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EXPLAINING THE RISE IN EDUCATIONAL GRADIENTS IN MORTALITY

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ABSTRACT

The long-standing inverse relationship between education and mortality strengthened substantially later in the 20th century. This paper examines the reasons for this increase. We show that behavioral risk factors are not of primary importance. Smoking has declined more for the better educated, but not enough to explain the trend. Obesity has risen at similar rates across education groups, and control of blood pressure and cholesterol has increased fairly uniformly as well. Rather, our results show that the mortality returns to risk factors, and conditional on risk factors, the return to education, have grown over time.

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I. Introduction

Mortality in the United States has declined by much more among the better educated than among the less educated. Between 1960 and 2000, education-related gaps in mortality grew dramatically. For example, between 1960 and 1986, education-related gaps in mortality grew 20 percent (Gregory Pappas et al. 1993). The gap widened further, and more rapidly in the decade after 1990, when life expectancy of those attending college increased an additional 1.6 years with no change among those who did not go to college, yielding a 30 percent growth in life expectancy gaps by education (Ellen Meara, Seth Richards, and David M. Cutler 2008). By 2000, college-educated 25-year olds could expect to live 7 years longer than their peers with less schooling. These patterns have thrust the issue of health disparities onto the political agenda. Reducing health disparities (by race and ethnicity as well as economic status), along with improving population health, are two major components of the *Healthy People 2010* objectives (U.S. Department of Health and Human Services 2000).

Despite this policy focus, sources of the increase in these educational disparities remains poorly understood. Some studies, focusing on medical care providers, show that advantaged individuals receive better and earlier care than their less advantaged counterparts (e.g. Saif S. Rathore et al. 2000). Other analyses stress behavioral differences: the better educated are less likely to smoke, drink, and (at least among women) to be obese (David M. Cutler and Adriana Lleras-Muney 2008). Still other research suggests the possibility that high status individuals are less exposed to unalleviated stress (Michael G. Marmot 2006).

This paper analyzes the extent to which behavioral differences (smoking and obesity), and their immediate medical correlates (hypertension and high cholesterol), can explain changes

in educational mortality gradients occurring over the past three decades.[1] Smoking and obesity are natural to examine because they are the two leading behavioral causes of death in the United States. Tobacco use is responsible for about 435,000 premature deaths annually (Ali H. Mokdad et al. 2004) and obesity for between 100,000 and 400,000 early deaths per year (Katherine M. Flegal et al. 2005; Walter C. Willett et al. 2005). Since the obese often develop high blood pressure (hypertension) and high cholesterol (hypercholesterolemia), and management of these risk factors is itself a behavioral issue, we further examine how disease management varies across education groups. Finally, we separately consider deaths due to cardiovascular disease (CVD, mostly heart disease) and cancer, since these represent major sources of premature mortality and are responsive to changes in modifiable risk factors.

David M. Cutler, Edward L. Glaeser, and Allison B. Rosen (2009) show that behavioral risk factors play an important role in understanding changes in overall mortality trends overall. They estimated the contribution of demographics and risk factors (smoking, drinking, obesity, and blood pressure) to 10-year mortality risk during the last three decades of the 20th century and provide evidence that decreases in smoking and better control of hypertension contributing most to the substantial reductions in age-adjusted deaths rates, while increases in obesity raised mortality risk (also see S. Jay Olshansky et al. 2005, and Susan Stewart, David M. Cutler, and Allison B. Rosen 2009). We use similar data but, instead of focusing on the entire population, assess the extent to which differential changes in risk factors explain secular increases in education-related mortality gaps.

[1] We are not the first to look at these issues. Evelyn M. Kitagawa and Philip M. Hauser (1968) identified educational differentials in age-specific mortality, present in 1960, and there has been substantial related recent research (e.g. Jacob J. Feldman et al. 1989; Harriet Orcutt Duleep 1989; Pappas et al., 1993; Eileen M. Crimmins and Yasuhiko Saito 2001; Kyle Steenland, Jane Henley, and Michael Thun 2002; Mitchell D. Wong et al. 2002; Charles C. Lin et al. 2003; Gopal K. Singh and Mohommad Siahpush 2006; Meara, Richards, and Cutler 2008), most finding that the educational gaps have increased over time.

Our analysis reveals three primary findings. First, education-gradients in mortality are stronger for men than women but have increased over time for both sexes. Second, despite the importance of smoking, obesity, hypertension, and cholesterol as determinants of population health, differential changes in these risk factors do not explain the widening educational gap in mortality since the 1970s. Deaths from cancer, rather than CVD, account for the bulk of this increase, and this occurs both because the share of deaths due to cancer is growing over time and because educational differentials in cancer mortality have increased. Finally, the mortality returns to risk factors, and conditional on risk factors, the return to education, have grown over time for reasons that are not yet understood. Thus, even if less educated populations were able to achieve risk factor profiles that mirrored those with more education, widening mortality differentials would likely persist.

We do not directly address why the impact of risk factors and education have increased over time, but three explanations seem likely. First, access to medical care may have become more important for detecting disease early and treating it appropriately, and the better educated have superior access to care. Second, the living environments (i.e. the exposure to environmental health risks) may have improved more over time for the better educated. Third, the management of chronic health problems may have become more sophisticated over time in ways that favor the highly educated. Our data are not adequate to test these theories, which we leave to subsequent research.

Sections II and III present the data we analyze and descriptive trends in mortality. The fourth section presents our empirical approach. The fifth section reports our mortality regressions, and the sixth section uses these to understand changes in the education-gradient in mortality. Section VII examines CVD and cancer deaths. The last section concludes.

II. Data and Key Variables

Our analysis utilizes data from various waves of the National Health and Nutrition Examination Surveys (NHANES) and multiple years of the National Health Interview Survey (NHIS). The NHANES and NHIS both provide samples that are nationally representative of the non-institutionalized U.S. population in the specified period, using stratified, multi-stage probability cluster designs. Information on subsequent mortality is obtained for some of the surveys, as described below, and is used to estimate education-related differences in death rates. The remainder of this section describes the surveys and key explanatory variables.

The National Health and Nutrition Examination Survey

Baseline information for this study was obtained from NHANES I, covering the period 1971-1975. Subsequent descriptive data are from the NHANES II (1976-1980) and NHANES III (1998-1994), and the first six years (1999-2004) of the continuous NHANES survey (hereafter referred to as NHANES IV).[2]

The NHANES is particularly useful since it includes a physical exam component providing clinical measures of weight, height, blood pressure, and cholesterol. Because we are interested in trends in mortality determinants for specified groups, rather than changes in the composition of U.S. residents, we exclude Hispanics from the analysis. We further limit the samples to whites, since the earlier NHANES does not provide adequate size to estimate mortality among non-white populations. All results are age-adjusted and separated by gender, so that trends do not reflect changes in these characteristics.

[2] See: http://www.cdc.gov/nchs/nhanes.htm for further information on the NHANES.

The absence of persons institutionalized at baseline leads to lower estimates of future mortality than would be obtained for the full population. However, Meara, Richards and Cutler (2008) demonstrate that, conditional on surviving one year, the death rates for adults initially living in the community closely resemble those reported in published statistics for the entire population. Therefore, we limit the sample to individuals surviving at least one year from the baseline interview for all mortality models estimated below.

