Educational Attainment and Age, Period, and Cohort Patterns of U.S. Adult White Male Mortality*

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Abstract

We investigate educational differences in the temporal changes to U.S. non-Hispanic white men's adult mortality risk between 1986 and 2002. Using recently developed hierarchical age-period-cohort cross-classified random effects models (HAPC-CCREM), we simultaneously measure age, period, and cohort effects of mortality risk between 1986 and 2002 for men aged 40 and above with a less than high school education, a high school education, and a more than high school education, respectively. All-cause mortality risk and mortality risk for heart disease, lung cancer, non-lung cancer, and residual causes are examined. Findings reveal that temporal changes to U.S. non-Hispanic white male adult mortality between 1986 and 2002 were driven entirely by cohort reductions in mortality. Findings also demonstrate that disparate cohort effects between education groups widened the education gap in mortality risk across this time period. This research highlights the widening educational differences in U.S. men's mortality and supports the contention that a cohort perspective is needed in order to best understand recent temporal changes in U.S. mortality risk.

Introduction

It is well documented that U.S. adult all-cause mortality risk declined substantially across the twentieth century (NCHS 2009; Jemal et al. 2008; Guyer et al 2000; Crimmins 1981; Fogel 2004). Across this time period, steady gains in overall life expectancy have been made by reducing mortality risk of degenerative diseases for middle and older age groups (Yang 2008). Recent research has further demonstrated that all-cause mortality has continued to decline during the 2000s as well (NCHS 2009; Jemal et al 2008). This research has largely attributed U.S. mortality declines to temporal changes in a handful of specific causes of death, chief among these being degenerative diseases like heart disease, lung cancer, and other cancers (Jemal et al 2008; Guyer et al 2000; Cooper et al. 2000). Recent findings, however, have also shown that declines in mortality rates for some causes of death have slowed or halted altogether (Yang 2008). Furthermore, despite continued declines in both all-cause and specific-cause death rates, education disparities in U.S. mortality risk have persisted or even widened across time (Lauderdale 2001; Pappas et al. 1993; Meara et al. 2008; Montez et al. 2009).

In this paper, we analyze educational differences in temporal changes in adult mortality risk of U.S. non-Hispanic white men between 1986 and 2002. We do so in the following way. First, we unite three perspectives of mortality risk – Link and Phelan's "fundamental cause" theory (1995), the life course perspective, and Fogel and Costa's theory of "technophysio evolution" (1997) – to frame recent temporal changes in educational disparities of mortality risk. We argue that each perspective lends itself to understanding components of U.S. mortality changes across the latest portion of the twentieth century. However, by uniting the three, we believe we are able to better understand how education's effects on mortality risk are changing over time. Second, we employ new data and methods to simultaneously examine how age, period, and cohort effects of U.S. adult mortality risk differ by educational attainment. Lastly, we

conclude by advocating the use of a cohort perspective of mortality change over the more commonly used period perspective. Indeed, we argue that only by analyzing temporal changes in mortality and education within a cohort perspective can we fully understand trends in U.S. mortality risk as well as differential trajectories of those trends.

Theoretical Background

The general trend in U.S. mortality risk over the twentieth century was toward increasing life expectancy and a steady reduction in mortality risk for most infectious and degenerative diseases. Recent research has documented that this pattern of mortality reduction continued into the 2000s as well (Jemal et al 2008; NCHS 2009). Overall, U.S. life expectancy at birth rose about 63 percent, from an estimated 47.3 years in 1900 to 77.0 years in 2000, and was recently projected to be 78.3 years in 2010 (NCHS 2009). However, this overall trend is comprised of variations in patterns of specific diseases and causes of death, differences in ages most affected, and disparate rates of reductions across subpopulations. To fully understand factors behind the overall trends, it is necessary to analyze these different components.

The majority of research on overall mortality reductions in the United States has emphasized the changing educational composition of the U.S. population (Lleras-Muney 2005; Meara et al. 2008), changing lifestyle and risk factors – especially smoking patterns and diet – (Pampel 2002, 2003; Manton et al. 1997), and advances in medical technologies to combat degenerative and chronic diseases (Chang and Lauderdale 2009). Much of the literature on U.S. mortality trends, however, has framed the reductions within a period perspective. That is, many studies merely document changes to age-specific death rates across time periods (Jemal et al. 2008). Further, it is often the case that reductions in age-specific mortality rates across a given time period are attributed to policies or health-related efforts *in* that time period (Healthy People 2010). Only recently have researchers begun to appreciate the degree to which cohorts' differing

experiences fundamentally shape mortality risk, and how these differing experiences affect mortality trends (Fogel 2004, 2005; Costa 2002; Yang Yang 2008; Finch and Crimmins 2004; Manton, Corder, and Stallard 1997; Lauderdale 2001; Preston and Wang 2006). Compared to age and period effects, cohort effects have been given much less attention in studies of variations in U.S. mortality risk. While many period changes have influenced past temporal shifts in mortality risk (e.g., water and sewage treatment efforts reduced mortality risk early in the 20th century), recent reductions in U.S. mortality have been predominantly driven by cohort phenomena (Yang 2008).

Our goal in this paper is to build off existing cohort perspectives of mortality risk by integrating the important role education has played in shaping cohort exposures to mortality risk. What follows is a brief overview of, first, the relationship between educational attainment and mortality risk, and, second, the emerging cohort perspective of mortality risk.

Educational Attainment and Adult Mortality Risk

The association between education, health, and mortality is widely studied in the population sciences. In the United States, the education-mortality relationship was most thoroughly demonstrated with comprehensive national-level data by Kitagawa and Hauser in 1973. Since then, a number of theories have been advanced and many studies undertaken to explain and document the ways by which education affects health, morbidity, and mortality risk (Adler and Newman 2002; Beckett 2000; Elo and Preston 1996; Feldman et al. 1989; Freedman and Martin 1999; Goesling 2007; Link and Phelan 1995; Lynch 2003, 2006; Meara et al. 2008; Mirowsky and Ross 1998, 1999, 2003, 2004; Pappas et al 1993; Preston and Elo 1995; Ross and Wu 1995, 1996; Sorlie et al. 1995). In general, it has been repeatedly found that those in the United States with relatively low levels of education have significantly higher risks of mortality than those

with higher levels of education. The association between education and mortality risk has fascinated health researchers, in one part because educational disparities in mortality risk are found even after controlling for other measures of socioeconomic status, such as income (Lynch 2006; Rogers et al. 2000). While it is widely acknowledged that education's association with health and mortality is largely mediated by economic resources such as income, the association has been explained in terms of social-psychological support and health behaviors/risk factors as well (Mirowsky and Ross 2003; Ross and Wu 1995; Lynch 2006). In short, beyond its association with improved economic and material resources, extensive research has shown that education plays a vital role in reducing exposure to stress (Ross and Wu 1995), increasing self-efficacy and control over one's life (Mirowsky and Ross 1998, 2003), and increasing awareness about, and practice of, beneficial health behaviors (Link and Phelan 1995; Chang and Lauderdale 2009; Lynch 2003).

In this sense, education has recently been framed as a "fundamental social cause" of health and mortality risk (Link and Phelan 1995, 1996, 2000; Link et al. 2008). This is because education shapes individual-level "resources like knowledge, money, power, prestige, and social connections that strongly influence people's ability to avoid risks and to minimize the consequences of disease once it occurs" (Link and Phelan 1996: 472). A central tenet of the fundamental social cause theory of health and mortality risk is the staying power of the effects of education. That is, despite changes to our understanding of disease, changes to our healthy behaviors and risk factors, and changes to our treatment of disease and disability, the association between education and mortality persists. As a fundamental cause, the mechanisms by which education shapes health, morbidity, and mortality risk may change, but the strong relationship between education and health and mortality risk remains. That is, the well-educated have largely been, and continue to be, the most likely to have access to, and take advantage of, new knowledge, practices, and/or technologies that are related to morbidity and mortality risk (Link

and Phelan 1995; Link 2008; Chang and Lauderdale 2009). Unfortunately, direct tests of the fundamental cause theory are few, largely because we lack the data to track education's association with mortality risk across long periods of time. Our paper aims to add to this literature by detailing education's lasting effects on mortality risk during a period of rapid health technology development, rising socioeconomic inequality (Campbell et al. 2005), and decreasing mortality risk (NCHS 2009). But beyond this, we aim to unite this fundamental cause literature with the growing literature on the cohort-life course perspective of mortality trends.

Educational Attainment and Trends in U.S. Adult Mortality Risk

Efforts to study trends of the association between education and health and mortality risk in the United States have generally focused on disparate effects across ages, periods, or cohorts (APC) separately. No effort has been made to simultaneously measure the education-mortality association across all three APC components. The literatures concerned with the latter two, period and cohort, have generally concluded that the effect of overall socioeconomic status, education included, was quite strong at the beginning of the twentieth century (Warren and Hernandez 2007), but that the relationship had waned by the mid-twentieth century as the U.S. population underwent the epidemiologic transition. This is evidenced by the fact that the leading emerging degenerative diseases in the mid-twentieth century United States – heart disease, stroke, and other undiagnosed and/or at the time untreatable diseases – affected all subpopulations in a similar way. In fact, for the U.S. male population, a positive association between SES and coronary heart disease existed during the 1940s and 1950s, due in large part to high levels of smoking and meat consumption among high-SES men (Manton et al. 1997). Only after greater knowledge of the risk factors was gained, and developments of medical technologies to prevent and treat degenerative diseases were made, did researchers begin to note

a protective educational effect in U.S. prevalence rates of heart disease, stroke, and some cancers.

Beyond the general consensus that education significantly affects mortality risk, research has become increasingly concerned with the ways by which the relationship changes across age as well as time. The life course literature has emphasized that education significantly and substantively conditions mortality and morbidity risk across age (House et al. 1994; Beckett 2000; Ross and Wu 1995; Lynch 2003). Furthermore, debate has arisen concerning the way(s) by which the substantive effect changes across age. While some research has found that educational differences in mortality risk are lessened and/or disappear at the oldest age groups (Beckett 2000), most mortality work in this area strongly suggests that mortality selection is responsible for much of the smaller relative educational gaps in old age mortality (Crimmins 2005; Hummer and Lariscy 2010; Lynch 2003). Unfortunately, the existing literature has largely omitted cohort effects from life course theories of health and mortality risk, an omission that has limited the field's understanding of the relationships between education, age, and mortality. Specifically, most life course research on the relationship between education and health and mortality focuses solely on age effects. That is, much of the literature is concerned only with the ways in which education affects disability and mortality risk across age. Only minimal efforts have been made to understand how the aging process may be changing across cohorts (Lauderdale 2001). And yet, one could argue that there is no single "life course" to speak of, but rather that each cohort experiences a distinct life course, shaped by the confluence of age effects and each cohorts' unique experience of history. While it may be both possible and useful to theorize about an abstract or ideal-type life course (since age patterns of mortality indeed show robust regularities across populations and historical times), we should also consider the possibility that cohort-specific life courses exist, and can vary tremendously in their effects on health and mortality risk.

