{***}
1-4 drinks	38.62	55.68	46.55	36.11 ^{***}	20.47 ^{***}
5 or more drinks	8.06	3.94	7.84 ^{**}	8.42 ^{***}	11.43 ^{**}
Missing	13.60	12.67	12.19 ^{***}	13.00	16.41 ^{***}
Hours of exercise per week					
None	32.98	18.45	24.19 ^{**}	34.28 ^{***}	50.71 ^{***}
7 hours	33.14	33.27	33.44	34.47	30.94
> 7 hours	33.88	48.28	42.37 ^{**}	31.25 ^{***}	18.35 ^{***}
Physiological Indicators					
Obesity					
BMI					
< 18.5	2.15	2.78	1.95	1.47+	2.71
18.5 and <30	74.60	81.51	76.08 ^{**}	73.10 ^{***}	69.28 ^{***}
30 and < 35	14.86	11.50	14.01+	15.35 ^{**}	17.89 ^{***}
35	8.39	4.21	7.96 ^{***}	10.08 ^{***}	10.12 ^{***}
Inflammation					
Albumin					
3.5 g/dL	2.20	1.93	1.51	2.41	2.71
> 3.5 - 4.0 g/dL	23.24	19.54	22.09	24.08 ^{**}	26.33 ^{**}
> 4.0 - 4.5 g/dL	43.81	45.68	46.24	44.32	39.49 ^{**}
> 4.5 g/dL	11.47	15.12	8.29 [†]	10.25 ^{***}	8.84 ^{***}
Missing because of infection	4.52	3.14	13.00	4.55 [†]	5.81 ^{***}
Missing	14.76	14.59	8.87	14.39	16.82

		Education Category			
	All	16 yrs	13-15 yrs ^a	12 yrs ^a	0-11 yrs ^a
C-Reactive Protein					
< 0.3mg/dL	46.05	54.59	48.39 ***	44.21 ***	39.07 ***
0.3 mg/dL	16.10	11.43	15.15 **	17.62 ***	18.93 ***
Missing because of infection	23.36	19.63	23.10 *	24.07 **	25.95 ***
Missing	14.49	14.35	13.36	14.10	16.05 *
<i>Cardiovascular Risk Factors</i>					
Hypertension					
Normotensive (< 120 SBP and < 80 DB)	61.43	68.69	67.44	61.72	49.73 *
Prehypertensive (120-139 SBP or 80-89 DBP)	9.88	10.95	8.87	9.98 **	9.59 ***
Hypertensive stage 1 (140-159 SBP or 90-99 [	9.27	6.91	8.22	9.72 ***	11.58 ***
Hypertensive stage 2 (160 SBP or 100 DB	17.89	11.78	14.47 *	16.97	27.37 ***
Missing	1.53	1.67	1.00	1.61	1.73
Total cholesterol					
< 150	5.75	6.62	5.85	5.81	4.78 *
150-249	67.26	68.72	69.15	67.30	64.38 **
250-349	12.70	10.36	12.17	12.91 *	14.95 ***
350	0.33	0.14	0.19	0.32	0.64 **
Missing	13.96	14.16	12.64	13.66	15.25
Glycosylated hemoglobin					
< 6.4 percent	81.23	83.63	83.35 *	82.31 ***	75.85 ***
6.4 percent	5.54	2.50	4.20 *	5.16 **	9.90 ***
Missing	13.23	13.87	12.45	12.53	14.25
N	15,691	2,075	2,349	4,643	6,624
Died over follow-up period	4,985	406	521	1,157	2,901

Source: Derived from NHANES III Linked Adult Interview, Examination, and Laboratory Files.

^aAdjusted Wald tests show whether the education category is statistically different from 16 years of education.