We further restrict the sample to those aged 25-74.[3] This provides 5,942 respondents from NHANES I (1971-75), and 8,408, 4,903 and 5,143 for NHANES II, III and IV respectively. NHANES I and NHANES III include mortality follow-up surveys, in which respondents are matched to National Death Index records providing information on the timing and cause of death. For NHANES I, the follow-up survey covers a period of 10 years (1981-85). For NHANES III we utilize public use data including deaths through 1998-2000, providing 10 years of information for around half of survey respondents.[4] Relatively small sample sizes for the NHANES III follow-up reduce the precision of these estimates, so they are only used as a supplementary source of information on mortality differentials. The main data set employed during the later sample period is the NHIS, which is described further below. In addition to the analysis of total mortality, we examine deaths due to cardiovascular disease (CVD) and cancer, as defined in NHANES I using ICD-9 codes.[5]

[3] The upper age limit was 74 years old for NHANES I and II.
[4] The NHANES III public use mortality data include perturbations of data elements to protect confidentiality of respondents. These do not affect the estimates of demographic differences in death rates.
[5] The three-digit ICD-9 codes corresponding to cancer and cardiovascular disease are 140-290 and 390-460.

The National Health Interview Surveys

We supplement NHANES with data from the 1987-88, 1990-95, and 1997-2000 years of the National Health Interview Survey.[6] The NHIS data have been linked to deaths through 2002, and we compute survival for a follow-up period of a decade, or through 2002 for individuals interviewed after 1992. The advantage of using the NHIS is its large sample size (over 200,000 observations in our analysis sample), which allows us to estimate mortality functions with much greater precision than in the NHANES (particularly NHANES III). The cost is that, unlike NHANES, all information is self-reported. Therefore, the NHIS lacks the clinical markers used to evaluate the role of hypertension or cholesterol control in NHANES. Also, self-report data underestimate obesity prevalence since height is over-reported and weight is understated (Marie F. Kuczmarski, Robert J. Kuczmarski, and Matthew Najjar 2001; Elmer V. Villanueva 2001).

As with NHANES, our mortality estimates are for persons surviving at least one year, to adjust for the exclusion of the institutionalized at baseline. Similarly, we restrict the sample to non-Hispanic whites aged 25-74 at baseline, leaving a final sample size of 206,364.[7] Cause-of-death in the NHIS is classified using "U codes" that represent broad categories compiled from the underlying *International Classification of Diseases-10th Revision* codes. We use the latter to identify cancer and CVD mortality.[8]

[6] 1989 and 1996 are excluded due to a lack of information on smoking. Information on the NHIS is available at: http://www.cdc.gov/nchs/nhis.htm.
[7] The age and race/ethnicity restrictions eliminate 155,763 respondents. Another 11,454 observations were deleted due to missing information (on education, race/ethnicity, weight, height, smoking status or future mortality from the National Death Index follow-up) and 1,598 were excluded because they died within a year of the baseline interview.
[8] The ICD-10 codes indicating cancer are C00-C97, and for cardiovascular disease the codes are I00-I99.

Education

We divide the population into two broad education groups: those who completed more than 12 years of schooling (college attendees) and those with less education. These broad categories were chosen to minimize errors introduced by the known tendency to over-report the attainment of a high school degree (Paul D. Sorlie and Norman J. Johnson 1996). Secular changes in the composition of the education groups are potentially problematic, since the share of individuals attending college has grown dramatically over time: the proportion of college educated men in our sample grew from 33 to 60 percent between 1971-75 and 1999-2004; the corresponding increase for women was from 24 to 61 percent. Following Meara, Richards, and Cutler (2008), we test for the importance of such compositional changes by estimating specifications where individuals on the margin between the low and high education group are randomly reassigned so as to equalize the compositional shares across time periods.

Behavioral Risk Factors

We focus on smoking, obesity, blood pressure and cholesterol as potentially important risk factors for mortality. Current smokers as those who report smoking at the time of interview; former smokers are those who had previously smoked at least 100 cigarettes but do not smoking at the interview date.[9] In supplemental specifications we also control for the number of cigarettes smoked per day (for current smokers) and the time since last use (for former smokers), although these are measured with greater error than current/former/never status, and so they are not included in the main models.

[9] The exact wording of the questions changed in the 1992 NHIS to measure individuals who smoked on some days. There is no reason to believe this change would differentially affect smoking rates by education.

We distinguished five weight categories based on body mass index (BMI): weight in kilograms divided by height in meters squared. Height and weight are obtained from medical examinations employing standardized procedures and equipment in NHANES but from (less accurate) self-reports in the NHIS. Following national and international standards, we define "underweight", "healthy weight", "overweight", "class 1 obese", and "class 2 or class 3 obese" for BMI of: <18.5, 18.5 to <25; ≥ 25 to <30, ≥ 30 to <35 and ≥ 35 (World Health Organization 1997; National Heart, Lung and Blood Institute 1998). Persons with a BMI of 35 or more are sometimes referred to as "severely obese". Although BMI is less accurate than laboratory measures of body composition, since it does not account for variations in muscle mass or in the distribution of body fat, it is a favored method of assessing excess weight because it is simple, rapid, and inexpensive to calculate.[10]

One reason excess body weight increases mortality is because obese individuals tend to have high rates of hypertension and hypercholesterolemia (Aviva Must et al. 1999; Cutler, Glaeser, and Rosen 2009), placing them at risk of serious cardiovascular events. The development of effective medications to control high blood pressure and cholesterol may have substantially reduced the risk of death from these conditions, raising the possibility that larger mortality gradients might reflect increased success in controlling these health problems for high but not low educated individuals. We investigate this by estimating models that include controls for blood pressure and cholesterol.

Blood pressure was divided into four groups, following the recommendations of the National Heart, Lung, and Blood Institute (2004): normal (systolic blood pressure (SBP) <120 mmHG and diastolic blood pressure (DBP) <80 mmHG); pre-hypertension (120≤ SBP<140 or

[10] Some researchers prefer anthropometric measures such as waist circumference (K. M. Sönmez et al. 2003), waist-hip ratio (M. A. Dalton et al. 2003), or waist-height ratio (Brian D. Cox and Margaret J. Whichelow 1996).

80≤ DBP<90); stage 1 hypertension (140≤ SBP<160 or 90≤ DBP<100); and stage 2 hypertension (SBP≥ 160 or DBP≥ 100). Total cholesterol levels were divided into three groups, based on guidelines in the National Heart, Lung, and Blood Institute (2002): normal (<200); borderline high cholesterol (200 to <240); and high cholesterol (≥ 240). We focus on total cholesterol rather HDL and LDL because the latter were not available in NHANES I.

Other risk factors also influence mortality, but they are decidedly less important in overall deaths. The third leading behavioral cause of death is alcohol use. In comparison to smoking and obesity, alcohol use accounts for only around 22,000 deaths annually (Melonie Heron et al. 2009), and many of these deaths result from automobile accidents involving persons younger than 25, who are excluded from our analysis.

III. Descriptive Trends

5-year mortality and education

Table 1 displays 5-year age-adjusted death rates by sex and education group. Between the early 1970s and the 1990s, mortality rates fell overall, with particularly large reductions for men and the college-educated. Among less educated males, the estimated 5-year death rate declined from 6.3% in NHANES I (1971-75) to 5.8% in the NHIS (1987-96), a drop of 0.5 percentage points; this compares to a 2.0 point reduction (from 5.8% to 3.8%) for college attendees.[11] The fatality rates of non-college educated women increased 0.5 points (from 3.1 % to 3.6%) over the same period, while falling 1.0 percentage point (from 3.7% to 2.7%) among

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Burkhauser and Cawley (2008) recommend the use of Bioelectrical Impedance Analysis (BIA).

[11] The five-year mortality period begins after the initial restriction of surviving one year after the interview, to account for the exclusion of the non-institutional population from the baseline survey. Since the NHIS mortality were analyzed through 2002, 1996 is the last interview year for which we have five years of follow-up data.

college attendees.[12] Thus, survival gains were large in recent decades but mainly for the highly educated. Table 1 also illustrates that education gradients were stronger for males than for females in each time period.