It follows, then, that the way by which education conditions mortality risk across age is itself changing across cohorts. This could be the result of changes to education composition across cohorts, changes to the qualitative meaning of education across cohorts, or changes to other cohort-related risk-factors as well. Figure 1 presents the changes to non-Hispanic white men's educational attainment across birth cohorts within the 1986-2002 data that we will use to analyze temporal changes to educational disparities in mortality risk. The most obvious and dramatic change in the educational composition is in the precipitous drop in percent of the population with a less than high school education between the 1900 and 1945 birth cohorts. The overall pattern demonstrates a rapid shift in the educational attainment of the U.S. non-Hispanic white male population across cohorts, from a majority of the population having a less than high school education (1900 birth cohort) to the vast majority of the population having at least a high school education (>90 percent by the 1945 birth cohort). This dramatic increase in educational attainment across the twentieth century can have tremendous implications for the way morbidity and mortality risk unfolds across age. First, at the population-level, we know both the distribution and content of education has changed over time, suggesting that the relationship between education and mediators of health (e.g., income and other resource attainment, knowledge and practice of health-enhancing behaviors, and/or other resources such as autonomy, coping mechanisms, social support, etc.) have also changed over time (Lynch 2003). Second, the association between education's mediators and health have likely changed (Lynch 2006), as inequality has risen (Campbell et al. 2005), healthcare costs have increased (Kronick and Gilmer 1999), and/or medical technologies' effectiveness at preventing disease and/or preserving life is improving (Chang and Lauderdale 2009). Taken together, the exposure to risk factors of morbidity and mortality has become more varied. Third, nearly all increases in life expectancy are now made by reductions of mortality risk in mid- and older-adult age groups (NCHS 2009). That is, the reductions in infant- and child-mortality risk have assured survival into young

adulthood for nearly all Americans and survival into adulthood for most Americans. With the greatest variation in mortality risk occurring later in life, pathways to poorer health may have changed, and the effect of education may become ever more important. As Link (2008) argues, "social factors [such as education] have become more important precisely because epidemiological and biomedical knowledge has shifted the causes and consequences of disease from fate, accident, and bad luck to factors that are under some human control" (367). As a result of these potentially changing factors, it is becoming more important to integrate a cohort perspective into the life course understanding of mortality risk.

Cohort Perspectives

Increasingly, evidence is supporting the contention that life course effects of mortality risk, education-based or not, are indeed changing across cohorts. Cohorts differ in their exposure to the benefits of medical inventions, public health measures, and improvements in nutrition. And thus, cohorts' varying exposures to risk factors and health-enhancing knowledge and technologies will invariably influence cohorts' morbidity and mortality risks across their life courses. The changes in both the endowment of health capital across birth years and the depreciation of health across age should affect both disability and mortality risk across different cohorts' life courses. Indeed, evidence increasingly points to the important effect that lifetime exposure to infectious disease and bouts of inflammation have on subsequent health and mortality risk (Blackwell, Hayward, and Crimmins 2001; Finch and Crimmins 2004; Costa 2000; Fogel 2004, 2005). Analyzing mortality experiences of Swedish cohorts between 1751 and 1940, Finch and Crimmins (2004) find that those cohorts that were the first to experience lowered infant and childhood mortality were also the first to experience subsequent declines in older age mortality. It is believed, as such, that reductions in exposure to inflammation and infectious in early life has directly led to decreases in subsequent chronic disease morbidity and mortality later

in life. Finch and Crimmins thus argue that "improved childhood health and survival along with reduced chronic infections and inflammation…help to explain the widespread recent declines in old-age mortality" (1739). The authors posit that the aggregated insults of these early infections essentially scar a cohort, and this scarring persists across their life course. Indeed, in their own words, these "enduring effects of early environment, even if conditions improved at later periods, could be designated as a 'cohort morbidity phenotype'" (1737). As cohorts differ in the magnitude of their "morbidity phenotype," it follows that they also differ in the subsequent older age mortality risk.

Fogel (2005), Costa (2002), and Fogel and Costa (1997) have found similar results with various data sources, although their work has generally emphasized the synergism between improved nutrition, intergeneration transmissions of health endowments at birth, improved health-enhancing technologies, and reductions in early-life hardships across cohorts. Their theory of "technophysio evolution" thus implies strong cohort effects in terms of both changes to health endowments at birth, and disparate exposures to health risk across the life course. Arguing on behalf of a cohort perspective, Fogel (2005) states, "not all improvements in the outcome of exposure to health risks between, say 1970 and 1990 are due to health interventions during that period. It could also reflect the improved physiologies experienced by later birth cohorts that are due to improved technologies in food production, public health practices, personal hygiene, diets, and medical interventions put into place decades before 1970, and hence cannot be attributed exclusively, perhaps even primarily, to health inputs between 1970 and 1990" (S163). Manton et al. (1997) made similar arguments in their demonstration of cohort effects on both survival and functional capacity across age. A number of improvements to diet (e.g., vitamin D supplementation during the 1920s, increases in vitamin B_6 fortified foods across the 1940s and 1950s, commercial food processing and increases in food regulation after the 1950s) and medical knowledge and practices (e.g., Jones Criteria for identifying and treating rheumatic fever) were

made across time. The combined effects of these health-enhancing developments on reducing chronic disease and mortality risk were largely related to cohorts' varying exposure times to their benefits.

Thus, like Finch and Crimmins, Fogel (2005) also argues that reductions in disparities in childhood mortality are leading to subsequent reductions in degenerative disease-related mortality risk at older ages as well. Citing work using Union Army data from the Early Indicators Project, Fogel shows that significant delays in the onset of chronic diseases across the twentieth century are linked to reductions of exposure to poor health early in life. Costa (2000) estimates that as much as 10-25 percent of the decline in specific older aged chronic disease in the United States between 1900-1910 and 1971-1980 was due to decreases to specific infectious diseases during early ages. Further, as Fogel (2005) shows, both the overall prevalence and the disparities in poor early life conditions in the United States have decreased substantially. Consequently, as bouts with infection, malnutrition, and inflammation early in life have been greatly reduced across the twentieth century, the "insults" they imprint on the "cohort morbidity phenotypes" of successive birth cohorts are becoming less and less important in determining the risk of older adult morbidity and mortality risk. It follows, then, that improvements in adult conditions, rather than early life conditions, across cohorts are becoming increasingly important in shaping adult mortality risk.

Current Aim

In this paper, we draw from the life course and cohort perspectives of mortality risk to analyze trends in educational disparities in U.S. non-Hispanic white male adult mortality risk between 1986 and 2002. Specifically, we hypothesize that: (1) the educational gap in U.S. adult mortality risk is widening, and (2) that the educational gap in mortality is widening across cohorts. These hypotheses are predicated on the following argument. First, as asserted by Finch

and Crimmins (2004), “changes in the epidemiological environment that occur within a given historical period…affect surviving members of cohorts for the rest of their lives” (1737). These enduring effects leave imprints on the “cohort morbidity phenotype,” shaping the life course mortality risk of cohorts. Second, as demonstrated by Fogel (2004, 2005), Costa (2000 and 2002), and Fogel and Costa (1997), the importance of early childhood environment on subsequent morbidity and mortality risk has been greatly reduced since the beginning of the twentieth century. Fogel specifically shows that, while “insults from infectious diseases at early ages have a large impact on the prevalence rates of chronic diseases and disabilities [and mortality] in middle and late ages” (Finch and Crimmins 2004), “the age-specified prevalence rates of chronic diseases were much lower at the end of the 20th century than they were at the beginning of the century” (Fogel 2005: S152). Thus, while it was once common in the United States for infants, children, and young adults to endure bouts of infectious diseases, inflammation, and other forms of “insults” to their “cohort morbidity phenotypes” and lasting health, improvements in childhood environments, better nutrition at all ages, and advancements in medical knowledge and care have, over time, greatly reduced the severity of early-life hardships. As the effects of early childhood health and infectious diseases on subsequent chronic disease and mortality have been reduced, the relative effects of one’s individual attributes in adulthood are argued to have increased. Third, according to fundamental cause theory, educational status conditions one’s exposure to health-related resources, knowledge, and/or use of these resources and knowledge. Consequently, educational attainment ought to be growing increasingly important in conditioning U.S. cohorts’ knowledge and use of health-related resources, especially to reduce both exposure and vulnerability to chronic degenerative diseases. That is, if life course risk factors of degenerative disease-related morbidity and mortality have indeed been changing across cohorts, it is likely the case that education has profoundly affected the pattern of these changes.

While this cohort effect of education on health and mortality risk has been widely discussed (Yang 2008), it has been infrequently analyzed (Lauderdale 2001; Link 2008; Lynch 2006). Thus, our goal in this paper is to demonstrate that temporal changes to U.S. adult mortality risk have primarily been a cohort-based phenomenon, and that these changes have been significantly conditioned by educational attainment. It is believed that education should be expected to influence (1) the time one is exposed to "chronic inflammatory mechanisms" and/or health-enhancing knowledge and practices (Finch and Crimmins 2004), (2) the subsequent health effects this exposure time has, or (3) both. If any of these are the case, we should expect to see significant educational differences in cohort-based reductions of U.S. adult mortality risk. To achieve our aim, we employ recently develop hierarchical age-period-cohort cross-classified random effects modeling techniques (HAPC-CCREM) to analyze age, period, and cohort patterns of non-Hispanic white men's U.S. mortality risk between 1986 and 2002.

Data

We use the National Health Interview Survey (NHIS) 1986 through 2000, linked to the National Death Index (NDI) via the Multiple Cause of Death (MCD) file, through the end of 2002 (NCHS 2006) [1]. The NHIS uses a multistage probabilistic sampling design, and respondents of the NHIS are matched to the MCD mortality files using a 14-item identification scheme (NCHS 2009). Respondents of the NHIS not eligible for matches to the NDI are dropped from the final sample, and the use of analytical weights makes results from the NHIS-LMF representative of the noninstitutionalized U.S. adult population. The resulting 1986-2002 National Health Interview Survey-Linked Mortality Files (NHIS-LMF) are a unique combination of repeated cross-sectional survey waves coupled with longitudinal yearly records of individual respondents' mortality status. These data have several advantages for studying the trends in educational differences in U.S. mortality risk across cohorts and time. First, ages of NHIS

respondents are self-reported. This important feature of the NHIS increases confidence in old-age mortality and older-cohort estimates. Secondly, combining the repeated cross-sections of the NHIS with the individual-level longitudinal mortality histories breaks the linear dependency of age, period, and cohort. Third, because links between the NHIS surveys and mortality follow-up range from 1986 to 2002, there is sufficient overlap between age, period, and cohort to estimate stable and reliable effects of all three variables [2]. In order to improve confidence in age reports, we further refined the NHIS-LMF data by computing new ages at time of survey based on respondent-reported month and year of birth, and the year of interview and quarter-year of interview [3].

In order to assure enough time for individuals to complete all levels of educational attainment, to focus on ages where mortality risk is high, and to limit the use of data where age is top coded, we restricted the NHIS-LMF to respondents aged 40 to 84 at time of survey. Current analyses also focus exclusively on non-Hispanic white men, because U.S. mortality is highest among men and because of the high level of complexity of the methods and models in this analysis. Limiting the data to this sub-population at these ages trimmed the starting sample size to 319,574 non-Hispanic white male respondents for survey years 1986 through 2000. After subsequent mortality and exposure times were calculated, the resulting person-period dataset consisted of 2,458,826 person-years, ranging from ages 40 to 100. These 2,458,826 person-years were then stratified by three levels of educational attainment and collapsed into subsamples of five-year age-period-cohort blocks.

The three categories of educational attainment used in the analyses are less than high school, high school or equivalent, and greater than high school. The coding of birth cohort is comprised of 16 five-year blocks ranging from the five-year 1900-1904 group to 1960-1964, and coding for period is comprised of four five-year blocks ranging from 1985-1989 to 2000-2004. The period measurements, however, are not complete five-year blocks for the 1985-1989 and

2000-2004 groupings. This is because the NHIS-LMF data used in these analyses begins in 1986 and ends in 2002. Aggregated counts of deaths as well as aggregated person-years lived across the five-year time frame were used to compute five-year age-specific mortality rates for each education subsample. The final aggregated samples were each comprised of 87 five-year age-period-cohort blocks. Table 1 displays descriptive statistics for the individual-level sample, the person-period sample, and the collapsed age-period-cohort-education samples.

