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**Can Educational Gradients in Smoking and Obesity Explain Educational
Gradients in Mortality?**

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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, and with each new study on the topic, the gap between education groups is rising (Kitagawa and Hauser 1973; Pappas et al. 1993; Crimmins and Saito 2001; Lin et al. 2003; Singh et al. 2006; Meara et al. 2008; Mazzid et al. 2008; Krieger et al. 2008). For example, although life expectancy increased by 1.6 years in the decade from 1990 to 2000 among those attending college, there was no change in life expectancy among those who did not, and by 2000 25 year old college attendees could expect to live 7 years longer than their non-college bound peers (Meara et al. 2008). Findings like this are not new. Since the revelation of Kitagawa and Hauser and updates by Pappas et al. (1993) differential mortality and life expectancy has thrust the issue of health disparities high onto the political agenda. The most recent set of health goals for the country lists reducing health disparities (including race and ethnicity as well as economic status) along with improving population health as the two major goals for the country (Healthy People 2010).

Despite this focus, the issue of why health outcomes are so disparate has yet to be determined. Some studies focus on physician behavior, and whether physicians treat higher income patients better than lower income patients (CITE). Other analyses stress the behavioral differences between education groups: the better educated are less likely to smoke, drink, and be obese (at least among women) than the less educated (Cutler and Lleras-Muney 2007). Still other analyses link socioeconomic differences in outcomes to social status: the high status of better educated people may reduce exposure to unalleviated stress, improving health in the same way that medications do (Marmot et al., 1997).

In this paper, we seek to understand the differential trend in health by education group over the past three decades. We focus specifically on the period from the early 1970s to the early 1990s, and given the implication of tobacco and obesity to gaps in mortality by education (Wong et al. 2000; Lin et al. 2001; Meara et al. 2008), we focus on behavioral factors. Our primary concern is with behavioral differences such as smoking and obesity. We also examine some of the immediate correlates of these risk factors, hypertension and cholesterol. Smoking and obesity are the two leading behavioral causes of death in the United States. Smoking accounts for 18 percent of all mortality, and obesity accounts for up to 17 percent – though with much more uncertainty about the estimate (Mokdad et al. 2004; Flegal et al. 2005; Willett et al. 2005). People who are obese often develop high blood pressure (hypertension) and high cholesterol. Given that management of these risk factors is itself a behavioral issue, we examine how disease management varies across education groups.

We document trends in behavioral risk factors, and demonstrate that for some groups (white women) some factors (smoking) can explain part of the growing mortality gaps. However, when we simulate mortality from the 1970s to the late 1990s, using only changes in the distribution of risk factors within education groups, we find that the risk profile (smoking and obesity in particular) explains very little of the growing gap in mortality.

We can find explanations for some of the increase in health disparities over time. In particular, the rising disparity among women is due in part to differential trends in smoking. During a period of rapid declines in smoking rates among better educated women, women who did not attend college actually increased rates of smoking. But among men, the source of disparities are elusive. We suggest possible explanations for this and present suggestive evidence in support of our leading explanation for this trend, competing risk of tobacco. Over

time, even if risk factors move in tandem among low and high education groups, if the level of risk differs across groups and the relative return/penalty for a given risk factor changes over time, this can create rising disparities in mortality. We argue that advances in the treatment of cardiovascular disease in recent decades made the returns to being a non-smoker higher, and thus the relative mortality risk for smokers (who are disproportionately found among less educated groups) increases over time. We present suggestive evidence in support of this hypothesis and also consider alternatives.

In section II, we describe the empirical approach and data, section III presents descriptive trends in mortality and the results of simulations of changing risk factors, section V considers alternative hypotheses for the trends we observe in the data, and section VI concludes.

II. Empirical Approach and Data

An important pre-cursor to our work is that by Cutler, Glaeser, and Rosen (2007), which we refer to as CGR. The goal of this earlier work was to understand how competing trends, such as a one third reduction in smoking from 1960 to 2000, and a doubling of obesity in recent years would contribute to population health and mortality on net. Among behavioral risk factors, smoking contributes the most to deaths in a given year, about 435,000 annually (Mokdad et al. 2004).