It is worth noting that college-educated women have slightly higher estimated mortality rate than their peers in NHANES I (3.7% versus 3.2%) and NHANES III (3.2% versus 2.9%). Given the data required to construct 5-year mortality rates, the sample sizes are relatively small (2,200 and 700 in NHANES I and 1,500 and 950 in NHANES III) and these differences are not statistically significant. The finding that education conferred no mortality advantage to adult women in the 1970s echoes similar findings of a flat mortality gradient for females, especially for deaths due to Cancer and Cardiovascular disease, obtained in earlier analysis of the National Cancer Society cohorts from 1959-1971 (Samuel H. Preston and Irma T. Elo 1995; Steenland, Henley, and Thun 2002). The lack of sample size in the NHANES III data prompted us to use the larger NHIS samples for these years. While the small NHANES I samples preclude us from drawing firm conclusions regarding the nature of female mortality in the early 1970s, data from the NHIS unambiguously indicates that there were large education-mortality gradients for both sexes at the end of the 20th century,.

Table 2 reveals an equally important trend of rising education over time. For men, college attendance rose from 33 to 54 percent between NHANES I and the NHIS. Among women the growth was even larger: from 24 to 49 percent.[13] Such large changes make it important to ensure that results are not due to compositional changes in education groups, rather

[12] These patterns resemble recent estimates by Meara, Richards, and Cutler (2008).
[13] Corresponding secular increases in education are found in other national data sets. For instance, Current Population Survey data indicate that the fraction of white males (females) 25 and older with a high school degree or more rose from 54.0% to 84.8% (55.0% to 85.0%) between 1970 and 2000 (U.S. Census Bureau 2009; Table 222).

than differential patterns of survival conditional on education. We address this issue in section VI.

Age-adjusted risk factors

Tables 3A and 3B show how risk factors related to smoking, obesity, hypertension and hypercholesterolemia differ across education groups and over time. The results are presented separately for males and females and we compare changes from 1971-1975 (using NHANES I) to either 1987-96 (from the NHIS) or 1996-2004 (using NHANES IV).

An inverse relationship between education and current smoking exists in all periods but has increased with time, particularly for women. Current smoking fell 8 percentage points between 1971-75 and 1999-2004 for less educated males, versus 15 points among college attendees. Smoking increased by one percentage point for non-college educated females but dropped 12 points for college attendees during the same period. These results, which mirror findings previously obtained by Meara, Richards, and Cutler (2008) lend credence to the possibility that differential reductions in smoking have contributed to increasing educational gaps in mortality.

The trends in obesity are somewhat sensitive to the choice of endpoints and data sets, with larger secular growth observed for NHANES IV than the NHIS.[14] These qualifications notwithstanding, Table 3 demonstrates a rapid growth in obesity for all groups but without strong educational differences. Between 1971-75 and 1999-2004, the fraction of less educated males who were obese (class I or higher) increased 17 percentage points (from 14% to 31%) and the proportion severely obese (class II or greater) rose 8 points (from 2% to 10%). The

corresponding expansion in obesity and severe obesity for college educated men was 20 points (from 9% to 29%) and 8 points (from 1% to 9%). Among females, the 18 percentage point rise in obesity (from 11% to 29%) for college-educated women was only slightly less than the 20 point increase (from 17% to 37%) for those with less schooling, and both groups had an 11 percentage point growth in severe obesity.[15] Although obesity growth is smaller when using the NHIS rather than the NHANES IV, similar changes are again observed for the two education groups. These similarities imply that obesity trends are unlikely to explain much of the growth in education gaps in mortality that are the focus of this analysis.

The bottom portion of tables 3A and 3B document success stories in the battle to reduce risk factors for cardiovascular disease, as measured by the reductions in high blood pressure and cholesterol. Hypertension (Stage I or II) dropped 29 (18) percentage points for less educated males (females) and 26 (17) points for college attendees. Corresponding reductions in severe (Stage II) hypertension were 14 (10) for less educated males (females), and 13 (8) percentage points for college attendees. The prevalence of high cholesterol declined by between 10 and 13 percentage points for both sexes and education categories. The less educated are more likely to have high blood pressure or cholesterol at both points in time but decreases in the severe forms of these health risks tend to be slightly larger for them (except for high cholesterol among men), so that the secular changes will not be able to explain the widening in education-mortality gradients.

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[14] This occurs because the self-report data in the NHIS lead to an understatement of BMI, as discussed, and since the NHANES IV covers a later period (1999-2004 rather than 1987-96) and so picks up the substantial growth in weight occurring at the very end the 20th century.

[15] These results are consistent with previous research indicating that SES-BMI differentials are constant or narrowing over time (Qi Zhang and Youfa Wang 2004; Virginia W. Chang and Diane S. Lauderdale 2005).

IV. Empirical Approach

We examine whether trends in risk factors contribute to widening education differentials in death rates, using an approach similar to Cutler, Glaeser, and Rosen's (2009) investigation of the sources of overall gains in life expectancy. Our main empirical analysis involves three steps. First, we estimate mortality hazard rates as a function of behavioral risk factors and education, using information from a single data set (such as NHANES I or the NHIS). This equation describes the relationship between the control variables and mortality given other determinants (e.g. medical technologies) present at that point in time. The schooling coefficient indicates the educational differential for persons with given risk factors. Second, we use the parameter estimates generated in the first stage to simulate how mortality would change within an education group, given the distribution of risk factors present at a different time period. Finally, we calculate ratios of predicted death rates for less versus more educated individuals for each of the four possible combinations of population characteristics and mortality equations from combinations of the earlier (NHANES I, 1971-75) and later (NHIS 1987-2000) period. For example, the actual mortality gradient during 1971-75 is calculated using the NHANES I sample characteristics and the mortality hazard rates estimated from this same data set. Conversely, the 1987-2002 education-mortality gradient predicted if the population retained the risk characteristics of 1971-75 is obtained by applying the NHIS mortality equation to the NHANES I data. The difference between these two estimates shows the change in the gradient that is due to trends in population risk factors. Since educational and mortality trends vary dramatically between men and women, we estimate all models separately for subsamples stratified by sex.

The basic equation is a proportional hazard model of mortality risk taking the form:

$$(1) \quad mortality\left(X, college, age\right)_s = \exp\left(\beta' * X_s + \lambda * college_s + \gamma * age_s\right),$$

where the *s* subscript specifies the survey (NHANES I or the NHIS in the main models). *College* is an indicator of whether a respondent reported >12 years of education, *age* is expressed in single years between 25 and 74, and *X* contains the set of behavioral risk factors. This specification offers a natural fit for the log-linear relationship between mortality risk and age.[16] The models are estimated using maximum likelihood and we report hazard ratios throughout.

In the basic specification, *X* includes two indicators of smoking status ("current" and "former" smokers, with those never smoking the reference group) and four indicators of weight (underweight, overweight, obese class 1, and obese class 2 or 3, with healthy weight as the reference group). We experimented with more complete or flexible models of risk factors by including controls for hypertension and hypercholesterolemia, interactions of smoking and obesity with education, or both. These results are discussed briefly below. One caveat to the overall modeling approach is that we assume there are no changes in year effects within a given survey, so that we average mortality effects of risk factors across these years.

As mentioned, counterfactual estimates of death rates were calculated using data from one survey but the mortality equation from another. For instance, we calculate what the mortality risk would have been in the NHANES I period if the population contained the risk factors present in the NHIS between 1987 and 2000 by applying the parameters estimated from NHANES I (denoted with the subscript 71-75) to the distribution of risk-factors in the NHIS (subscripted with 87-00). For the college educated, this is calculated as:

[16] This specification gives rise to a Gompertz distribution of time to death.

$$\hat{E}\left[mortality\left(X, college, age; \theta_{1971-75}\right) \mid college = 1, survey = 1987-2000\right] = \frac{1}{N_i} \sum_{i \in 87-00, college=1} \exp\left(\beta'_{71-75} * X_i + \lambda_{71-75} * college_i + \gamma_{71-75} * age_i\right) \quad (2)$$

We can compare this estimate to actual predicted mortality hazard in NHANES I to see how changes in the behavioral risks have affected mortality between the two time periods. Alternatively, we can compare the mortality rate calculated from (2) to that obtained using both the NHIS mortality equation and risk factors, to indicate how much of the total secular trend is due to changes in the model coefficients – that is how much occurs for reasons other than changes in the risk factors. In the tables, we perform the estimates described above separately by education groups and calculate both actual and counterfactual educational gradients.