[Table 1 About Here]

Analytic Methods

To model age, period, and cohort patterns of U.S. mortality risk for educational groups of non-Hispanic white men, we use recently developed hierarchical age-period-cohort (HAPC) models for repeated cross-sectional survey data (Yang and Land 2006). These methods utilize a cross-classified random effects model (CCREM) to embed each respondent within both a five-year time period and birth cohort at a given five-year age group. Goodness-of-fit statistics (see Table 2) from fixed effects models of age-period-cohort analyses verify that all three effects should be included in the final models.

[Table 2 About Here]

Because the NHIS-LMF 1986-2002 follows individual mortality risk as each respondent ages across periods, each respondent can occupy several age-period-cohort combinations. Thus, while collinearity between the three effects is very high, these data do not suffer the "identification problem" induced by an absolute linear dependency between age, period, and cohort (Mason 1973; Glenn 2004). Nevertheless, the HAPC-CCREM modeling is an appropriate

methodological tool to measure the three processes simultaneously, and has been shown to be more efficient than a fixed effects approach when data, such as the NHIS-LMF, are unbalanced (Yang and Land 2006). Also, fixed effects models (HAPC-CCFEM) were run to both compare and test results from the HAPC-CCREMs, and also to guide our choice of constrained covariance parameters when needed (HAPC-CCREM results not shown). The HAPC-CCREM estimates fixed effects of the five-year age groups and random effects of the five-year period and five-year cohort groups, and is structured in the following way:

Level-1 within cell model: $$\ln(R_{ijk}) = \alpha_{jk} + \beta_{jk} A_i + \ln(\exp_{ijk}) + e_{ijk}$$

where R_{ijk} stands for the counts of deaths of the ith age group for $i = 1, \ldots, n_{jk}$ age groups within the jth period for $j = 1, \ldots, J$ time period and the kth cohort for $k = 1, \ldots, K$ birth cohort; A_i denotes the dummy five-year age groups $1,\ldots, n_{jk}$; α_{jk} is the intercept indicating the reference age group (65-69) who was in period j and belong to cohort k; $\ln(\exp_{ijk})$ is the natural log of the aggregated exposure time lived during the five-year age-period-cohort cell; and e_{ijk} is the random cell residual.

Level-2 between cell random intercept model: $$\alpha_{jk} = \pi_0 + t_{0j} + c_{0k}$$

in which α_{jk} specifies that the fixed age effects vary from period to period and from cohort to cohort. π_0 is the expected mean at the reference age (65-69) averaged over all periods and cohorts; t_{0j} is the overall 5-year period effect averaged over all five-year birth cohorts with variance σ_{t0}; and c_{0k} is the overall 5-year cohort effect averaged over all five-year periods with variance σ_{k0}.

We combine the level-1 and level-2 models to estimate counts of deaths in each 5-year age-period-cohort cell using SAS PROC GLIMMIX with a log-linear Poisson family, offsetting the aggregated person-years lived across the cells to generate age-period-cohort specific

mortality rates. Due to collinearity and small cell sizes in some age-period-cohort combinations, HAPC-CCREM models did not converge at some education levels for some causes of death. When this was the case, we either dropped one or several age-period-cohort cells from the analyses and/or constrained either the period or cohort covariance parameters with appropriate values. Multiple values were chosen and results were contrasted with results from corresponding HAPC-CCFEMs as well as other HAPC-CCREMs to guide our final selection of constrained values [4].

Results

Table 3 presents estimates of fixed effects age coefficients and random effects period and cohort coefficients from analyses of all-education/all-cause and all-education/specific-cause mortality risk. We first present these overall trends of all-cause and specific-cause mortality between 1986 and 2002 to introduce the general patterns of age, period, and cohort patterns of non-Hispanic white men's U.S. adult mortality risk. Next we discuss the educational differences in age, period, and cohort patters of all-cause mortality risk between 1986 and 2002. These results are found in Table 4. To conclude, we then examine the educational differences in age, period, and cohort patterns of mortality risk from several leading causes of death. The logged rates of both age and cohort effects of mortality from each education model are presented in graphical form to illustrate key findings.

Trends in All-education/All-cause and All-education/Specific-cause Mortality

Results from HAPC-CCREM analyses of all-cause adult mortality risk between 1986 and 2002 for non-Hispanic white men in the United States are consistent with similar analyses that Yang (2008) conducted using vital statistics data across the latter part of the twentieth century. As presented in Table 3, age effects follow the traditional log-linear pattern, with slight tapering at

the oldest-old age groups (85+). This is the case for all-cause and most specific-causes of death, with the exception of lung-cancer mortality risk. The distinct age pattern of lung-cancer mortality risk has age effects rising much more steeply than other specific-causes of death, but these taper off around age 65 and plateau thereafter across all older age groups. More relevant to our current aim, and also consistent with Yang's (2008) findings, the results from these HAPC-CCREM analyses of all-cause and specific-cause mortality risk suggests that temporal changes in U.S. mortality risk across 1986 and 2002 were driven entirely by cohort processes.

[Table 3 About Here]

The period covariance coefficients in all models displayed in Table 3 are insignificant and substantively very small. On the other hand, the cohort covariance parameters, while variable across the causes of death, are all quite large. These preliminary results are consistent with previous findings and support the argument that cohort processes were driving temporal changes in U.S. mortality risk between 1986 and 2002. Specifically, the residual cohort variation for all-cause non-Hispanic white male adult U.S. mortality risk is both significant and substantively large at .330, whereas the residual period variation is quite small and insignificant at most commonly used α-levels.

Educational Differences in Trends in All-cause Mortality

Table 4 presents estimates of fixed effects age coefficients and random effects period and cohort coefficients from HAPC-CCREM analyses of all-cause U.S. mortality risk of non-Hispanic white men, stratified by the aforementioned three levels of educational attainment. Overall, the stratified models reveal tremendous educational variation in the size of both age and

cohort effects, but very little variation across periods. The effects of age for each education group are best displayed in Figure 2.

[Figure 2 About Here]

Notable in these results is the finding that the educational difference in mortality risk exists at all age-groups. In the education-life course literature, much is made about the way education conditions the aging process. While some researchers have found that the education-gap in health and mortality risk is preserved or even wider at older ages (Ross and Wu 1996; Lynch 2003), others contend that educational differences in mortality risk converge or even crossover at the oldest ages (Beckett 2000). Our current results are consistent with the former argument. We see lower estimates of mortality risk for men with education "greater than high school" than men with education "less than high school" at all ages. While at the most advanced ages the estimated differences are insignificant, this is entirely due to small cell sizes and, thus, larger standard errors. We find no evidence of the three education groups' point estimates of mortality risk converging or crossing over at any age group.

The educational-based heterogeneity behind the shared cohort variation in the all-cause mortality analysis (.330) is apparent in the results of the education-stratified models presented in Table 3. Both the "high school" and "greater than high school" education groups have significantly large cohort residual variances, .222 and .253 respectively, while the "less than high school" group has a small and statistically insignificant .036 cohort residual variance. The cohort effects of all-cause mortality for the age reference category (age 65-69) averaged across all periods are graphically depicted as log-rates in Figure 3. Here we can see the dramatic declines in all-cause mortality across 1986-2002 experienced by men with either a "high school" education or a "greater than high school" education. While men with a "greater than high school"

education maintain their lower mortality risk across all birth cohorts, the reduction in mortality risk across birth cohorts for both education groups is roughly the same. Between 1986 and 2002, the mortality risk of each successive five-year birth cohort for the male population with at least a high school education was lower than the previous five-year birth cohort.

[Figure 3 About Here]

Contrasted with these declines in all-cause mortality risk is the experience of those men with a "less than high school" education. For this education group, between 1986 and 2002 we see that a very slow, but steady, reduction in all-cause mortality risk took place across cohorts 1905 to 1935. Starting with the 1940-1944 birth cohort, however, men with "less than high school" educational attainment experienced no significant cohort reduction in mortality risk between 1986 and 2002. It was as if men in these birth cohorts with this educational attainment level were being left behind of the mortality reduction experienced by the rest of the population. Thus, the education gap between the least educated and the most educated in all-cause mortality risk widened across birth cohorts between 1986 and 2002. However, there are essentially two processes behind the growing divide. First, between 1986 and 2002, the education gap widened for cohorts born between 1900 and 1935 because the rate of mortality reduction experienced by men with either a "high school" education or a "greater than high school" educational attainment outpaced the rate of mortality reduction experienced by men with a "less than high school" educational attainment. For cohorts born after 1935, however, the education gap in mortality risk widened much more, because men with a "less than high school" educational attainment born after 1935 did not experience any reduction in mortality risk between 1986 and 2002. Only men with a "high school" or "greater than high school" education level experienced mortality reductions in these cohorts.

As presented in Table 4, random effects period coefficients of all-cause mortality risk are found to be insignificant across all three levels of educational attainment. Nevertheless, the period effects of all-cause mortality risk for the age reference category (65-69) averaged across all cohorts are graphically depicted as log-rates in Figure 3. Evident is the fact that educational differences in mortality risk are retained across all periods, as no significant changes in period mortality risk occurs for any of the three education groups. Because period effects of non-Hispanic white men's all-cause mortality risk in the United States between 1986 and 2002 were found to be insignificant, no graphs are displayed of period effects of cause-specific mortality risk.

These findings suggest, first, that the reductions in non-Hispanic white men's U.S. adult all-cause mortality risk between 1986 and 2002 were driven entirely by cohort-related phenomena, and, second, that these reductions in mortality risk were significantly conditioned by educational attainment. As such, the education gap in non-Hispanic white men's U.S. adult mortality risk grew substantially across this period, and the trend suggests that the gap will continue to rise in the near future. This is because those birth cohorts that witnessed cohort declines in mortality risk for all education levels (1905-1935) will comprise an ever-shrinking portion of the future U.S. non-Hispanic white male population. As these cohorts die out, the population will increasingly be comprised of those birth cohorts (post-1935) that demonstrate a continued mortality decline for those populations with at least a "high school" education, but a stalling of mortality risk for those members with a "less than high school" education. To further illuminate the components of these trends, we next present the results from the educational-stratified HAPC-CCREM analyses of heart disease, lung cancer, non-lung cancer, and residual causes mortality risk.

Educational Differences in Trends in Mortality from Heart Disease, Lung Cancer, and non-Lung Cancer

Tables 5, 6, 7, and 8 present fixed effects age coefficients and random effects period and cohort coefficients of mortality risk for of U.S. non-Hispanic white men for specific-causes of death between 1986 and 2002. Results from HAPC-CCREM of educational differences in mortality risk from heart disease are in Table 5. Because heart disease was the leading cause of death between 1986 and 2002 in the United States, comprising upwards of one third of all deaths during the 1986-2002 period, the age, period, and cohort patterns of heart disease related mortality risk closely mimics the overall patterns of all-cause mortality risk.

[Table 5 About Here]

The educational differences in the fixed effects age coefficients are consistent with the educational differences in all-cause mortality risk. That is, the higher mortality risk for men with a "less than high school" education is estimated to be higher at all ages than for men with a "greater than high school education," further supporting the contention that education has protective health effects at all ages across the life course. Indeed, this is found to be the case for all-cause mortality risk, heart disease, lung cancer, non-lung cancer, and residual causes of death in the United States between 1986 and 2002. Regardless of the cause of death, in no case do we find evidence for an educational convergence or "crossover" in non-Hispanic white men's mortality risk after having controlled for cohort and period effects.