Obesity is next in importance for mortality, although the number are more controversial with obesity contributing 100,000 to 400,000 deaths per year (Flegal et al. 2005; Willett et al. 2005). In this earlier work, CGR estimated the contribution of demographics and risk factors including smoking, drinking, obesity, and blood pressure to 10-year mortality risk in the 1970s and again at the end of the 20th century, assuming that medical technology and access remained

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constant over time. Based only on changes in risk factors over this period, the population appears healthier since the age-adjusted risk of dying within 20 years for those aged 20 to 74 fell from 9.8 to 8.4 percent, a 14 percent reduction. This earlier work documented that smoking and control of high blood pressure contributed most to this improved risk profile, but recent increases in the rate of obesity have important offsetting effects that imply the population may be less healthy as time goes on, if current trends continue.

The National Health and Nutrition Examination Survey

The goal of our analysis is to understand whether differential trends in major behavioral risk factors of obesity and smoking can explain widening mortality gradients over the last three decades. To do this, we will use data come from a series of nationally representative surveys conducted by the National Center for Health Statistics, Centers for Disease Control and Prevention (CDC). Each survey selected a sample of the civilian noninstitutionalized population of specified ages using a complex, stratified, multi-stage probability cluster design. Baseline information was obtained from the first National Health Examination Survey (NHANES I), covering the period 1971-1975. Subsequent data are from the second and third NHANES, for 1976-1980 (NHANES II) and 1988-1994 (NHANES III), and from the first six years (1999-2004) of the most recent NHANES survey (hereafter referred to as NHANES IV) that does not contain breaks between cycles. (See: <http://www.cdc.gov/nchs/nhanes.htm> for further information).

The NHANES includes a physical exam component and biological measures of weight, height, blood pressure, and cholesterol. Because we are interested in changing determinants of mortality over time, rather than mortality trends related to the change in composition of U.S.

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residents, we mitigate the role of immigration by excluding Hispanics. We also limit the sample to whites, since the NHANES does not provide adequate sample size to estimate mortality among non-white populations.

As we are interested in the adult population, we limit the sample to those aged 25-74 (NHANES I and II do not interview persons older than 74.) This provides 5,942 respondents from NHANES I (1971-75), 8,408 respondents from NHANES II (1976-80), 4,930 respondents from NHANES III (1988-94), and 5,143 respondents from NHANES IV (1999-2004). The NHANES I and NHANES III also include mortality follow-up surveys, in which respondents are matched to National Death Index records over the 10 years after the survey ended. Thus, for these samples, we have information on the timing and cause of death for those who died in the 10 years following the survey (through 1981-85 for NHANES I and 1998-2000 for NHANES III).

Education measures

We followed the two broad educational categories used in Meara, Richards and Cutler (2008) and categorize education into those who completed more than 12 years of schooling (college attendees), and those with less education. During the period between the 1970s and the early years of the 21st century, the share of individuals attending college grew dramatically. Among men, the share attending college grew from 33 to 60 percent. Among women, the share of college attendees grew from 25 to 61 percent. Given the compositional changes in education, a two-category variable is not ideal. However, the data do not adequately support finer educational categories, and in other settings, analyses of the impact of changing rates of college

attendance yielded no difference in measured trends in educational gaps in mortality or other risk factors such as smoking (Meara, Richards and Cutler 2008; Kenkel 200xx).

Behavioral Risk Factors

As described above, smoking, obesity, control of blood pressure, and cholesterol are all potentially important risk factors for mortality. We used the NHANES data to characterize these risks as follows. We used simple indicators of current or former smoking status. Smoking status (as opposed to smoking intensity which is measured with much more error) is the single most important modifiable risk factor for mortality. Thus, for smoking risks, a simple measure of current or former smoking status is relatively informative.

We distinguished five body weight/obesity standards based on body mass index (BMI), which is calculated as weight in kilograms divided by height in meters squared, using measurements from medical examinations employing standardized procedures and equipment. Following national and international standard (World Health Organization, 1997; National Heart, Lung and Blood Institute, 1998), adults were defined to be “underweight”, “healthy weight”, “overweight”, “class 1 obese”, and “class 2/3 obese” if their BMI was: <18.5 , 18.5 to <25 ; ≥ 25 to <30 , ≥ 30 to <35 and ≥ 35 . BMI is less accurate than laboratory measures of body composition because it does not account for variations in muscle mass or in the distribution of body fat. It, nevertheless, is a favored method of assessing excess weight because it is simple, rapid, and inexpensive to calculate.