Because of small sample sizes in NHANES III, our primary trend decomposition is for NHANES I versus the NHIS. However, we also re-estimated mortality models for the 1990s and simulated predicted mortality using the smaller NHANES III sample.

The standard errors reported below are more conservative than those usually obtained in regression decompositions. We accounted for both estimating error in the mortality model and sampling error for the distribution of risk factors in different survey periods (rather than just the former as is typical).[17] Appendix A details the process used to derive the correct standard errors.

V. Mortality Hazard Rate Estimates

Table 4 displays estimates of the mortality equations described in equation (1) for subsamples stratified by sex, controlling for education, smoking and obesity status. Since we are

[17] We find that for our standard errors are 20-25% larger than those obtained without accounting for errors in the distribution of risk factors.

estimating hazard models, coefficients greater (less) than one indicate increased (decreased) mortality risk.

Two findings stand out. First, controlling for smoking and body weight (Model B), the college-educated have lower expected mortality rates than their less educated peers, and this differential has increased over time. The predicted hazard rates of highly educated NHANES I men are a statistically insignificant 12% below those of non-college attendees. For NHANES III and NHIS males, the corresponding differences are 26% and 25%, and these differences are significant. College-educated women in NHANES I have a (statistically insignificant) 9% higher hazard rate than their counterparts; for NHANES III and NHIS females, the predicted rates are 12% and 20% lower. For females, the estimated college education effect is only statistically significant in the NHIS.

Obesity and smoking do explain a portion of the educational differential in mortality rates at a point in time, as expected since both represent significant health risks. This can be seen by noting that the predicted risk reduction associated with a college education is always larger in specifications that do *not* control for smoking or body weight (Model A of the table) than when these are included (Model B). In such models, college attendance is associated with a risk reduction of 22%, 31% and 33% for males in the NHANES I, NHANES III and the NHIS, and -6%, 24% and 25% for corresponding females.

Second, current smoking is associated with a much larger increase in mortality rates than other risk factors, and with some suggestion that these adverse effects have increased over time. Averaging across specifications, the hazard rates of current smokers are around twice as high as for persons who have never smoked. Severe (class 2 or 3) obesity also confers a substantial elevation in mortality risk, raising the predicted hazard rates by 44% to 128%, although the

differences are not always statistically significant. Being underweight or mildly obese predicts increased death rates, although usually by statistically insignificant amounts. Overweight (but non-obese) persons sometimes have slightly reduced mortality risk. The mortality equations estimated using the NHIS resemble those estimated obtained from NHANES III, except that the former are more precise. For this reason, we focus on results using the NHIS below.

VI. Education-Mortality Gradients

We next use the hazard rate estimates, described above, to examine the extent to which trends in education-mortality gradients result from changes in behavioral risk factors. Throughout, we present mortality ratios with college attendees as the reference group, so that ratios above one indicate higher mortality for the less educated. Table 5 shows the simulated mortality ratios under different scenarios. Each *row* shows the data source used to estimate the mortality models – either NHANES I covering 1971-75 or the NHIS for 1987-2000. The *columns* indicate the data from which the population risk factors are obtained. Entries on the main diagonal therefore indicate actual education gradients. For instance, less educated males were predicted to have 31% higher hazard rates of death than male college attendees in 1971-75, and the gap rises to 52% in 1987-2000; the 21 percentage point increase is shown as the third element of the main diagonal. By contrast, less educated males would have had mortality 56% higher than male college attendees in 1971-75 if the hazard rate equation in 1987-2000 had applied in that time period. This figure would be 27% for the NHIS time period, using the NHANES I mortality model.

Table 5 shows that changes in population risk factors play essentially no role in explaining the trend increase in mortality gradients. As mentioned, less educated NHANES I

males have a 31% higher risk of death than their college-attending peers. The differential using the same hazard rate equation but the population risk factors from the NHIS – 15 to 25 years later – is a slightly smaller 27%. The implication is that changes in behavioral risks explain *none* of the rise in the education gap in mortality. Patterns of smoking and obesity play a similarly small role in all of the remaining comparisons in Table 5, regardless of whether estimates use parameters based on either the NHANES I or NHIS data.

By contrast, the mortality decrement for being less educated, holding patterns of smoking and obesity constant, worsens with time. Continuing with the previous example, predicted mortality hazard rates of the less educated NHANES I male sample would be 56% higher than for those with more schooling, had the parameters from the NHIS mortality equation applied, which is larger than the actual (52%) differential observed in the later period.

The bottom panel of Table 5 shows analogous results for females. Secular changes in smoking and obesity from the 1970s to the 1990s account for a 2 to 3 percentage point rise in the education-mortality ratio, out of a 42 point total increase (the mortality ratio rises from 0.92 to 1.34). Thus, trends in these risk factors explain less than 10% of the increase in the female education-mortality gradient, with more than 90% resulting from changes in the returns to behavioral risks and education (given a set of behavioral risks).

These results might be an artifact of the blunt measures of smoking used. To this point, we have controlled for tobacco use only at the extensive margin (in part because this is measured more accurately than smoking intensity). However changes at the intensive margin might be important if college-educated tobacco users have cut the amount they smoke by more than their non-college counterparts.[18] Similarly, there could be differences in the timing of last use among

[18] This possibility is salient given the importance of tobacco-related causes of death shown in prior literature (Wong et al. 2002; Meara, Richards, and Cutler 2008).

former smokers, which could be important because health risks continue to be elevated for a substantial period of time after smoking cessation. These possibilities are addressed in Table 6, which displays simulated mortality ratios based on mortality hazard rate models that include additional controls for smoking intensity (<5, 6-10, 11-19, 20, 21-39, > 39 cigarettes on smoking days) and the time since quitting smoking (<1, 2-5, 6-10, 11-15, 16-20, and >20 years).

The results in Table 6 are virtually identical to those in Table 5 for males, indicating that little is lost by using our parsimonious covariates for current or previous smoking. Conversely, differential trends in the intensity and timing of tobacco use do seem to explain some of the rise in the excess mortality of less educated females. Specifically, trends in risk behaviors are estimated to account for between 7 and 17 percentage points of the total 43 point (from 0.92 to 1.35) increase in the mortality gradient for women, implying that these explain 20% to 40% percent of the total change. Nevertheless, even in this case, the majority of the secular trend (60% to 80%) does *not* result from changes in the pattern of behavioral health risks.

As mentioned, education levels changed dramatically over the period studied. The proportion of males attending college rose over 60 percent (from 33% to 53%) and that of females more than doubled (from 24% to 49%) between NHANES I and the NHIS. The composition of individuals within these education groups therefore probably also changed (e.g. the non-college educated were likely to be lower in the ability distribution in the 1990s than the 1970s). We tested whether our results were sensitive to such compositional changes using the following procedure. First, to make the education shares in the latter period equal to those from NHANES I, we duplicated all observations with education equal to some college (13-15 years of education). Second, we re-weighted these observations to shift 24.89% of women and 20.72% of

men from the high to low education group in the NHIS data.[19] Third, we estimated the basic mortality models for the revised NHIS data, and simulated mortality as before.