In order for the heart disease HAPC-CCREMs to converge for the "less than high school" and "high school" samples, we had to constrain each period residual covariance to equal the period residual covariance produced in the all-education heart disease HAPC-CCREM. Also, in order for the heart disease HAPC-CCREM to converge for the "greater than high school"

sample, we constrained the cohort residual covariance to .600. This value was chosen to correspond to the results from the HAPC-CCFEM of heart disease mortality risk for the "greater than high school" sample. (A brief discussion of the sensitivity of selecting different constrained residual covariance values is found in footnote 4). Both the "less than high school" and "greater than high school" education groups had higher cohort residual covariance in their respective heart disease HAPC-CCREMs than in their all-cause HAPC-CCREM. That is, for the least educated and the most educated men, we find significantly more cohort variation in their heart disease mortality risk than in their respective all-cause mortality risk. The "high school" sample experienced nearly the same cohort variation all-cause mortality risk as in heart disease mortality risk.

[Figure 6 About Here]

The substantive impact of the educational differences in cohort reductions in heart disease mortality risk between 1986 and 2002 are displayed in the top-left panel of Figure 6. Very similar to the trends observed in the all-cause mortality analyses, the cohort trends in heart disease mortality risk can be discussed in two parts. For birth cohorts 1905-1940, we see that men with a "greater than high school" education maintain a lower heart disease mortality risk than both the "high school" and "less than high school" subpopulations. The gap between the "less than high school" and "greater than high school" education groups grew across these birth cohorts between 1986 and 2002, because the cohort-based reductions in heart disease related mortality risk was faster for the better educated population. Post-1940 birth cohorts, however, experience different trends in heart disease mortality. Other than a suspicious drop for the 1950 birth cohort, men with a "less than high school" education exhibited a stalling, or an even rising, heart disease mortality risk during the period 1986-2002. The education groups "high school"

and “greater than high school” continued to experience cohort-based reductions in heart disease mortality risk during this time, with the notable exception of men born in 1960 with a “high school” education level. Whereas the men with the highest educational attainment continued, and even increased, their rate of heart disease mortality risk for the 1960 birth cohort, men born in 1960 who had only a “high school” education saw a sharp increase in their heart disease mortality risk between 1986 and 2002. Yang (2008) found evidence of U.S. male heart disease mortality risk stalling in these same birth cohorts. Here, we find that this overall stalling reflects the aggregated effect of a continued decrease for men with a “greater than high school” education, and an increase in heart disease mortality risk for both men with a “high school education” and “less than high school education.”

Results from HAPC-CCREM of educational differences in U.S. non-Hispanic white men’s adult mortality risk from lung cancer are displayed in Table 6. In order for the lung cancer HAPC-CCREMs to converge for the “less than high school” and “high school” samples, we had to constrain each period residual covariance to equal the period residual covariance produced in the all-education lung cancer HAPC-CCREM. Also, in order for the lung cancer HAPC-CCREM to converge for the “greater than high school” sample, we constrained the cohort residual covariance to equal the cohort residual covariance produced in the all-education lung cancer HAPC-CCREM.

[Table 6 About Here]

The results provide evidence that temporal changes to lung cancer related mortality for non-Hispanic white males between 1986 and 2002 were substantially conditioned by educational attainment. White men with a “less than high school” education level effectively experienced no significant reduction in lung cancer mortality risk within this time period. Both the cohort and

period covariance parameters are insignificant, and the graphed point estimates of log rates across cohorts (top right panel in Figure 6) demonstrates very little cohort variation in lung cancer mortality risk for men with a “less than high school” education level. Men with a “high school” education level demonstrate cohort patterns consistent with past research (Preston and Wang 2006). Lung cancer mortality risk increased across the early birth cohorts, peaking between 1915 and 1925, and then dropped considerably across nearly all subsequent cohorts. There is a pronounced drop in lung cancer mortality risk for the 1955 birth cohort, followed by a rise for the 1960 birth cohort back to a level consistent with the general trend prior to precipitous drop in 1955. Both of the point estimates are statistically insignificant, however, and ignoring the pronounced drop for the 1955 cohort, the overall trend would illustrate a steady cohort-based reduction in lung cancer mortality risk from 1925 to 1960. Cohort patterns of lung cancer mortality risk between 1986 and 2002 for white men with a “greater than high school” education level are similar to the general pattern of men with a “high school” education. There are three key differences, however. First, it should be reemphasized that the education gap in lung cancer mortality risk between 1986 and 2002 is retained across all cohorts, save the 1955 birth cohort. Second, the highest lung cancer mortality risk between 1986 and 2002 for men with a “greater than high school” education level was for the 1930 birth cohort, demonstrating that men with the highest education levels lagged the men with a “high school” education in lung cancer mortality risk in terms of cohort mortality risk. Third, the drops in lung cancer mortality risk across the subsequent birth cohorts bottoms out for the 1950 birth cohort, and then stall thereafter. That is, unlike past research (Pampel and Rogers 2004; Rogers et al. 2005), we find that the continued cohort decreases in lung cancer mortality risk are not occurring for the well-educated. Nonetheless, consistent with past findings (Preston and Wang 2006), the overall trend of white men’s lung cancer mortality risk between 1986 and 2002 was highest for the birth cohorts between 1915 and 1925, with a steady reduction across all subsequent birth cohorts in the 1950s.

The reductions, however, have slowed considerably in the latest birth cohorts, and have possibly increased across cohorts for those men with a “less than high school” education level. Taken together then, as is the case with all-cause and heart disease mortality risk, the education gap in lung cancer mortality risk grew between 1986 and 2002, and was driven by cohort differences across this time.

Results from HAPC-CCREM of educational differences in U.S. non-Hispanic white men’s adult mortality risk from non-lung cancer are displayed in Table 7. In order for the non-lung cancer HAPC-CCREMs to converge for both the “high school” and “greater than high school” samples, we had to constrain each cohort residual covariance to equal the constrained cohort residual covariance produced in the all-education non-lung cancer HAPC-CCREM (.300). As such, these two samples experienced significant cohort declines in non-lung cancer mortality risk between 1986 and 2002, but men with a “less than high school” education level demonstrated no significant cohort reductions (covariance parameter of .015 with a standard error of .011).

[Table 7 About Here]

Unlike the results for all-cause, heart disease, and lung cancer mortality risk between 1986 and 2002, when we observe the cohort changes in Figure 6 we find no evidence of cohort-based educational differences in non-lung cancer mortality risk for white men in the United States before the 1940 birth cohort. Thereafter, however, we observe three distinct trends for each level of educational attainment. First, for non-Hispanic white men with a “less than high school” education level, insignificant cohort changes to non-lung cancer mortality risk continues across all recent cohorts. Second, white men born after 1935 and with a “high school” education level experienced a steady cohort-based reduction in non-lung cancer mortality risk between

1986 and 2002, dropping significantly below the risk for men with a “less than high school” education level. And, lastly, non-Hispanic white men born after 1935 with a “greater than high school” education level also saw a slow cohort-based reduction in non-lung cancer mortality risk until birth cohort 1950, but then produced a rapid reduction in mortality risk across cohorts 1955 and 1960. As a result of these most recent cohort trends, we observe that the educational divide in non-cancer mortality risk for non-Hispanic white men grew between 1986 and 2002.

Finally, results from HAPC-CCREM of educational differences in U.S. non-Hispanic white men’s adult mortality risk from all residual causes of death are displayed in Table 8. In order for residual causes HAPC-CCREMs to converge for both the “high school” and “greater than high school” samples, we had to constrain each cohort residual covariance to equal the cohort residual covariance produced in the all-education non-lung cancer HAPC-CCREM (.121). As such, these two samples experienced significant cohort declines in non-lung cancer mortality risk between 1986 and 2002, but men with a “less than high school” education level demonstrated no significant cohort reductions (covariance parameter of .018 with a standard error of .020).

[Table 8 About Here]

Many of the deaths included in this residual category stem from stroke, chronic obstructive pulmonary disease (COPD), diabetes mellitus, accidents, Alzheimer’s disease, and septicemia. This is evident for three reasons. First, stroke, chronic lower respiratory diseases, diabetes, accidents, and Alzheimer’s are, currently and respectively, the third through seventh leading causes of death in the United States (NCHS 2009). Secondly, the estimated age patterns of deaths stemming from residual causes suggest that these causes of death are highly age-related (see the bottom right panel in Figure 5), as is the case with stoke, COPD, accidents, Alzheimer’s

and septicemia. Lastly, the estimated cohort patterns of these residual deaths demonstrate that, regardless of educational attainment, these causes of deaths have been on the rise across recent cohorts. This latter point is consistent with recent research that has shown reductions in stroke-related mortality are slowing or stalling (Jemal et al. 2008) and prevalence of COPD, Alzheimer's, and septicemia-related mortality is rising (Jemal et al. 2008; NCHS 2009).

Across all cohorts, as we see in the bottom right panel in Figure 6, the educational gap in residual-cause mortality risk is retained between 1986 and 2002. For all three educational levels, a similar cohort-based trend is observed. Across the 1905 and 1935 birth cohorts we estimate a small but steady reduction in residual-cause adult mortality risk for non-Hispanic white men. The rates of reduction are quite similar across educational attainment, however, so there is no change to the educational gap in mortality risk. Across the 1940 and 1950 birth cohorts, the trend is reversed, as all three educational groups experience an increase in residual-cause mortality risk. Collectively, then, the cohort patterns of residual-cause mortality risk for non-Hispanic white men between 1986 and 2002 only minimally affect the overall educational disparities in U.S. adult mortality risk. Therefore, the bulk of the overall trend, in which we see, on the one hand, rapid cohort reductions in mortality risk for all men with at least a "high school" education level and, on the other hand, a stalling of cohort reductions in mortality risk for men with a "less than high school" education level after 1935, is being driven chiefly by reductions in heart disease, lung cancer, and non-lung cancer related mortality risk [5].

Discussion

Jones (1956) was early to note that "the physiological age of each new generation is remaining more youthful at the same chronological age" (281). Finch and Crimmins (2004) greatly expanded this observation to empirically demonstrate that cohorts indeed have disparate "morbidity phenotypes." They understood that cohorts' different exposures to poor health,

infections, and bouts of inflammation would, in turn, lead to disparate morbidity and mortality risk at older ages. Conversely, it is also the case that cohorts' different exposures to advances in health-enhancing and/or health-protecting knowledge, practices, and technology would have disparate health outcomes across the life course as well. Those cohorts that first experienced reductions in poor health and poor childhood environments would be the first to experience mortality reductions later in life.

In this paper, we argued that education has played an increasingly important role in this process. As the United States population experienced the epidemiologic transition during the early- and mid-twentieth century, the disease patterns and causes of deaths shifted from infectious and communicable diseases that largely affected the youth, to chronic and degenerative diseases that overwhelmingly affected the aged (Omran 1971; Olshansky and Ault 1986). Research has shown that beyond the immediate effects of these changing disease patterns, the transition has slowly unrolled enduring cohort effects of chronic disease susceptibility at older ages (Costa 200, 2002; Finch and Crimmins 2004; Fogel 2004, 2005; Fogel and Costa 1997; Manton et al. 1997). That is, cohorts born in the later stages and after the epidemiologic transition would endure fewer and less harsh "insults" as they aged, thus being increasingly comprised of a more robust cohort morbidity phenotype. As the effects of early childhood environment on subsequent mortality risk lessened across U.S. cohorts, we believe insults during adulthood and disparate access to health-related knowledge and resources have increased in importance.