CGR demonstrated that one of the reasons obesity has important consequences for mortality relates to the fact that obese individuals tend to have hypertension (elevated blood pressure) and/or high cholesterol that place them at risk of serious cardiovascular events (Cutler

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Glaeser and Rosen 2007). Effective medications to control high blood pressure and cholesterol have substantially reduced the mortality risks for individuals likely to suffer hypertension or high cholesterol. Thus, one might expect that over time, any differential reduction in hypertension or high cholesterol across education groups could contribute to expanding mortality gaps. We again follow the measures used in CGR.

Blood pressure was divided into four groups following the recommendations of the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure(JNC VII): normal blood pressure (systolic blood pressure (SBP) \leq 120 mmHG and diastolic blood pressure (DBP) \leq 80 mmHG); pre-hypertension (120 \leq SBP $<$ 140 or 80 \leq DBP $<$ 90); stage 1 hypertension (140 \leq SBP $<$ 160 or 90 \leq DBP $<$ 100); and stage 2 hypertension (160 \leq SBP or 100 \leq DBP). To maintain comparability of cholesterol measures over time, we use total cholesterol measures rather than separate measures of its components, since these weren't available in the earliest NHANES. Cholesterol levels were divided into three groups based on the recommendations of the Third Report of The National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (NCEP, 2001): normal cholesterol (total cholesterol $<$ 200); borderline high cholesterol (200 \leq total cholesterol $<$ 240); and high cholesterol (240 \leq total cholesterol).

Empirical specification for mortality equations

We adopted an approach of estimating mortality equations similar to those previously used in CGR, conducting our main empirical analysis in two steps. We first estimated mortality equations describing how mortality risk changes with different risk factors using NHANES I data linked to the mortality follow-up. In the second step, we used the estimated coefficients

from models to compute the predicted mortality for each education group in all four periods (NHANES I-IV) that we would expect if only the behavioral risk factors changed over time, but the equation translating demographics and risk factors remained fixed. We also estimated mortality equations using the NHANES III data linked to mortality and then applied these estimates to risk factors and demographics observed in NHANES I-IV.

In contrast to CGR, we implemented the framework slightly more flexibly to allow for differences by sex and education. Educational and mortality trends over the time period differ dramatically by sex, and therefore we expect the relevant equations that determine mortality as a function of risk factors may differ as well. For this reason, we estimated everything separately by sex. In another departure from CGR, we estimated proportional hazard models rather than models of 10-year mortality risk. This approach helped to maximize the efficiency of our estimation given relatively small numbers of respondents within sex and education groups, although the qualitative patterns were similar using either method.

To model how demographics, education, and behavioral risk factors relate to mortality risk, we specified mortality risk in the following form.

$$(1) \text{ mortality}(X, \text{college}, \text{age})_I = \exp(\beta' * X_I + \lambda * \text{college}_I + \gamma * \text{age}_I).$$

In this set-up, the I subscript indicates that all variables used in the estimation of the parameters come from the NHANES I survey and follow-up data. College is an indicator of whether a respondent reported >12 years of education, age is expressed in single years between 25 and 75, and X includes a set of behavioral risk factors. The specification in (1) imposes a log-linear relationship between mortality risk and age. We found that the log-linear specification in age

indeed fits the data well and could not be rejected in favor of more flexible specifications.

Separately for each sex, we estimated proportional hazard models using maximum likelihood estimation, and we report hazard ratios throughout. In the most basic specification, X included indicators of current and former smoking status, four weight indicators (underweight, overweight, obese class I, and obese class II or III v. normal weight). We estimated more flexible specifications of these risk factors in later models, but the basic results of the paper obtained from this baseline specification were unaffected by adding further risk factors or interacting risk factors with each other. One caveat to this approach is that it assumes no changes in year effects within survey.

Using the parameters estimated from (1), we then applied the distribution of risk-factors X from each of the four NHANES surveys to the risk model estimated on NHANES I.