The results of this exercise, summarized in Table 7, are similar to the main findings in Table 5, indicating that the latter are not being driven by compositional changes of education subgroups. Using the mortality equation from NHANES I, changing risk factors can explain no more than a small portion of the widening gap in mortality. Based upon the hazard rate estimates from the NHIS, changes in behavioral risks imply no change or declining educational gradients in mortality for men and 8 percentage points of a 53 point increase for women. Once again, these results indicate that the widening education gap in death rates results primarily from changes in the mortality function rather than in the distribution of risk factors.

As a final check, we replicated the results using NHANES III rather than NHIS data (see Appendix Table A.1). Although smaller sample sizes imply less precise estimates, changes in smoking and obesity again explain little of the trend in education-mortality gradients. The male mortality ratio increases 8 percentage points (from 1.26 to 1.34), between 1971-75 and 1988-94, with changes in smoking and obesity predicted to *reduce* the gap by 5 points. For women, trends in the risk factors account for 6 to 9 percentage points of a 28 point rise in the gradient (which grows from 0.95 to 1.23).

Using the NHANES III data, we also examined patterns for alternative mortality models that: 1) added interactions between smoking and obesity, and 2) included these interactions plus supplementary controls for high blood pressure and cholesterol. The results were essentially unaffected by these alternatives (also summarized in Table A.1), except that changes in risk factors provided even less of an explanation for the rising education gaps of women when

[19] This approach – randomly splitting the data until shares were equal by duplicating observations and re-weighting them – has the benefit of not introducing sampling error.

including the most comprehensive controls. Specifically, in this specification, changes in the risk factors were responsible for anywhere between 1 percentage point narrowing and a 6 percentage point widening in educational mortality ratios, compared with a 25 point increase in the female educational gradient (and, as observed before, educational gradients would have narrowed for men).

The bottom line is that trends in behavioral risk factors can explain, at most, only a small share of the observed widening in the mortality gap across education groups.

The finding that tobacco use explains a large share of point-in-time differences in mortality across groups but not changes over time seems surprising, given that tobacco use declined by more for the better educated than for the less educated. However, several factors could explain this surprising result. First, the mortality effects of "current smoking" are larger in magnitude in later time periods compared with earlier time periods, although the change in returns to smoking over time is not precisely measured. But if some characteristics of smokers such as their co-morbid conditions, other environmental exposures (living or working with other smokers), or other factors are related to smoking status, these could explain why the rate of smoking in the population is less important than the effect of smoking for those individuals who remain smoking. This, combined with a higher level of smoking among the less educated could generate the results we observed. Furthermore, smoking rates vary much less by education group among individuals over the age of 50 (results available upon request), among whom most deaths occur.

VII. Cardiovascular and Cancer Mortality

In this section, we separately analyze the education gradients in deaths due to CVD and cancer. These are the two most important sources of mortality in the United States[20] and both are influenced by behavioral risks such as smoking and obesity. Thus, they help indicate why mortality gradients have changed. Ideally, we would examine even more detailed categories – such as cancers closely linked to smoking – but sample size limitations prohibit this.

Table 8 presents age-adjusted 5-year mortality rates for these two causes, and from all causes combined. Cancer and CVD account for 69% to 78% of all deaths in our data, with slightly higher shares for women than men and in NHANES I than in the NHIS. In relative terms, cancer has become more important over time for men as cancer death rates have increased modestly (from 1.6% to 1.8%) while CVD mortality has fallen rapidly (from 3.1% to 1.6%). By contrast 5-year death rates from the two causes did not change for women over 30 year period.

These overall patterns conceal strong differences across education groups, particularly for cancer. Five-year death rates from cancer rose from 1.6% to 2.2% (1.0% to 1.2%) for less educated men (women), while declining from 1.4% to 1.3% (1.4% to .8%) among college attendees. Conversely, CVD death rates fell for both sexes and education groups, although more sharply for men than women. Changes in cancer deaths account for nearly half of the increased mortality rate gap by education occurring from the 1970s to the 1990s (.7 for males and .8 for females of the 1.5 and 1.7 percentage point increases). In contrast, patterns of CVD deaths narrowed the mortality gaps for males and had no impact on educational gaps for females. Thus, changes in cancer mortality play a key role in accounting for trends in educational gradients in total mortality.

Hazard rate estimates for deaths from cancer and cardiovascular disease are displayed in Table 9. Estimates for the early 1970s are imprecise and make comparisons across time periods difficult. For males, the findings suggest that the education gradient in cancer mortality rose between NHANES I and NHIS: the relative cancer mortality hazard associated with higher education declined from 1.12 to 0.78. In other words, the protective effect of education (or its correlates) increased over time. We do not observe a similar decline in mortality risk for CVD among college attendees. The pattern by broad disease is the same for females as among males – hazard rates for the college-educated remained unchanged for CVD, but declined significantly for cancer mortality (from 0.96 to 0.76).

We next calculated counterfactual cancer and CVD mortality ratios, corresponding to those for total mortality in Table 5. For both cancer and obesity, we find little evidence that the changing risk profiles explain the widening in the mortality gradient (Table 10). This is true regardless of the whether the model is estimated using NHANES I or the NHIS. Instead, the widening mortality gradient is due to the increase in relative risk of death associated with behaviors like smoking, and, conditional on these behaviors, the relative risk of low education for cancer mortality. Our most robust finding (supported in those specifications with the most precise estimates) is that risk factors did not change in ways that made CVD or cancer deaths change differentially over time by education. Instead, the changing return to education and to risk factors has favored the more educated.

Our findings create a puzzle regarding how changes in medical technology or other factors influencing the impact of risk factors on death might increase educational gaps in mortality. To garner more information on this puzzle with available data, we estimated models

[20] Heart and cerebrovascular diseases, both of which are included in CVD, were the first and third most common causes of mortality in 2006, together accounting for 31.7 percent of deaths; cancer was the second most frequent

of two risk factors for cardiovascular mortality, cholesterol and hypertension, as a function of education, smoking, and obesity in each of the four NHANES samples, controlling for gender. These two factors are both quantitatively important, and they provide a good example because prescription drugs to control cholesterol and blood pressure have been widely credited with mitigating the role of these risk factors in deaths due to cardiovascular disease. For brevity, these results are not shown, but they are available upon request. As expected based on the results in Table 3, the advantage of education for cholesterol control and hypertension did not change over time– education conferred a protective effect against high cholesterol that remained nearly identical in each NHANES sample, and rates of hypertension were similar across education groups in each of the four NHANES samples.[21] Taken together, the results suggest that the health risk of obesity, as measured by hypertension, fell significantly over time. However, there is no suggestion that access to medical care, or compliance with complex medical regimens, for example, contributed to educational disparities in these important risk factors for deaths due to CVD.

VIII. Discussion

This paper examines how education-related disparities in mortality rates changed between the early 1970s and the end of the twentieth century, and investigates the extent to which behavioral risk factors explain these trends. Consistent with previous research, we document that the educational gradients are steeper for men than women but have widened over time more for females than males.

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cause, being responsible for 23.0 percent of deaths (Heron et al. 2009).

[21] Our analysis revealed a striking change in the effect of obesity on hypertension over time. In NHANES III and IV, the elevated risk of hypertension among overweight individuals observed in earlier surveys nearly disappeared. This pattern did not hold for cholesterol.

Our most striking finding is that the widening educational gaps in death rates are *not* explained by secular changes in key behavioral risk factors. In particular, although higher *levels* of mortality among the less educated are partially due to their higher rates of smoking , trends in smoking explain little if any of the relative increase in mortality for the less educated over the last three decades. In our main estimates, the mortality gap between males without and with college rose 21 percentage points, whereas differential changes in smoking and obesity would have led to a 4 or 5 point decrease, *ceteris paribus*. For women, patterns of smoking and obesity can explain approximately 3 points out of the 42 percentage point increase. The risk factors play a more important role in some (but not all) alternative specifications but never explain more than a small fraction of the rising education-gradient in mortality.