Our results provide strong evidence consistent with this theory, and support the contention that analyses of temporal changes to U.S. adult mortality risk should employ a cohort perspective to better understand the processes behind the changes. Specifically, our investigation yielded the following four findings. First, we produced empirical evidence consistent with past findings that recent temporal changes to U.S. adult all-cause and specific-cause mortality risk

were driven entirely by cohort processes (Yang 2008). Second, we built off these findings to demonstrate, for the first time, that these cohort changes to U.S. adult mortality risk were conditioned by educational attainment. That is, we showed that men with a "less than high school" education did not experience the same reductions in all-cause and specific-cause mortality risk between 1986 and 2002 as those men with at least a high school education. Third, we showed that this was not only the case with all-cause mortality risk, but that disparate cohort reductions by educational attainment took place for heart disease, lung cancer, and other cancers as well. Fourth, taken together, we demonstrated that the education gap in non-Hispanic white men's U.S. adult mortality risk is growing across birth cohorts. As such, we found evidence supporting our two hypotheses. That is, first, the education gap in U.S. adult mortality risk for non-Hispanic white men is widening, and, second, it is widening across cohorts, not periods.

With this said, our analyses in this paper are limited in several ways. First, we recognize that using only non-Hispanic white men to analyze temporal trends in U.S. adult mortality risk overlooks the vastly different cohort histories experienced by women and other race/ethnic groups in the United States. As admitted early in this paper, however, our limiting the analyses to white males was not so much a choice, but a statistical necessity. To ensure both ample cell sizes and numbers of death necessary for model convergence, we limited the current study to non-Hispanic white men. Subsequent use of the soon-to-be-released expanded NHIS-LMF will replicate these analyses for other sex-race/ethnicity subpopulations. Second, the NHIS-LMF, while uniquely designed for this study, is limited to a rather recent and short period of time. While the lack of demonstrated period effects could be influenced by this data structure, we are encouraged by the similarities between our present results and Yang's (2008) results that spanned the time period between 1960 and 1999. Third, due to small cell sizes for certain combinations of education levels and specific causes of death, we were forced to measure educational attainment with only three levels. It was our hope to separate the "greater than high

school" education group into a "some college" group and a "bachelor degree or higher" group, thereby allowing us to measure the effect of accreditation at the college level.

Despite its limitations, the present provides strong findings about the nature of temporal changes to educational differences in U.S. adult mortality risk. Consistent with both a cohort perspective of mortality change and a fundamental cause framework for understanding education's effects on adult mortality risk, we found that cohort processes are driving educational disparities in U.S. adult mortality risk. Our next steps are to build off the current results to analyze the temporal changes to educational differences in U.S. adult mortality risk by sex and race/ethnicity, build in individual-level data, and consider greater variations in societal contexts of historical changes in mortality risk. While period effects might be worth considering during certain historical times, we conclude by emphasizing the need for researchers to both integrate a cohort perspective into questions of historical shifts in adult mortality risk, and to recognize the increasing importance that education will play in shaping those risks.

Tables and Figures

Table 1: Descriptive Statistics of non-Hispanic White Male NHIS-LMF 1986-2002 Samples

	Mean	St. Dev.	Min	Max
Person-level Sample (N=319,574)*				
Age	48.14	15.29	25	84
Year	1992.48	4.08	1986	2000
Birth Year	1944.35	15.70	1901	1975
Pr. Less than High School	.17	.38	0	1
Pr. High School Graduate	.35	.48	0	1
Pr. Greater than High School	.48	.50	0	1
Deceased	.14	.34	0	1
Person-period Sample (N=2,458,826)**				
Age	57.21	12.57	40	100
Year	1996.53	4.08	1986	2002
Birth Year	1939.32	13.19	1901	1962
Pr. Less than High School	.18	.39	0	1
Pr. High School Graduate	.35	.48	0	1
Pr. Greater than High School	.47	.50	0	1
Deceased	.02	.13	0	1
Collapsed APC-Education Samples***				
< High School Sample (Cells=87)				
5-year Age Block	(60) 7.36	2.65	3	14
5-year Period Block	(1995) 1.22	.93	0	3
5-Year Cohort Block	(1930) 8.99	2.80	3	15
Cell Count Deceased	247.88	225.47	1	800
Cell Count N	7382.96	3166.10	76	1456
High School Sample (Cells=87)				
5-year Age Block	(55) 5.99	2.48	3	14
5-year Period Block	(1995) 1.07	.89	0	3
5-Year Cohort Block	(1940) 7.46	2.65	3	15
Cell Count Deceased	183.06	146.39	1	680
Cell Count N	18242.85	10153.56	20	44258
> High School Sample (Cells=87)				
5-year Age Block	(50) 5.60	2.33	3	14
5-year Period Block	(1995) 1.02	.87	0	3
5-Year Cohort Block	(1940) 7.03	2.44	3	15
Cell Count Deceased	157.41	114.06	1	593
Cell Count N	29092.23	17049.62	30	62111

* Aged 25-84 at time of survey

** Aged 40-104 at any point during longitudinal mortality follow-up

*** Cell averages are weighted by proportion and cell frequencies

Table 2: Goodness-of-Fit Statistics for GLM Models

	A	AP	AC	APC
Deviance	1304.9	1246.8	1027.8	939.4
AIC	1336.9	1284.8	1089.8	1007.4
BIC	1380.4	1336.5	1174.0	1099.8
df	16	19	31	34

Table 3. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult Mortality Rates, by Cause

	All Causes	Heart Disease	Lung Cancer	Other Cancer	Residual Causes
Fixed Effects					
Age					
40-44	-1.433	-1.654	-2.855	-2.219	-1.231
	(.067)	(.101)	(.192)	(.139)	(.098)
45-49	-1.190	-1.196	-1.906	-1.650	-1.184
	(0.057)	(.085)	(.143)	(.115)	(.088)
50-54	-.899	-.837	-1.309	-1.220	-.998
	(.047)	(.072)	(.114)	(.095)	(.077)
55-59	-.568	-.592	-.685	-.707	-.687
	(.037)	(.059)	(.086)	(.075)	(.063)
60-64	-.295	-.281	-.253	-.334	-.455
	(.025)	(.043)	(.061)	(.053)	(.046)
65-69	0.000	0.000	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
70-74	.269	.333	.212	.256	.357
	(.022)	(.037)	(.055)	(.047)	(.038)
75-79	.539	.615	.353	.506	.759
	(.030)	(.046)	(.075)	(.064)	(.050)
80-84	.806	.953	.325	.680	1.135
	(.040)	(.056)	(0.107)	(.085)	(.064)
85-89	1.168	1.281	.487	1.014	1.627
	(.056)	(.078)	(.186)	(.124)	(.085)
90-94	1.416	1.624	.407	1.118	1.941
	(.129)	(.205)	(.758)	(.365)	(.197)
95-99	1.449	1.691	.037	1.409	2.019
	(1.166)	(1.995)	(9.728)	(3.225)	(1.771)
Random Effects					
Cohort					
1960-1964	-.772	-.682	-.348	-1.022	-.350
	(.186)	(.227)	(.311)	(.293)	(.150)
1955-1959	-.817	-.819	-.814	-.546	-.469
	(.171)	(.188)	(.215)	(.198)	(.127)
1950-1954	-.565	-.737	-.545	-.252	-.192
	(.168)	(.181)	(.177)	(.182)	(.119)
1945-1949	-.502	-.513	-.375	-.212	-.277
	(.166)	(.177)	(.161)	(.174)	(.114)
1940-1944	-.353	-.350	-.088	-.076	-.279
	(.164)	(.175)	(.152)	(.169)	(.111)
1935-1939	-.164	-.063	.101	-.007	-.251
	(.163)	(.174)	(.148)	(.167)	(.108)
1930-1934	.018	-.001	.277	.092	-.040
	(.163)	(.174)	(.147)	(.166)	(.107)
1925-1929	.221	.192	.442	.244	.101
	(.163)	(.174)	(.147)	(.167)	(.108)
1920-1924	.346	.337	.449	.280	.212
	(.164)	(.174)	(.151)	(.170)	(.110)
1915-1919	.544	.568	.409	.376	.410
	(.165)	(.176)	(.160)	(.175)	(.115)

(Table 3, continued)

	All Causes	Heart Disease	Lung Cancer	Other Cancer	Residual Causes
Cohort (cont.)					
1910-1914	.676	.757	.303	.511	.446
	(.168)	(.179)	(.181)	(.186)	(.123)
1905-1909	.768	.845	.194	.478	.506
	(.175)	(.193)	(.262)	(.223)	(.145)
1900-1904	.599	.466	-.004	.134	.183
	(.315)	(.423)	(.436)	(.479)	(.298)
Period					
2000-2004	.120	-3E-4	.003	.068	.167
	(.054)	(.006)	(.034)	(.043)	(.073)
1995-1999	.038	.002	-.040	.016	.037
	(.053)	(.006)	(.031)	(.039)	(.071)
1990-1994	-.047	-.002	.042	-.067	-.063
	(.053)	(.006)	(.034)	(.041)	(.072)
1985-1989	-.111	-4E-4	-.005	-.018	-.140
	(.058)	(.006)	(.041)	(.054)	(.083)
Intercept	-3.880	-5.030	-5.940	-5.413	-4.935
Covariance Parameters					
Cohort	.330	.356	.203	*.300	.121
	(.148)	(.164)	(.127)	---	(.076)
Period	.011	3.6E-5	*.002	.004	.019
	(.009)	(3E-4)	---	(.005)	(.018)
Model Fit					
-2LPL	-32.90	2.85	88.98	53.40	1.36

Note: Numbers in parantheses are standard errors

* Value is Constrained

Table 4. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult All-cause Mortality Rates, by Educational Attainment

	< HS	HS	> HS
Fixed Effects			
Age			
40-44	-1.817	-1.374	-1.640
	(.116)	(.103)	(.105)
45-49	-1.396	-1.134	-1.395
	(.097)	(.088)	(.091)
50-54	-1.090	-.832	-1.061
	(.078)	(.073)	(.079)
55-59	-.618	-.588	-.667
	(.057)	(.057)	(.065)
60-64	-.262	-.312	-.388
	(.037)	(.041)	(.047)
65-69	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
70-74	.276	.359	.242
	(.028)	(.038)	(.044)
75-79	.603	.601	.567
	(.036)	(.051)	(.061)
80-84	.849	.916	.921
	(.045)	(.067)	(.081)
85-89	1.231	1.246	1.374
	(.058)	(.100)	(.120)
90-94	1.466	1.691	1.599
	(.116)	(.284)	(.322)
95-99	1.626	1.715	1.603
	(.874)	(3.837)	(3.1945)
Random Effects			
Cohort			
1960-1964	-.014	-.616	-.783
	(.149)	(.186)	(.208)
1955-1959	-.173	-.731	-.675
	(.113)	(.160)	(.170)
1950-1954	-.149	-.398	-.376
	(.098)	(.152)	(.161)
1945-1949	-.148	-.335	-.354
	(.086)	(.147)	(.156)
1940-1944	-.180	-.228	-.280
	(.076)	(.143)	(.153)
1935-1939	-.180	-.071	-.106
	(.070)	(.141)	(.152)
1930-1934	-.108	.035	.094
	(.066)	(.141)	(.151)
1925-1929	.045	.186	.224
	(.064)	(.142)	(.153)
1920-1924	.090	.290	.354
	(.065)	(.145)	(.156)
1915-1919	.166	.516	.512
	(.067)	(.149)	(.162)

(Table 4, continued)

	< HS	HS	> HS
Cohort (cont.)			
1910-1914	.255	.593	.625
	(.072)	(.159)	(.174)
1905-1909	.295	.632	.670
	(.082)	(.194)	(.215)
1900-1904	.101	.127	.095
	(.147)	(.447)	(.474)
Period			
2000-2004	.046	.121	.109
	(.030)	(.060)	(.053)
1995-1999	.028	.026	.027
	(.027)	(.057)	(.051)
1990-1994	-.044	-.018	-.081
	(.028)	(.058)	(.052)
1985-1989	-.030	-.128	-.054
	(.035)	(.070)	(.068)
Intercept	-3.408	-3.923	-4.113
Covariance Parameters			
Cohort	.036	.222	.253
	(.031)	(.120)	(.138)
Period	.002	.012	.009
	(.003)	(.013)	(.009)
Model Fit			
-2LPL	-17.49	17.28	30.98