†
p .10

*
p .05

**
p .01

p .001

Table 2

Hazard Ratios for Educational Differences in Mortality, U.S. Adults Aged 25 and Over, 1988-2006

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Education (16 years)							
11 years	1.75 ***	1.40 ***	1.55 ***	1.43 ***	1.17 [†]	1.63 ***	1.13
12 years	1.59 ***	1.39 ***	1.49 ***	1.37 ***	1.22 **	1.54 ***	1.20 *
13-15 years	1.38 **	1.26 *	1.33 **	1.26 *	1.17 [†]	1.33 **	1.15
Demographic Factors							
Sex (female)	1.50 ***	1.55 ***	1.51 ***	1.41 ***	1.43 ***	1.61 ***	1.53 ***
Race/ethnicity (non-Hispanic white)							
Non-Hispanic black	1.33 ***	1.22 ***	1.29 ***	1.24 ***	1.16 **	1.12 *	1.01
Mexican American	0.97	0.90	0.97	0.94	0.91	0.96	0.91
Foreign-born (US-born)	0.77 **	0.76 **	0.77 **	0.81 *	0.80 *	0.77	0.80 **
Economic Resource							
Income-to-poverty ratio		0.90 ***			0.94 ***		0.95 **
Social Resources							
Marital status (married)							
Divorced/separated			1.51 ***		1.35 ***		1.35 ***
Never married			1.24 *		1.17		1.08
Widowed			1.14 *		1.06		1.03
Visit with friends, relatives (1 visit per wk)			1.39 ***		1.30 ***		1.27 ***
Religious attendance (> once a week)							
No attendance			1.54 ***		1.36 **		1.31 **
< once a week			1.18 [†]		1.12		1.07
Once a week			1.10		1.06		1.04
Club involvement (yes)			1.19		1.11 *		1.11 *
Health Lifestyle							
Smoking status (never)							
Former				1.32 ***	1.29 ***		1.29 ***
Current				2.14 ***	2.00 ***		2.01 ***
Drinking status (1-4 drinks)							
Never				1.00	1.03		1.13 [†]
Former				1.08	1.08		1.11
5 or more drinks				1.43 **	1.30 *		1.40 **
Missing				2.37 [†]	1.77		1.77
Hours of exercise per week (> 7 hours)							

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
None				1.59***	1.45***		1.38***
7 hours or less				1.16*	1.11		1.09
Physiological Indicators							
Obesity							
BMI (18.5 and <30)							
< 18.5						2.24***	1.73***
30 and < 35						0.96	0.98
35 or more						1.21*	1.27**
Inflammation							
Albumin (> 4.5 g/dL)							
3.5 g/dL						1.81***	1.91***
> 3.5 - 4.0 g/dL						1.13	1.14
> 4.0 - 4.5 g/dL						0.98	1.00
Missing because of infection						1.40*	1.45**
Missing						1.41	1.41
C-reactive protein (< 0.3mg/dL)							
0.3 mg/dL						1.19**	1.10*
Missing because of infection						1.38***	1.17***
Missing						1.13	0.95
Cardiovascular Risk Factors							
Normotensive (< 120 SBP and < 80 DBP)							
Prehypertensive (120-139 SBP or 80-89 DBP)						0.83*	0.84*
Hypertensive stage 1 (140-159 SBP or 90-99 DBP)						1.17**	1.16**
Hypertensive stage 2 (160 SBP or 100 DBP)						1.26***	1.27***
Missing						1.41**	1.30*
Total cholesterol (150-249)							
< 150						1.41**	1.39**
250-349						1.01	1.01
350 or more						1.73*	1.75*
Missing						1.01	1.06
Glycosylated hemoglobin (< 6.4 percent)							
6.4 percent or more						1.54***	1.54***
Missing						1.00	1.10

Notes: Referent is listed in parentheses. Sample size is 15,691 individuals and 4,985 deaths.

Source: Derived from NHANES III Linked Mortality File (2010).

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p .10

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p .05

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p .01

p .001

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Table 3
Hazard Ratios for Age-Specific Education Differences in Mortality, U.S. Adults Aged 25 and Over, 1988-2006