Instead, it is the return to education (conditional on health behaviors) and changes in returns to behaviors that are important. For example, the detrimental impact of smoking has strengthened over time for both men and women, as have the negative consequences of severe obesity for females (although small NHANES 1 sample sizes limit the precision of these estimates). We found suggestive evidence that these effects are particularly pronounced for cancer, which has become a more important source of death over time.

There are several possible explanations for these findings. One is that the highly educated have better access to medical care, and thus achieve superior results due to higher productive efficiency as described by Michael Grossman (1972) and Donald Kenkel (1991). This might have become more pronounced over time, as sophisticated treatments have improved the survival of those receiving them[22] and new technologies have led to earlier detection.[23]

[22] Productive efficiency is discussed at length by Michael Grossman (1972) and Donald Kenkel (1991); there is some evidence on this point from Dana Goldman and James Smith (2002) based on better adherence to complex treatment regimens for HIV and diabetes, and Cutler and Lleras-Muney (2008), who document better adherence to cancer prevention and screening among better educated. There is evidence of substantial SES differences in the

Adherence to prescribed regimes may also have become both more important and more difficult over time, yielding large gains in life expectancy for highly educated individuals who have better adherence rates.[24]

The issues are especially salient for cancer, which may play a particularly important role in the increasing gradients observed for total mortality and where the trend reduction in death rates appears to be due to improvements in screening, earlier diagnosis, and treatment (Cutler 2008). For instance, there is recent evidence that breast and prostate cancer are detected at later stages and treated less aggressively, once detected, for low than high SES individuals and that these differences are associated with an inverse SES-mortality relationship (Bouchardy et al., 2006; Byers et al., 2008; Rapiti et al., forthcoming).

A second explanation is that environmental risks may have declined more over time for the highly educated. For instance, changes in the nature of employment may have led to larger reductions in job stress and work-related health hazards for professional than manual occupations.[25] There could also be similar patterns for geographically-based risks, such as those due to pollution or local government spending on health and the infrastructure, particularly if housing has become more geographically segregated by education. Chay and Greenstone (2003) for example, demonstrated that recession-induced factory slowdowns and closings had beneficial

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receipt of cardiac catheterization and coronary revascularization following heart attacks (David A. Alter, et al. 1999; Rathore et al. 2000). Similar differences in access to expensive cancer diagnostic technology and treatments might be expected.

[23] For instance, Smith (2007) shows that the overall prevalence of undiagnosed diabetes fell dramatically between 1976-80 and 1998-2002 but with much larger reductions for college attendees than for the non-college educated.

[24] Chen and Lange (2009) show that less educated individuals have relatively low rates of screening for various cancers, especially among those with significant risk factors for cancer. The same patterns are observable in self-assessed cancer risk, suggesting that education might play a role in making individuals aware of their risks *and* consequently in obtaining adequate treatment. Goldman and Smith (2002) find that educated individuals have greater adherence rates for the treatment of HIV and diabetes, with consequent improvements in health outcomes.

[25] For example, Ickovics, et al. (1997) show that life stress, social isolation and depression were all inversely related to social class (as measured by education and occupation) among men hospitalized following heart attacks.

impacts on infant health (due to the associated reductions in certain air pollutants), especially among disadvantaged groups, measured based on black versus white infant deaths.

Understanding the mechanisms for the effects we identify is important for achieving national goals of reducing disparities in longevity and medical outcomes. Our results do *not* imply that improvements in the health-related lifestyles of the less educated would yield no benefits. To contrary, reducing smoking, obesity, hypertension and hypercholesterolemia would improve health. However, our results suggest that even the complete elimination of disparities in behavioral risks across education groups would be unlikely to do away with education-differentials in mortality.

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TABLE 1: AGE-ADJUSTED 5-YEAR MORTALITY RATES (PERCENT) BY SEX, EDUCATION AND TIME PERIOD

EDUCATION	NHANES I (1971-75)	NHANES III (1988-94)	NHIS (1987-96)	CHANGE FROM NHANES I to NHIS
		MALES		
≤12 Years	6.3 (0.57)	5.0 (0.63)	5.8 (0.13)	-0.5 (0.58)
> 12 Years	5.8 (0.86)	3.6 (0.60)	3.8 (0.11)	-2.0 (0.87)
		FEMALES		
≤12 Years	3.1 (0.37)	2.9 (0.43)	3.6 (0.09)	+0.5 (0.38)
> 12 Years	3.7 (0.71)	3.2 (0.57)	2.7 (0.09)	-1.0 (0.72)

Note: Reported statistics are age-adjusted to the year 2000, gender specific age distribution of the NHIS. Five-year mortality was computed for individuals surviving at least one year from baseline. Reported mortality rates are in percent and changes in percentage points. In parentheses: standard errors.

TABLE 2: COMPLETED EDUCATION BY GENDER AND DATA-SET (PERCENT)

EDUCATION	NHANES I (1971-75)	NHANES II (1976-80)	NHANES III (1988-94)	NHANES IV (1999-2004)	NHIS (1987-96)
			MALES		
< 12 years	34 (0.9)	27 (0.7)	19 (0.8)	12 (0.7)	13 (0.1)
12 years	33 (0.9)	35 (0.8)	32 (1.0)	27 (0.9)	33 (0.2)
> 12 years	33 (0.9)	38 (0.8)	50 (1.1)	60 (1.0)	54 (0.2)
			FEMALES		
< 12 years	34 (0.9)	29 (0.7)	17 (0.8)	12 (0.7)	13 (0.1)
12 years	42 (0.9)	43 (0.8)	40 (1.0)	27 (0.9)	38 (0.1)
> 12 years	24 (0.8)	28 (0.7)	43 (1.0)	61 (1.0)	49 (0.1)

Note: See note on Table 1.

TABLE 3A: SUMMARY STATISTICS FOR AGE ADJUSTED RISK FACTORS, BY TIME PERIOD, MALES

Risk Factor	Education (Years)	% with Risk Factor					Change in Risk Factors (%)	
		NHANES I 1971-75	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2004	NHIS (1987-2000)	NHANES I -NHANES IV	NHANES I - NHIS
Smoking								
Current	≤12	48 (1.1)	45 (1.0)	43 (1.4)	40 (1.6)	41 (0.2)	- 8 (1.9)	- 7 (1.1)
Former	≤12	31 (1.1)	35 (0.9)	36 (1.4)	32 (1.5)	31 (0.2)	1 (1.9)	0 (1.1)
Current	> 12	34 (1.7)	33 (1.4)	20 (1.3)	19 (1.1)	21 (0.2)	-15 (2.0)	-13 (1.7)
Former	> 12	30 (1.7)	35 (1.4)	38 (1.6)	31 (1.3)	32 (0.2)	1 (2.1)	+2 (1.7)
Obesity								
Class 1	≤12	12 (0.7)	12 (0.6)	17 (1.0)	21 (1.0)	15 (0.2)	9 (1.2)	3 (0.7)
Class 2/3	≤12	2 (0.3)	3 (0.3)	7 (0.7)	10 (1.0)	5 (0.1)	8 (1.0)	3 (0.3)
Class 1	> 12	8 (1.0)	8 (0.8)	14 (1.1)	20 (1.1)	12 (0.1)	12 (1.5)	4 (1.0)
Class 2/3	> 12	1 (0.4)	2 (0.4)	5 (0.7)	9 (0.8)	4 (0.1)	8 (1.0)	3 (0.4)

TABLE 3A, CONTINUED: SUMMARY STATISTICS FOR AGE ADJUSTED RISK FACTORS, BY TIME PERIOD, MALES

Risk Factor	Education (Years)	% with Risk Factor				Change in Risk Factors (%)
		NHANES I 1971-75	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2004	NHANES I - NHANES IV
Hypertension						
Stage I	≤12	31 (1.1)	29 (0.9)	19 (1.1)	16 (1.2)	-15 (1.6)
Stage II	≤12	17 (0.9)	18 (0.7)	4 (0.6)	3 (0.6)	-14 (1.1)
Stage I	> 12	26 (1.6)	28 (1.3)	15 (1.2)	13 (0.9)	-13 (1.8)
Stage II	> 12	16 (1.3)	17 (1.1)	4 (0.6)	3 (0.4)	-13 (1.4)
High Cholesterol	≤12	29 (1.1)	29 (0.9)	25 (1.2)	19 (1.3)	-10 (1.7)
	> 12	30 (1.7)	31 (1.4)	22 (1.3)	17 (1.0)	-13 (2.0)

Note: All reported statistics are age-adjusted to the year 2000, gender specific age distribution of the NHIS. Class 1 obesity refers to individuals with a BMI of 30 to 34.9. Class 2/3 obesity refers to persons with a BMI of 35 or greater. Hypertension stages I and II are defined in text. High cholesterol refers to total cholesterol >240 mmHG.