Note: Numbers in parantheses are standard errors

Table 5. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult Heart Disease Mortality Rates, by Educational Attainment

	< HS	HS	> HS
Fixed Effects			
Age			
40-44	-1.901	-1.651	-1.965
	(.207)	(.151)	(.173)
45-49	-1.311	-1.152	-1.514
	(.166)	(.130)	(.151)
50-54	-1.074	-.744	-1.035
	(.133)	(.109)	(.134)
55-59	-.612	-.593	-.736
	(.096)	(.091)	(.114)
60-64	-.217	-.319	.388
	(.064)	(.071)	(.086)
65-69	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
70-74	.329	.399	.307
	(.047)	(.064)	(.079)
75-79	.651	.670	.605
	(.056)	(.079)	(.103)
80-84	.912	1.016	1.130
	(.065)	(.097)	(.132)
85-89	1.228	1.285	1.582
	(.082)	(.148)	(.192)
90-94	1.589	1.721	1.884
	(.179)	(.497)	(.536)
95-99	1.775	1.838	1.619
	(1.401)	(6.928)	(6.331)
Random Effects			
Cohort			
1960-1964	-.093	-.242	-1.123
	(.250)	(.227)	(.391)
1955-1959	-.212	-.673	-.699
	(.191)	(.180)	(.266)
1950-1954	-.539	-.595	-.357
	(.171)	(.167)	(.250)
1945-1949	-.100	-.422	-.304
	(.141)	(.159)	(.244)
1940-1944	-.232	-.230	-.259
	(.124)	(.154)	(.240)
1935-1939	-.097	-.030	.032
	(.113)	(.152)	(.238)
1930-1934	-.079	-.031	.031
	(.108)	(.152)	(.238)
1925-1929	-.029	.106	.272
	(.105)	(.153)	(.239)
1920-1924	.096	.212	.410
	(.105)	(.154)	(.243)
1915-1919	.227	.513	.539
	(.107)	(.158)	(.251)

(Table 5, continued)

	< HS	HS	> HS
Cohort (cont.)			
1910-1914	.385	.685	.699
	(.110)	(.171)	(.268)
1905-1909	.477	.655	.697
	(.123)	(.241)	(.337)
1900-1904	.197	.052	.062
	(.236)	(.464)	(.735)
Period			
2000-2004	-6.3E-4	-.001	.008
	(.006)	(.006)	(.031)
1995-1999	1.6E-4	.001	.021
	(.006)	(.006)	(.028)
1990-1994	5.1E-4	4E-4	-.033
	(.006)	(.006)	(.032)
1985-1989	-4E-5	-2.6E-4	.005
	(.006)	(.006)	(.037)
Intercept	-4.539	-5.017	-5.332
Covariance Parameters			
Cohort	.093	.223	*.600
	(.062)	(.122)	---
Period	*3.6E-5	*3.6E-5	.001
	---	---	(.003)
Model Fit			
-2LPL	18.77	79.50	96.08

Note: Numbers in parantheses are standard errors

* Constrained Value

Table 6. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult Lung Cancer Mortality Rates, by Educational Attainment

	< HS	HS	> HS
Fixed Effects			
Age			
40-44	-3.036	-2.654	-3.837
	(.287)	(.255)	(.327)
45-49	-2.313	-1.706	-2.408
	(.241)	(.200)	(.228)
50-54	-1.579	-1.266	-1.484
	(.171)	(.166)	(.190)
55-59	-.682	-.700	-.819
	(.116)	(.129)	(.158)
60-64	-.201	-.306	-.321
	(.083)	(.096)	(.119)
65-69	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
70-74	.231	.129	.314
	(.063)	(.094)	(.119)
75-79	.312	.313	.534
	(.077)	(.123)	(.162)
80-84	.224	.318	.559
	(.099)	(.175)	(.243)
85-89	.333	.561	.562
	(.156)	(.337)	(.502)
90-94	.074	.440	.748
	(.637)	(1.840)	(1.968)
Random Effects			
Cohort			
1960-1964	.021	-.378	-.234
	(.133)	(.348)	(.410)
1955-1959	-.017	-.922	-.311
	(.129)	(.255)	(.292)
1950-1954	-.048	-.306	-.337
	(.124)	(.204)	(.223)
1945-1949	-.112	-.172	-.167
	(.115)	(.185)	(.196)
1940-1944	.037	-.086	-.055
	(.097)	(.172)	(.185)
1935-1939	.003	.119	.053
	(.084)	(.166)	(.180)
1930-1934	-.120	.298	.373
	(.076)	(.164)	(.177)
1925-1929	.154	.379	.285
	(.068)	(.166)	(.182)
1920-1924	.166	.351	.236
	(.069)	(.171)	(.193)
1915-1919	.059	.400	.047
	(.074)	(.186)	(.226)

(Table 6, continued)

	< HS	HS	> HS
Cohort (cont.)			
1910-1914	-.068	.265	.038
	(.088)	(.240)	(.296)
1905-1909	-.072	.058	.073
	(.114)	(.375)	(.399)
1900-1904	-.002	-.006	---
	(.133)	(.442)	---
Period			
2000-2004	-.006	.003	.005
	(.020)	(.020)	(.057)
1995-1999	-.002	-.010	-.065
	(.019)	(.020)	(.054)
1990-1994	.006	.010	.055
	(.019)	(.020)	(.060)
1985-1989	.001	-.003	.005
	(.021)	(.021)	(.072)
Intercept	-5.253	-5.916	-6.258
Covariance Parameters			
Cohort	.018	.197	*.203
	(.014)	(.175)	---
Period	*4.5E-4	*4.5E-4	.006
	---	---	(.008)
Model Fit			
-2LPL	100.82	140.14	147.99

Note: Numbers in parantheses are standard errors

* Constrained Value

Table 7. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult non-Lung Cancer Mortality Rates, by Educational Attainment

	< HS	HS	> HS
Fixed Effects			
Age			
40-44	-2.616	-2.316	-2.371
	(.229)	(.194)	(.196)
45-49	-1.870	-1.649	-1.803
	(.179)	(.164)	(.166)
50-54	-1.342	-1.198	-1.387
	(.144)	(.138)	(.145)
55-59	-.625	-.830	-.818
	(.107)	(.112)	(.121)
60-64	-.318	-.255	-.520
	(.080)	(.083)	(.092)
65-69	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
70-74	.214	.493	.178
	(.060)	(.076)	(.090)
75-79	.670	.622	.471
	(.069)	(.099)	(.121)
80-84	.758	.876	.810
	(.083)	(.130)	(.162)
85-89	1.056	1.316	1.266
	(.109)	(.212)	(.255)
90-94	1.540	1.619	1.475
	(2.477)	(.797)	(.834)
Random Effects			
Cohort			
1960-1964	---	-.237	-1.225
	---	(.309)	(.389)
1955-1959	-.054	-.261	-.513
	(.114)	(.222)	(.232)
1950-1954	.068	-.133	-.160
	(.108)	(.202)	(.205)
1945-1949	.015	-.048	-.146
	(.099)	(.190)	(.194)
1940-1944	.007	-.013	.005
	(.087)	(.183)	(.187)
1935-1939	-.178	-.006	.174
	(.079)	(.180)	(.184)
1930-1934	-.064	.001	.207
	(.069)	(.180)	(.183)
1925-1929	.074	.111	.247
	(.062)	(.181)	(.187)
1920-1924	-.005	.123	.287
	(.061)	(.184)	(.194)
1915-1919	-.072	.180	.450
	(.064)	(.192)	(.209)

(Table 7, continued)

	< HS	HS	> HS
Cohort (cont.)			
1910-1914	.163	.180	.349
	(.071)	(.219)	(.248)
1905-1909	.043	.101	.310
	(.092)	(.333)	(.358)
1900-1904	.002	.003	.015
	(.118)	(.539)	(.540)
Period			
2000-2004	-.006	.021	.074
	(.014)	(.026)	(.056)
1995-1999	.006	-.006	.008
	(.014)	(.024)	(.052)
1990-1994	-.002	-.015	-.079
	(.014)	(.026)	(.058)
1985-1989	.001	2.2E-4	-.002
	(.015)	(.029)	(.074)
Intercept	-5.081	-5.373	-5.534
Covariance Parameters			
Cohort	.015	*.300	*.300
	(.011)	---	---
Period	2.2E-4	8.5E-4	.007
	(.001)	(.003)	(.009)
Model Fit			
-2LPL	96.01	83.38	108.95

Note: Numbers in parantheses are standard errors

* Constrained Value

Table 8. HAPC-CCREM Estimates for U.S. non-Hispanic White Male Adult Residual Causes Mortality Rates, by Educational Attainment

	< HS	HS	> HS
Fixed Effects			
Age			
40-44	-1.413	-1.265	-1.488
	(.125)	(.140)	(.140)
45-49	-1.185	-1.259	-1.402
	(.120)	(.128)	(.129)
50-54	-1.023	-1.025	-1.204
	(.110)	(.113)	(.119)
55-59	-.803	-.719	-.797
	(.092)	(.094)	(.106)
60-64	-.427	-.540	-.516
	(.066)	(.074)	(.083)
65-69	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)
70-74	.346	.475	.355
	(.047)	(.064)	(.076)
75-79	.740	.883	.884
	(.056)	(.081)	(.096)
80-84	1.115	1.348	1.270
	(.067)	(.101)	(.122)
85-89	1.628	1.790	1.865
	(.083)	(.146)	(.173)
90-94	1.833	2.433	2.180
	(.176)	(.409)	(.495)
95-99	1.890	2.631	2.157
	(1.389)	(4.934)	(5.086)
Random Effects			
Cohort			
1960-1964	.039	-.328	-.087
	(.121)	(.183)	(.184)
1955-1959	-.052	-.340	-.240
	(.103)	(.148)	(.146)
1950-1954	.069	.066	-.028
	(.095)	(.136)	(.133)
1945-1949	-.057	-.055	-.107
	(.090)	(.129)	(.127)
1940-1944	-.133	-.125	-.191
	(.082)	(.123)	(.125)
1935-1939	-.185	-.110	-.282
	(.075)	(.120)	(.125)
1930-1934	-.110	-.048	-.008
	(.066)	(.119)	(.124)
1925-1929	-.016	.026	.022
	(.059)	(.121)	(.126)
1920-1924	.006	.111	.124
	(.058)	(.125)	(.131)
1915-1919	.131	.296	.248
	(.060)	(.133)	(.141)

(Table 8, continued)

Cohort (cont.)			
1910-1914	.126	.242	.303
	(.067)	(.152)	(.164)
1905-1909	.169	.235	.229
	(.084)	(.213)	(.230)
1900-1904	.012	.030	.018
	(.125)	(.342)	(.342)
Period			
2000-2004	.148	.143	.087
	(.071)	(.067)	(.049)
1995-1999	.056	-.012	.001
	(.069)	(.064)	(.046)
1990-1994	-.117	-.028	-.058
	(.070)	(.066)	(.050)
1985-1989	-.088	-.103	-.31
	(.081)	(.087)	(.068)
Intercept	-4.497	-4.945	-5.105
Covariance Parameters			
Cohort	.018	*.121	*.121
	(.020)	---	---
Period	.017	.013	.006
	(.017)	(.015)	(.007)
Model Fit			
-2LPL	29.59	55.55	73.41