	Ages 25-59							Ages 60 and Older						
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Education (16 years)														
11 years	2.93 ***	2.16 ***	2.53 ***	1.91 ***	1.52 *	1.77 **	1.41 †	1.44 ***	1.18 †	1.27 **	1.28 *	1.05	1.22 *	1.03
12 years	2.11 ***	1.75 ***	1.95 ***	1.63 **	1.43 *	1.54 *	1.33 †	1.37 ***	1.21 **	1.30 ***	1.24 **	1.12 †	1.22 **	1.12
13-15 years	1.79 **	1.59 *	1.73 **	1.54 *	1.44 *	1.51 *	1.40 †	1.20	1.11	1.15	1.14	1.07	1.10	1.05
Demographic Factors														
Sex (female)	1.38 ***	1.41 ***	1.34 ***	1.29 *	1.29 **	1.40 ***	1.39 ***	1.55 ***	1.60 ***	1.57 ***	1.46 ***	1.51 ***	1.57 ***	1.60 ***
Race/ethnicity (non-Hispanic white)														
Non-Hispanic black	1.69 ***	1.49 **	1.61 ***	1.56 ***	1.4 **	1.22 †	1.11	1.16 **	1.07	1.12 *	1.08	1.02	0.93	0.90 *
Mexican American	0.99	0.87	1.06	1.01	0.99	0.95	0.93	0.92	0.88 †	0.90	0.87	0.83 *	0.89	0.86 †
Foreign-born (US-born)	0.72	0.72 †	0.74	0.78	0.8	0.83	0.86	0.77 **	0.76 ***	0.76 **	0.81 **	0.79 *	0.79 **	0.77 **
Economic Resource														
Income-to-poverty ratio	0.87 ***				0.92 *		0.93 *	0.92 ***				0.94 ***		0.95 **
Social Resources														
Marital status (married)														
Divorced/separated			1.51 **		1.31 *		1.34 *			1.5 ***		1.36 ***		1.34 ***
Never married			1.50 *		1.44 *		1.36 †			1.07		1		0.90
Widowed			1.25		1.07		1.06			1.15 *		1.08		1.05
Visit w/ friends, relatives (1 visit per wk)			1.36 *		1.3 *		1.28 +			1.4 ***		1.3 **		1.27 **
Religious attendance (> once a week)														
No attendance			1.91 **		1.72 *		1.56 +			1.44 ***		1.28 **		1.23 *
< once a week			1.42 †		1.39		1.30			1.14		1.08		1.04
Once a week			1.24		1.24		1.13			1.07		1.03		1.01

	Ages 25-59							Ages 60 and Older						
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Club involvement (yes)		1.15			1.01		1.05			1.21***		1.15		1.13**
Health Lifestyle														
Smoking status (never)														
Former				1.21	1.21	1.21	1.23				1.32***	1.29***	1.31***	1.28***
Current			2.12***	2.12***	1.95***	2.23***	2.09***				2.08***	1.96***	2.02***	1.92***
Drinking status (1-4 drinks)														
Never			1.17	1.17	1.23	1.17	1.22				0.96	0.98	1.06	1.07
Former			1.23†	1.23†	1.27*	1.24†	1.27†				1.03	1.02	1.08	1.06
5 or more drinks			1.54*	1.54*	1.42*	1.62**	1.50*				1.34†	1.20	1.45*	1.31 +
Missing			---	---	---	---	---				3.26*	2.28*	3.57**	2.56**
Hours of exercise per week (> 7 hours)														
None			1.73***	1.73***	1.57**	1.63***	1.49				1.54***	1.41***	1.45***	1.34***
7 hours or less			1.15	1.15	1.13	1.09	1.07				1.16*	1.11†	1.14*	1.10†
Physiological Indicators														
Obesity														
BMI (18.5 and <30)														
< 18.5						1.42	1.32						1.97***	1.93***
30 and < 35						1.13	1.11						0.93	0.90
35 or more						1.37*	1.33†						1.18 +	1.18 +
Inflammation														
Albumin (> 4.5 g/dL)														
3.5 g/dL						2.07**	2.03**						2.01***	1.98***
> 3.5 - 4.0 g/dL						0.98	0.98						1.24	1.23
> 4.0 - 4.5 g/dL						0.84	0.86						1.09	1.09
Missing because of infection						1.32	1.35						1.50*	1.52*