TABLE 3B: SUMMARY STATISTICS FOR AGE ADJUSTED RISK FACTORS, BY TIME PERIOD, FEMALES

Risk Factor	Education (Years)	% with Risk Factor					Change in Risk Factors (%)	
		NHANES I 1971-75	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2004	NHIS (1987-2000)	NHANES I - NHANES IV	NHANES I - NHIS
Smoking								
Current	≤12	35 (1.0)	37 (0.8)	33 (1.2)	36 (1.6)	35 (0.2)	1 (1.9)	0 (1.0)
Former	≤12	13 (0.7)	15 (0.6)	23 (1.1)	21 (1.3)	20 (0.2)	8 (1.5)	7 (0.7)
Current	> 12	28 (1.7)	25 (1.3)	18 (1.2)	16 (1.0)	20 (0.2)	-12 (2.0)	-8 (1.7)
Former	> 12	19 (1.4)	24 (1.3)	29 (1.5)	27 (1.2)	26 (0.2)	8 (1.8)	7 (1.4)
Obesity								
Class 1	≤12	12 (0.7)	12 (0.6)	16 (0.9)	21 (1.3)	12 (0.1)	9 (1.5)	0 (0.7)
Class 2/3	≤12	5 (0.5)	7 (0.5)	12 (0.8)	16 (1.2)	7 (0.1)	11 (1.3)	2 (0.5)
Class 1	> 12	8 (1.0)	8 (0.8)	11 (1.0)	15 (1.0)	9 (0.1)	7 (1.4)	1 (1.0)
Class 2/3	> 12	3 (0.6)	3 (0.6)	8 (0.9)	14 (0.9)	5 (0.1)	11 (1.1)	2 (0.6)

TABLE 3B, CONTINUED: SUMMARY STATISTICS FOR AGE ADJUSTED RISK FACTORS, BY TIME PERIOD, FEMALES

Risk Factor	Education (Years)	% with Risk Factor				Change in Risk Factors (%)
		NHANES I 1971-75	NHANES II 1976-80	NHANES III 1988-94	NHANES IV 1999-2004	NHANES I To NHANES IV
Hypertension						
Stage I	≤12	22 (0.9)	21 (0.7)	12 (0.8)	14 (1.1)	-8 (1.4)
Stage II		15 (0.7)	16 (0.6)	4 (0.5)	5 (0.7)	-10 (1.0)
Stage I	> 12	19 (1.4)	23 (1.3)	11 (1.0)	10 (0.8)	-9 (1.6)
Stage II		13 (1.2)	10 (0.9)	4 (0.7)	5 (0.6)	-8 (1.3)
High Cholesterol	≤12	36 (1.0)	35 (0.8)	29 (1.2)	23 (1.4)	-13 (1.7)
	> 12	29 (1.7)	32 (1.4)	27 (1.4)	19 (1.0)	-10 (2.0)

Note: All reported statistics are age-adjusted to the year 2000, gender specific age distribution of the NHIS. Class 1 obesity refers to individuals with a BMI of 30 to 34.9. Class 2/3 obesity refers to persons with a BMI of 35 or greater. Hypertension stages I and II are defined in text. High cholesterol refers to total cholesterol >240 mmHG.

TABLE 4: MORTALITY MODEL WITH CONTROLS FOR EDUCATION, SMOKING AND OBESITY

Characteristic	(1) NHANES I 1971-75	(2) NHANES III 1988-94	(3) NHIS 1987-2000	(4) NHANES I 1971-75	(5) NHANES III 1988-94	(6) NHIS 1987-2000
	MALES			FEMALES		
Model A						
Some College	0.78 (0.12)	0.69 (0.11)**	0.67 (0.02)**	1.06 (0.22)	0.76 (0.14)	0.75 (0.02)**
Model B						
Some College	0.88 (0.14)	0.74 (0.12)*	0.75 (0.02)**	1.09 (0.22)	0.88 (0.17)	0.80 (0.02)**
Former Smoker	1.04 (0.18)	1.43 (0.30)*	1.30 (0.03)**	1.26 (0.29)	1.27 (0.24)	1.38 (0.04)**
Current Smoker	1.88 (0.33)**	1.99 (0.48)**	2.41 (0.08)**	1.69 (0.32)	2.57 (0.52)**	2.43 (0.07)**
Underweight	2.11 (0.48)**	2.18 (0.71)**	1.61 (0.07)**	1.13 (0.28)	1.09 (0.35)	1.38 (0.05)**
Overweight	0.85 (0.12)	0.79 (0.13)	0.88 (0.02)**	0.72 (0.14)*	1.12 (0.23)	1.04 (0.03)
Class 1 Obesity	1.71 (0.33)**	0.70 (0.16)	1.11 (0.04)**	1.32 (0.29)	1.07 (0.28)	1.45 (0.05)**
Class 2/3 Obesity	1.44 (0.70)	1.44 (0.47)	1.70 (0.10)**	1.71 (0.49)*	2.28 (0.59)**	1.94 (0.09)**

Note: Table displays the relative risk obtained from hazard rate models. Robust standard errors are in parentheses. Model A controls only for educational status; model B adds covariates for smoking and obesity. The reference group in model B is normal weight individuals who never smoked. Individuals are followed for 10 years after the NHANES I and III interview dates. The NHIS covers the 1987-2000 surveys, with mortality follow-up continuing for 10 years or through 2002 if this is less than 10 years. * (**) denote a difference in relative risk from 1 that is significant at a 0.10 (0.05) level.

TABLE 5: OBSERVED AND PREDICTED MORTALITY RATIOS AND DIFFERENTIALS

Model estimated on:	Distribution of Risk Factors		
	NHANES I (1971-1975)	**NHIS (1987-2000)**	**CHANGE: NHANES I to NHIS**
		MALES	
NHANES I	**1.31**	1.27	-0.04
	(0.22)	(0.20)	(0.09)
NHIS	1.56	**1.52**	-0.04
	(0.10)	(0.04)	(0.09)
Difference	0.25	0.25	**0.21**
	(0.21)	(0.20)	(0.22)
		FEMALES	
NHANES I	**0.92**	0.95	0.02
	(0.19)	(0.19)	(0.06)
NHIS	1.31	**1.34**	0.03
	(0.08)	(0.04)	(0.07)
Difference	0.38	0.39	**0.42**
	(0.19)	(0.19)	(0.20)

Note: Mortality ratios refer to the ratio of predicted mortality between those without and with college educations. Ratios greater than one imply greater mortality for the less educated. In-sample risk ratios are shown in bold. Counterfactual risk ratios or differentials are based on the estimates from table 4, but for a different survey wave. Standard errors are in parentheses.