Note: Numbers in parantheses are standard errors

* Constrained Value

Figure 1: Education of non-Hispanic White Male Birth Cohorts in NHIS-LMF, 1986-2002

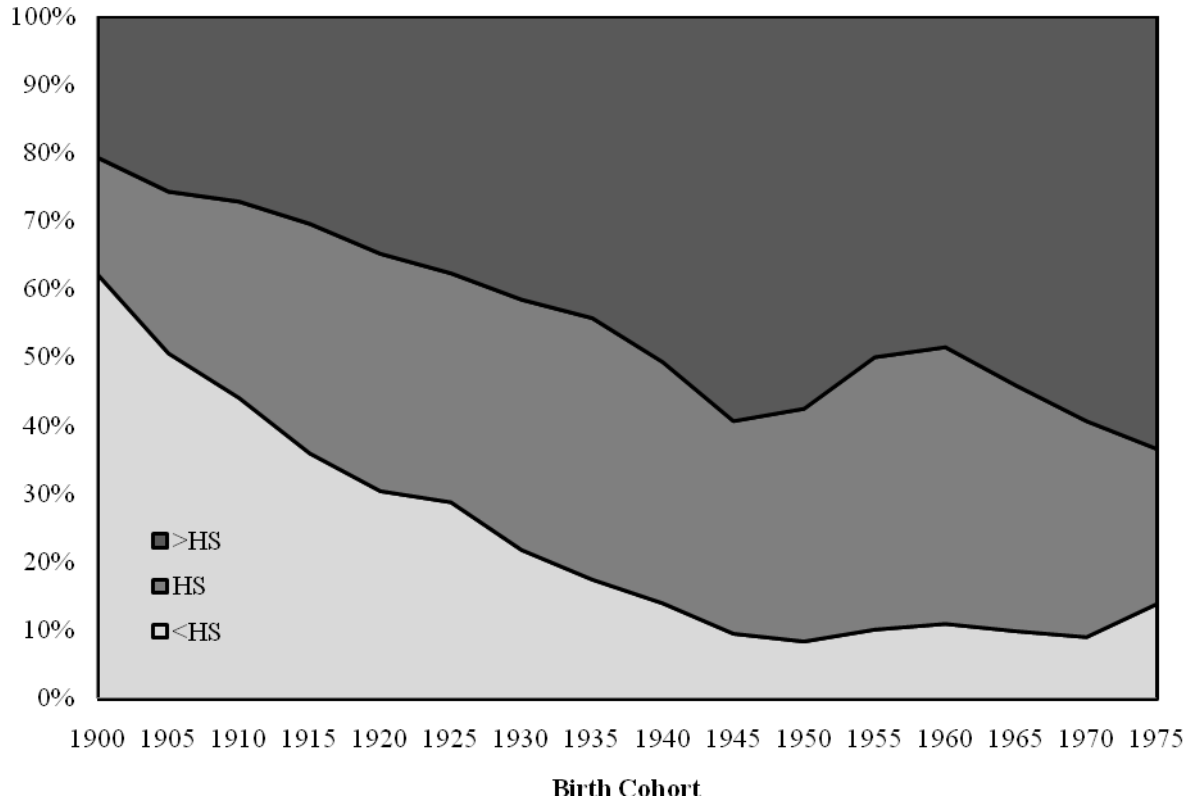


Figure 2: Age Effects of non-Hispanic White Men's U.S. All-cause Mortality Risk, 1986-2002, by Educational Attainment

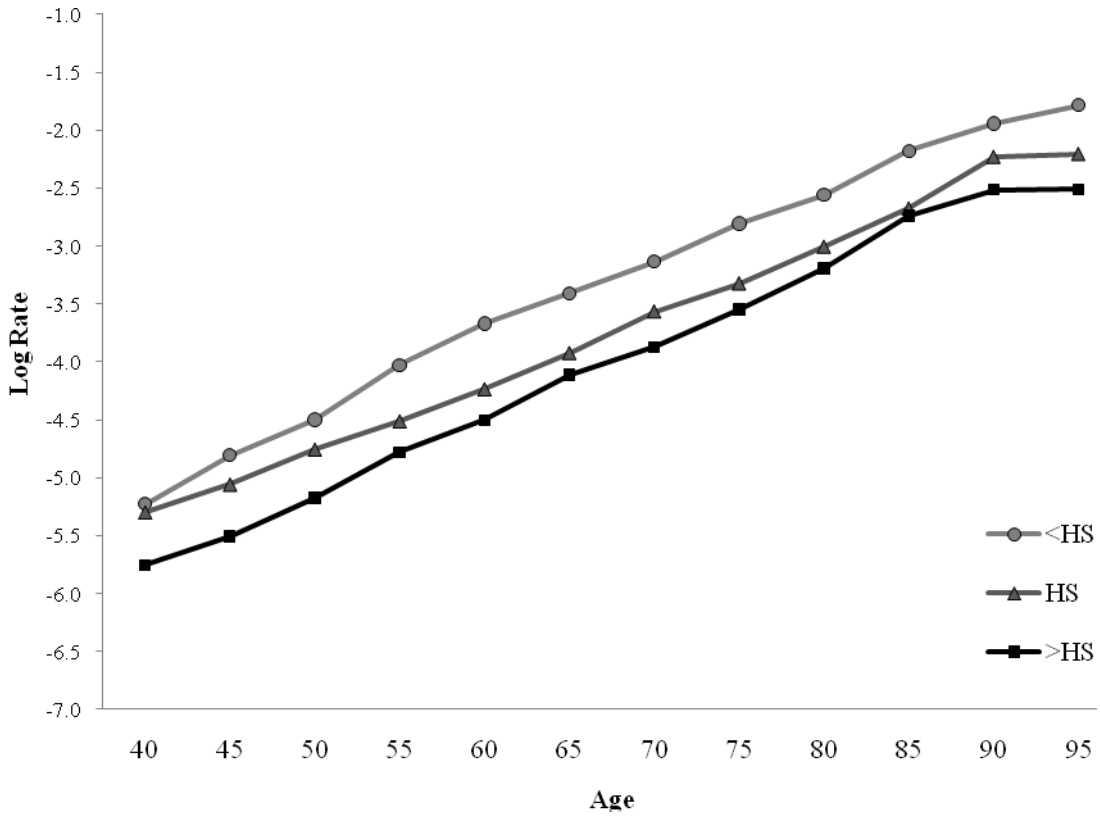


Figure 3: Cohort Effects of non-Hispanic White Men's U.S. All-cause Mortality Risk, 1986-2002, by Educational Attainment

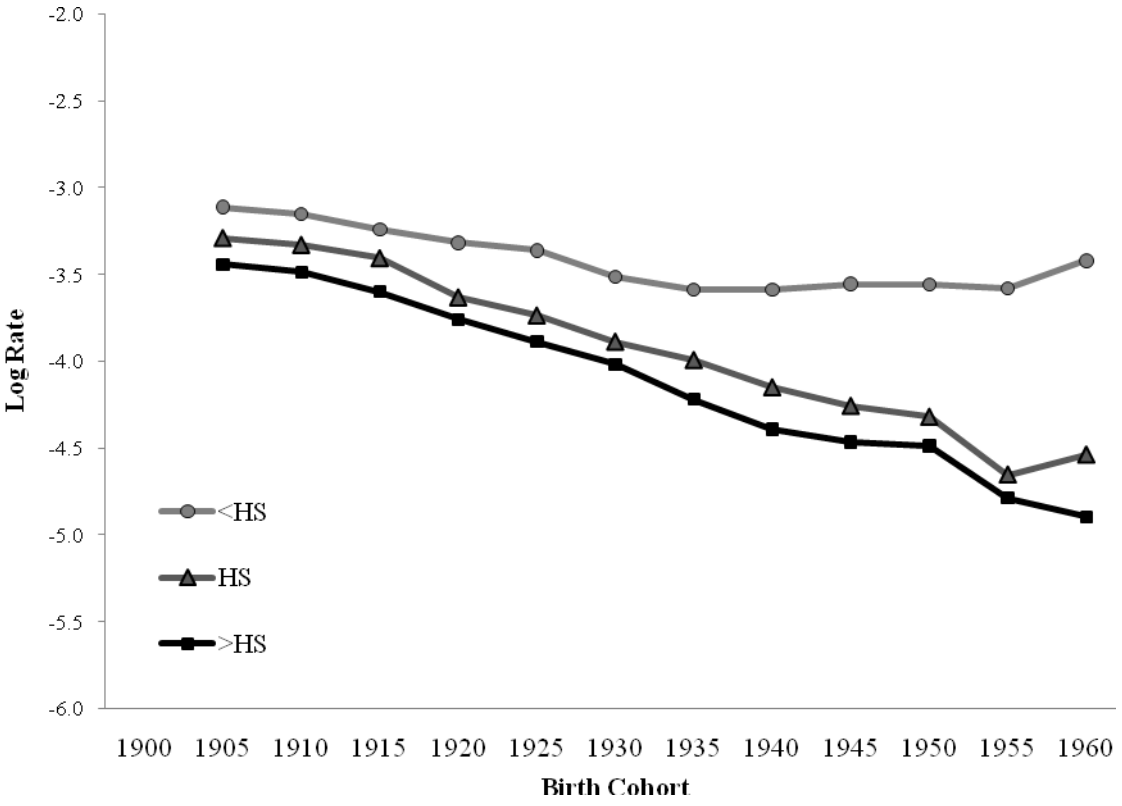


Figure 4: Period Effects of non-Hispanic White Men's U.S. All-cause Mortality Risk, 1986-2002, by Educational Attainment

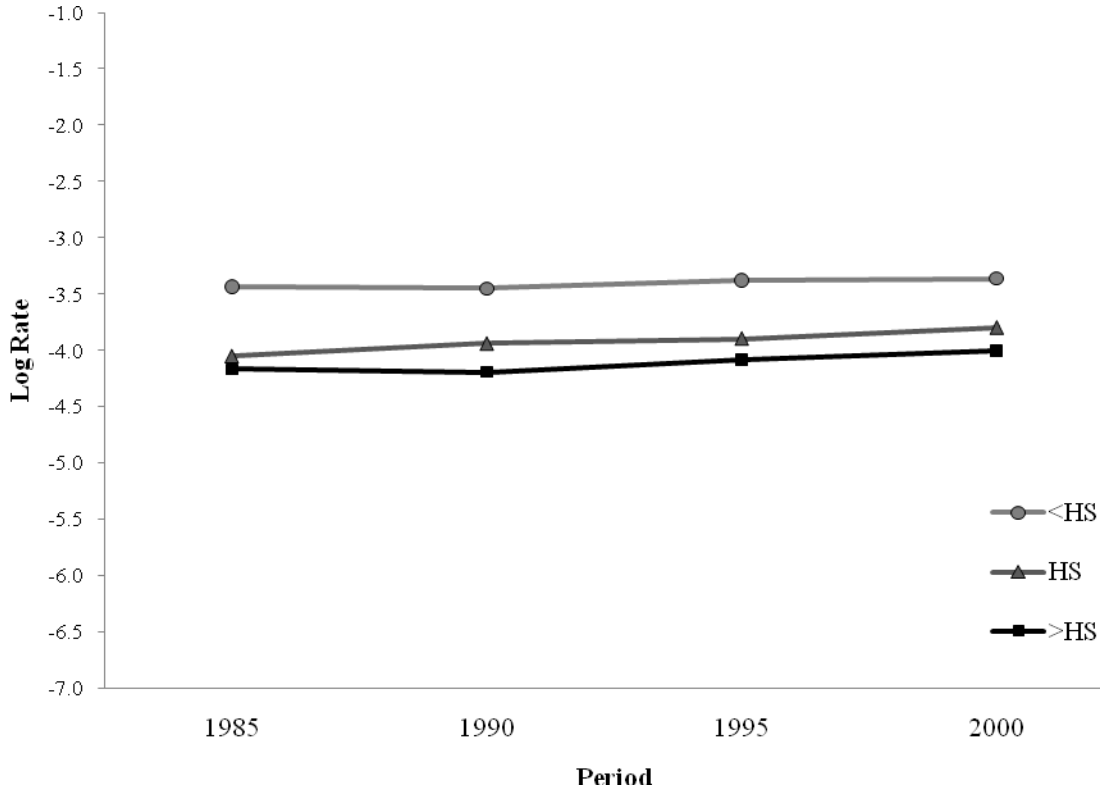


Figure 5: Age Effects of non-Hispanic White Men's Specific-cause Mortality Risk, 1986-2002, by Educational Attainment