	Ages 25-59							Ages 60 and Older						
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Missing						0.55	0.60					1.78		1.74 [†]
C-reactive protein (< 0.3mg/dL)														
0.3 mg/dL						1.34 [*]	1.31 [*]					1.05		1.04
Missing because of infection						1.15	1.13					1.23 ^{***}		1.20 ^{**}
Missing						1.13	1.11					1.03		0.97
Cardiovascular Risk Factors														
Normotensive (< 120 SBP and < 80 DBP)														
Prehypertensive (120-139 SBP or 80-89 DBP)						0.86	0.86					0.83 [†]		0.84 [†]
Hypertensive stage 1 (140-159 SBP or 90-99 DBP)						1.52 [*]	1.11 [*]					1.04		1.04
Hypertensive stage 2 (160-179 SBP or 100-109 DBP)						1.76 ^{***}	1.33 ^{***}					1.17 ^{**}		1.15 [*]
Missing						1.22	1.19					0.82 [*]		1.29 [*]
Total cholesterol (150-249)														
< 150						2.10 ^{***}	1.97 ^{***}					1.12		1.11
250-349						0.88	0.87					1.07		1.05
350 or more						1.70	1.59					1.79 [*]		1.88 [*]
Missing						2.11	1.85					0.82		0.90
Glycosylated hemoglobin (< 6.4 percent)														
6.4 percent or more						1.31 [*]	1.35 [*]					1.58 ^{***}		1.57 ^{***}
Missing						1.13	1.18					1.13		1.08

Notes: Referent is listed in parentheses. Sample size (with deaths in parentheses) is 6,532 (333) for ages 25 to 44, 2,957 (537) for ages 45 to 59, and 6,202 (4,115) for ages 60 and over.

Source: Derived from NHANES III Linked Mortality File (2010).

[†] p .10
^{*} p .05
^{**} p .01

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Table 4

Hazard Ratios for Education Differences in Mortality by Major Causes of Death, U.S. Adults Aged 25 and Over, 1988-2006.

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Cardiovascular Diseases							
Education (16 years)							
11 years	1.70 ***	1.41 **	1.46 **	1.41 **	1.09	1.32 *	1.06
12 years	1.53 ***	1.34 **	1.43 **	1.34 **	1.17	1.30 *	1.15
13-15 years	1.44 *	1.32	1.38 †	1.32	1.21	1.26	1.17
Cancer							
Education (16 years)							
11 years	1.56 *	1.30	1.47 *	1.24	1.07	1.20	1.06
12 years	1.65 ***	1.48 **	1.57 ***	1.37 *	1.25	1.34 *	1.23
13-15 years	1.21	1.14	1.18	1.07	1.02	1.06	1.02
Respiratory Diseases							
Education (16 years)							
11 years	2.11 *	1.64 *	1.78 *	1.65 *	1.36	1.61 *	1.34
12 years	2.15 **	1.85 *	1.97 *	1.75 *	1.56 †	1.76 *	1.59 †
13-15 years	1.57 *	1.43 †	1.49 †	1.37	1.28	1.39	1.29
External Causes							
Education (16 years)							
11 years	4.53 *	5.95 *	3.69 *	3.98 *	4.52 *	3.88 *	3.43 †
12 years	1.41	1.66	1.26	1.29	1.41	1.24	1.16
13-15 years	2.15	2.38	2.06	2.05	2.22	1.98	1.97
Other Causes							
Education (16 years)							
11 years	1.63 **	1.25	1.48 *	1.41 *	1.13	1.26	1.05
12 years	1.37 *	1.16	1.32 †	1.24	1.10	1.16	1.05
13-15 years	1.25	1.13	1.21	1.19	1.10	1.13	1.05

Notes:

Model 1 controls for sex, race/ethnicity, and nativity.

Model 2 controls for variables in Model 1 and economic resources (income-to- poverty ratio).

Model 3 controls for variables in Model 1 and social resources (marital status, religious attendance, club membership, and visits from friends).

Model 4 controls for variables in Model 1 and health lifestyles (smoking status, alcohol consumption, and exercise).

Model 5 controls for variables in Model 1, Model 2, Model 3, and Model 4.

Model 6 controls for variables in Model 1 and physiological indicators (obesity, albumin, CRP, hypertension, cholesterol, and glycosolated hemoglobin).

Model 7 controls for variables in Models 1 through 6.

Source: Derived from NHANES III Linked Mortality File (2010).

χ^2
p .10

*
p .05

**
p .01

p .001