TABLE 6: OBSERVED AND PREDICTED MORTALITY RATIOS AND DIFFERENTIALS WITH ADDITIONAL CONTROLS FOR SMOKING INTENSITY AND QUIT TIMING

Model estimated on:	Distribution of Risk Factors		
	NHANES I (1971-75)	**NHIS (1987-2000)**	**CHANGE: NHANES I to NHIS**
		MALES	
NHANES I	**1.31** (0.22)	1.26 (0.20)	-0.05 (0.10)
NHIS	1.52 (0.10)	**1.53** (0.04)	+0.01 (0.09)
Difference	0.21 (0.21)	0.27 (0.20)	**0.22** (0.23)
		FEMALES	
NHANES I	**0.92** (0.19)	1.09 (0.23)	0.17 (0.15)
NHIS	1.28 (0.08)	**1.35** (0.04)	0.07 (0.07)
Difference	0.36 (0.19)	0.25 (0.23)	**0.43** (0.20)

Note: See note on Table 5. Mortality ratios in this table are estimated in the same way, except that the mortality model includes additional controls for the intensity of smoking and time since last tobacco use for former smokers.

TABLE 7: OBSERVED AND PREDICTED MORTALITY RATIOS AND DIFFERENTIALS WITH CONSTANT SHARE OF INDIVIDUALS IN HIGH EDUCATION GROUP

Model estimated on:	Distribution of Risk Factors		
	NHANES I (1971-75)	**NHIS (1987-2000)**	**CHANGE: NHANES I to NHIS**
		MALES	
NHANES I	**1.31**	1.28	-0.03
	(0.22)	(0.20)	(0.09)
NHIS	1.69	**1.65**	-0.03
	(0.11)	(0.05)	(0.10)
Difference	0.38	0.37	**0.34**
	(0.21)	(0.21)	(0.23)
		FEMALES	
NHANES I	**0.92**	0.97	0.05
	(0.19)	(0.19)	(0.06)
NHIS	1.37	**1.45**	0.08
	(0.09)	(0.06)	(0.08)
Difference	0.45	0.48	**0.53**
	(0.20)	(0.20)	(0.20)

Note: See note on Table 5. Mortality ratios in this table are estimated in the same way, except that observations have been reweighted to maintain constant shares in each education category, using the procedure discussed in the text.

TABLE 8: AGE-ADJUSTED 5-YEAR MORTALITY RATES BY YEARS OF EDUCATION AND CAUSE OF DEATH

Cause of Death	NHANES I (1971-75)			NHIS (1987-2000)		
	<=12	>12	Difference	<=12	>12	Difference
			MALES			
All Causes	6.3	5.8	0.5	5.8	3.8	2.0
	(0.57)	(0.86)	(1.03)	(0.13)	(0.11)	(0.17)
Cancer	1.6	1.4	0.2	2.2	1.3	0.9
	(0.29)	(0.43)	(0.52)	(0.08)	(0.07)	(0.11)
Cardiovascular	3.5	2.2	1.3	1.8	1.3	0.5
	(0.43)	(0.54)	(0.69)	(0.08)	(0.06)	(0.1)
			FEMALES			
All Causes	3.1	3.7	-0.6	3.6	2.7	1.1
	(0.37)	(0.71)	(0.80)	(0.09)	(0.09)	(0.13)
Cancer	1.0	1.4	-0.4	1.2	0.8	0.4
	(0.21)	(0.44)	(0.49)	(0.05)	(0.05)	(0.07)
Cardiovascular	1.5	1.3	0.2	1.4	1.2	0.2
	(0.26)	(0.42)	(0.49)	(0.06)	(0.06)	(0.08)

Note: See note on Table 1. Cancer and cardiovascular disease account for 74% (69%) of all male deaths in the NHANES I (NHIS) follow-up and 78% (70%) of corresponding female deaths.

TABLE 9: MORTALITY MODEL FOR MALIGNANT NEOPLASMS & CARDIOVASCULAR DISEASE

	NEOPLASMS				CARDIOVASCULAR DISEASE			
	MALES		FEMALES		MALES		FEMALES	
	NHANES I (1971-75)	NHIS (1987-2000)	NHANES I (1971-75)	NHIS (1987-2000)	NHANES I (1971-75)	NHIS (1987-2000)	NHANES I (1971-75)	NHIS (1987-2000)
Some College	1.12	0.73**	0.91	0.76**	0.72	0.78	0.96	0.90*
	(0.32)	(0.04)	(0.29)	(0.04)	(0.17)	(0.04)	(0.35)	(0.04)
Former Smoker	1.00	1.34**	1.30	1.27**	0.99	1.41**	1.25	1.41**
	(0.34)	(0.09)	(0.46)	(0.08)	(0.25)	(0.08)	(0.46)	(0.07)
Current Smoker	1.89*	2.53**	0.78	2.23**	1.80*	2.78**	2.74**	2.58**
	(0.56)	(0.13)	(0.28)	(0.13)	(0.46)	(0.17)	(0.70)	(0.12)
Underweight	2.01+	1.26*	0.68	1.23**	1.33	1.43*	0.59	1.14*
	(0.80)	(0.13)	(0.45)	(0.08)	(0.51)	(0.15)	(0.27)	(0.08)
Overweight	0.85	1.00	1.25	1.16**	0.77	0.97	0.51*	1.02
	(0.21)	(0.04)	(0.40)	(0.06)	(0.15)	(0.05)	(0.15)	(0.05)
Class 1 Obesity	1.23	1.47**	1.48	1.74**	1.76*	1.00	1.36	1.34**
	(0.48)	(0.09)	(0.64)	(0.10)	(0.46)	(0.07)	(0.40)	(0.09)
Class 2/3 Obesity	2.35	2.26**	1.93	2.46**	1.61	1.17	1.77+	1.37**
	(1.85)	(0.20)	(1.15)	(0.19)	(1.00)	(0.15)	(0.59)	(0.12)

Note: See note on Table 4. + significant at 10%; * significant at 5%; ** significant at 1%

TABLE 10: OBSERVED AND PREDICTED MORTALITY RATIOS AND DIFFERENTIALS FOR MALIGNANT NEOPLASMS AND CARDIOVASCULAR DISEASE

	MALIGNANT NEOPLASMS DISTRIBUTION OF RISK FACTORS			CARDIOVASCULAR DISEASE DISTRIBUTION OF RISK FACTORS		
MODEL ESTIMATED ON:	**NHANES I (1971-75)**	**NHIS (1987-2000)**	**CHANGE: NHANES I to NHIS**	**NHANES I (1971-75)**	**NHIS (1987-2000)**	**CHANGE: NHANES I to NHIS**
			MALES			
NHANES I	**1.02**	1.00	-0.02	**1.57**	1.55	-0.02
	(0.30)	(0.29)	(0.09)	(0.37)	(0.35)	(0.12)
NHIS	1.66	**1.57**	-0.09	1.53	**1.47**	-0.06
	(0.13)	(0.06)	(0.11)	(0.12)	(0.07)	(0.09)
DIFFERENCE	0.64	0.57	**0.55**	-0.04	-0.08	-0.10
	(0.30)	(0.29)	(0.31)	(0.36)	(0.36)	(0.37)
			FEMALES			
NHANES I	**1.13**	1.11	-0.02	**1.05**	1.11	0.06
	(0.37)	(0.34)	(0.08)	(0.39)	(0.39)	(0.09)
NHIS	1.42	**1.42**	0.00	1.14	**1.20**	0.05
	(0.12)	(0.07)	(0.09)	(0.07)	(0.05)	(0.05)
DIFFERENCE	0.29	0.31	**0.29**	0.09	0.08	**0.15**
	(0.37)	(0.35)	(0.37)	(0.39)	(0.40)	(0.39)

SEE NOTE TO TABLE 5