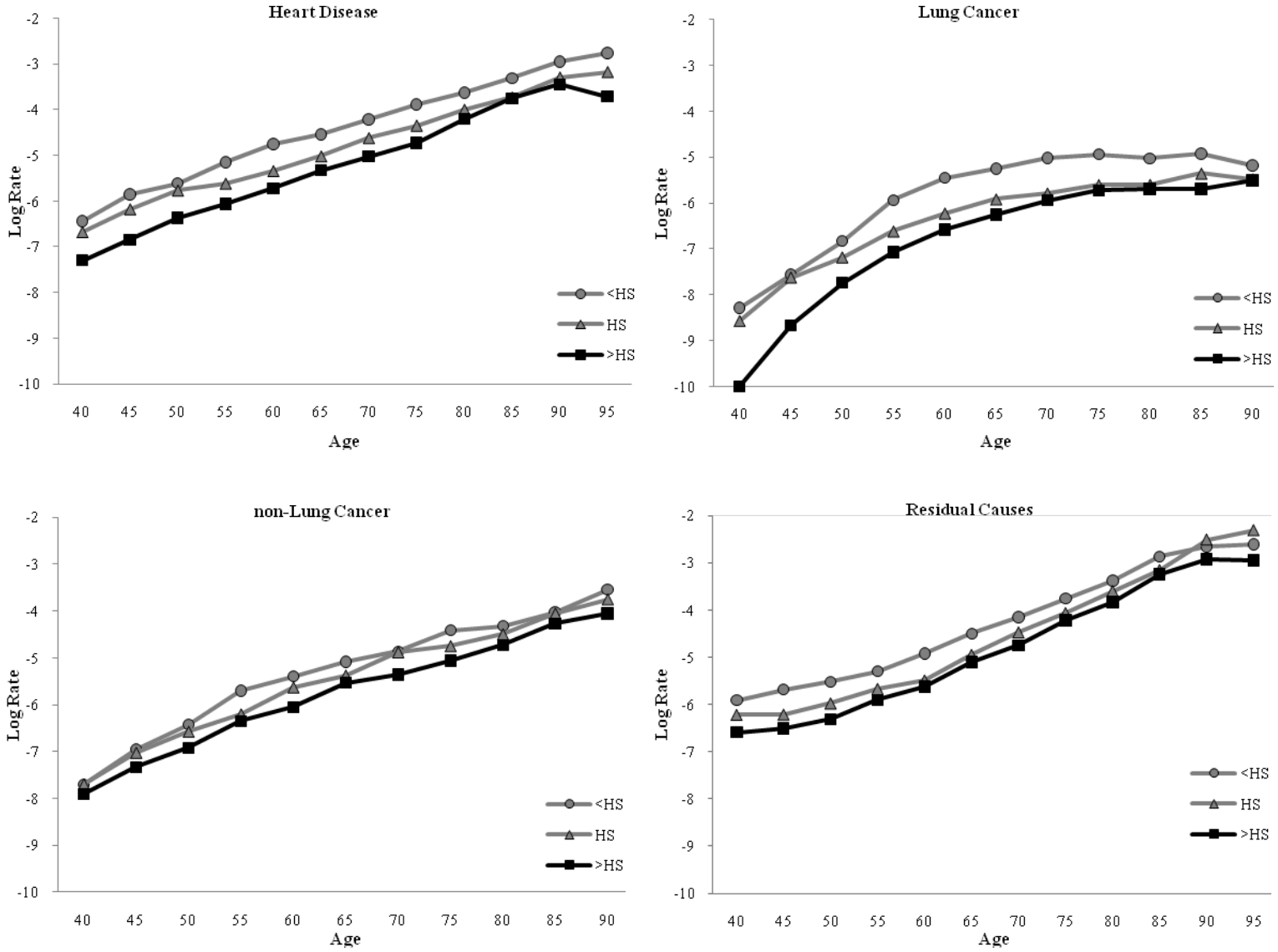
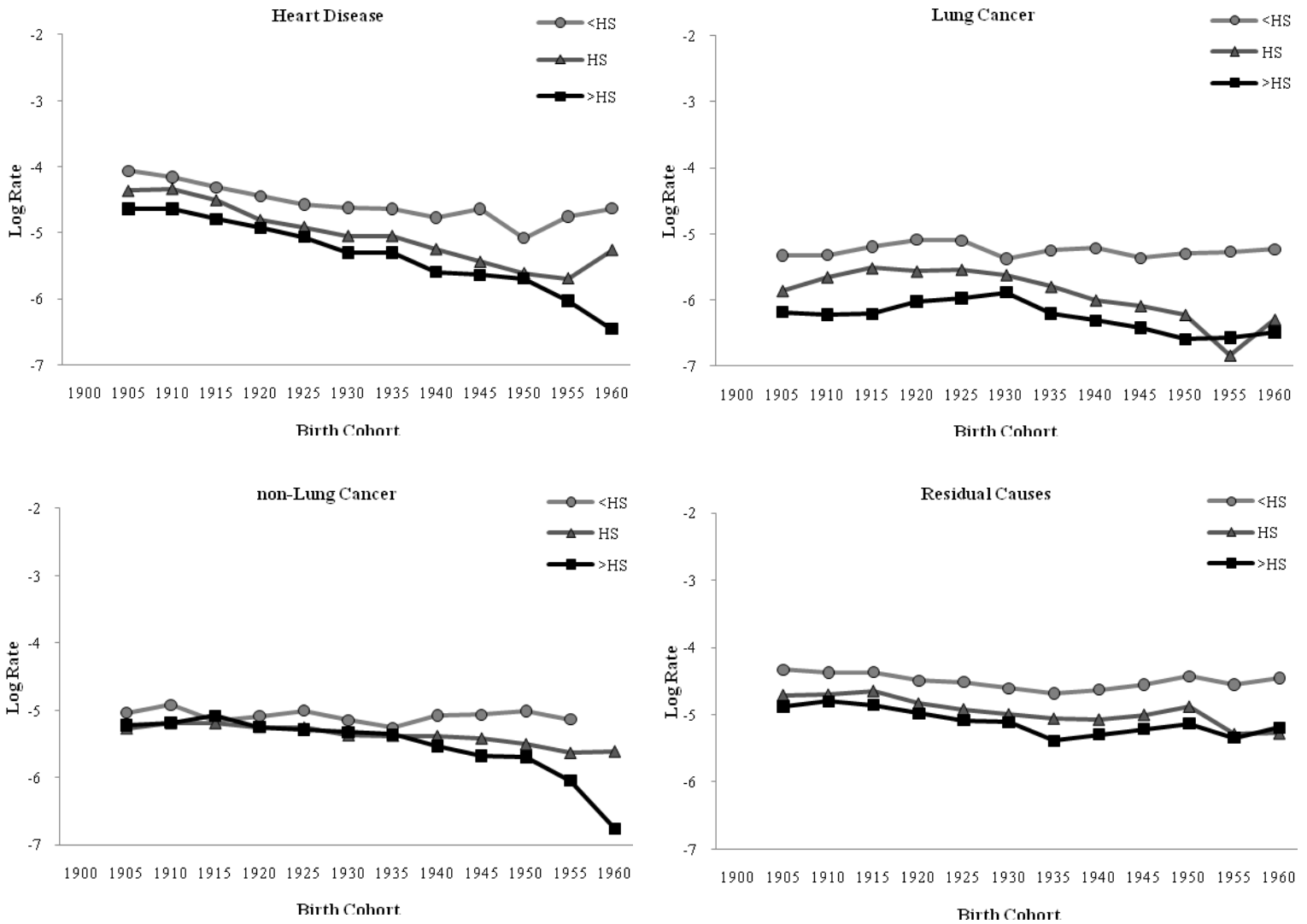


Figure 6: Cohort Effects of non-Hispanic White Men's Specific-cause Mortality Risk, 1986-2002, by Educational Attainment



Footnotes

1. The most recent version of the NHIS-LMF, 1986-2006, is set to be released in March 2010. Once available, these most recent data will be used to shore up the analyses used in this paper, providing both greater exposure of cohorts and periods and also more cases and more occasions of death.

2. Unfortunately, this is not always the case for all sex-race analyses of specific-cause mortality risk by educational attainment. Indeed, the sole reason this paper examines mortality risk only for non-Hispanic white men is that we have sufficient cell counts and enough deaths at each combination of age-period-cohort to generate reliable estimates. We hope that the updated NHIS-LMF 1986-2006 data resolves this limitation.

3. We suspect that the matching scores between NHIS respondents and death records at the NDI are much lower at older ages than at younger ages. Consequently, we believe that the mortality rates at these advanced ages are less reliable not only because of age misreports, but also because deaths may not have been accurately attributed to NHIS respondents. That is, it is possible that certain identifiers such as social security numbers, middle name, and birth information are difficult to obtain for those who died at very old ages. The likelihood of deriving this information from a spouse, next of kin, or other credible source is presumed to be much lower for the deceased at such advanced ages. Such unidentified deaths were most likely to have been dropped from the sample, removing important contributions from both the numerator and denominator of the mortality rate. Also, while less likely, it is still possible that a NHIS respondent who died in the mortality follow-up was not identified as such and kept in the sample as a living respondent. This would incorrectly keep the respondent in the denominator of the hazard, but would also not count him/her in the numerator. As such, we omit from the final sample any respondent aged over 104. Age heaping and other forms of age misreports have been found to be common in older adult age reports (Newell 1971) and also to have quite detrimental effects on old-age estimates of mortality risk (Preston et al. 1999). Also, selection bias confounds estimates and can occur for a number of reasons. First, because the NHIS-LMF is comprised only of non-institutionalized members of the population, all residents of nursing homes, hospitals, assisted living centers, or other institutions are excluded from the sampling frame. Sample biases could exist if education and/or cohort membership is correlated with the likelihood of living in institutional quarters among the older population. Second, respondents of the NHIS are included in the final NHIS-LMF sample only if the NCHS determine that the respondent can be adequately matched to the NDI death records. Sample bias could exist if education is correlated with the matching process. Last, a healthy participant effect occurs when a sample is comprised of participants that tend to be healthier than nonparticipants.

4. In the instance of a model not converging, our first attempt to improve stability was to drop the oldest age and/or oldest birth cohort cells (those with the smallest cell sizes). When dropping thin data points from our models did not resolve the problem, we then constrained the period covariance parameter to be the value derived from the all-education model. Only when these two efforts did not help the model to converge did we constrain the values of the cohort covariance parameters. When this was the case, we proceeded in a trial-and-error fashion, comparing the estimated random effects cohort coefficients with the estimated fixed effects cohort coefficients derived from the HAPC-CFEMs. These comparisons are available upon request.

5. This is not to say, however, that research ought not to pay attention to the individual causes making up the residual-causes category. Indeed, there have been tremendous changes to patterns

of diabetes, stroke, and other causes of death, which, we suspect, are conditioned by educational attainment.

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