For example, the predicted mortality in NHANES III for individuals with some college education was calculated as:

$$(2) \quad \hat{E} \left[\text{mortality} (X, \text{college}, \text{age}; \theta_I) | \text{college} = 1, \text{survey} = III \right] = \frac{1}{N_{III,>12}} \sum_{i \in III, \text{college}_i=1} \exp(\beta'_I * X_i + \lambda_I * \text{college}_i + \gamma_I * \text{age}_i)$$

By calculating this predicted mortality by NHANES survey and education group, we obtained the change in excess mortality of the less educated holding the risk model constant. We then estimated (1) using NHANES III data and again applied these estimates to each NHANES. By holding constant the distribution of risk factors, X, we used this to compute how excess mortality changed due to changes in conditional risk, or the change in the relationship between risk factors and mortality. The standard errors on the mortality ratios implied by these simulations were obtained by deriving their asymptotic variances using the delta method and iterated expectations. These standard errors account for both the estimation error for the mortality model and the

sampling error for the distribution of risk factors X in the different survey years. Details are in an appendix.

III. Descriptive Results

Table 1 reports the basic trends in mortality and education, separately for men and women. Education data are provided for each of the four NHANES samples; 5-year mortality rates are only available for NHANES I (deaths in 1972-1981) and NHANES III (deaths in 1989-1999). Education has risen over time, with a somewhat larger increase observed for women than men: the fraction of college-educated men rose from 33 percent in 1971-1975 to 60 percent in 1999-2004, whereas the corresponding increase for women was from 25 to 61 percent. XXX add a reference showing how these results compare to other national statistics XXX

Mortality rates fell over time for every group but by considerably larger amounts for more highly educated individuals. For non-college educated men, five-year death rates declined from 6.3 for the NHANES I sample to 5.9 percent for the NHANES III group, while the corresponding decrease for males who had been to college was from 5.0 to 3.7 percent. Similarly, mortality rates fell from 3.4 to 2.8 percent for college-educated females but changed little (declining from 2.7 to 2.6 percent) for those who had not been to college.

Table 2 provides information on how risk factors related to smoking, obesity, hypertension and hypercholesterolemia differ across education groups and have changed over time. The results are again presented separately for males and females. Highly educated individuals have lower rates of smoking in all periods but the differential has increased over time, particularly for women. For example, college-educated men were 70 percent as likely to smoke as their less schooled counterparts in 1971-1975 (34 versus 48 percent) but did so less

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than half as often in 1999-2004 (19 versus 40 percent). The more educated women were four-fifths as likely to smoke in the earlier time period (29 versus 36 percent) and smoked only about two-fifths as often in the later period (17 versus 39 percent). Indeed, it is noteworthy that the smoking rates of non-college educated females have actually *increased* over time. These results lend credence to the possibility that increasing education-related mortality gradients may partially be explained by patterns of smoking.

Obesity has risen rapidly over time, as is well-known, but it is not obvious whether these changes will play much role in explaining the steepening education-mortality gradient. In particular, although obesity is increasing for both education groups, for the last decade (from NHANES III to IV) the growth has been larger for the college-educated than for those with less schooling. Between 1988-1994 and 1999-2004, the obesity rates of college educated men rose from 22 to 34 percent, compared to a change that is smaller in both absolute and relative terms (from 31 to 38 percent) for non-college educated males. Corresponding increases for females are from 23 to 32 percent for the college-educated and from 33 to 43 percent for women with less schooling. The increase in obesity is even larger but shows a similar pattern if we consider all four waves of the NHANES. On the other hand, the most severe forms of obesity (class 2 and class 3) have been growing more rapidly in absolute (but not relative) terms for the less educated, which could explain some of the change in the gradient. However, although severe obesity has the most adverse consequences, its prevalence may still be too small to explain a large share of the rising gap in mortality.

Health risks related to high blood pressure and high cholesterol have been declining for all groups. Between 1971-1975 and 1999-2004, the fraction of non-college educated males (females) with either stage 1 or stage 2 hypertension fell from 47 to 21 (35 to 18) percent while

the corresponding prevalence of college-educated men (women) decreased from 41 to 17 (30 to 14) percent. More severe (stage 2) hypertension has also declined for all groups and it is not apparent whether there are sufficient differences in the levels or rates of these decreases to account for changes in the mortality gradient.

The story is similar for hypercholesterolemia. The prevalence of high cholesterol decreases with education for women (but not men) and has fallen over time by roughly similar amounts for all groups. Thus, 29 (35) percent of non-college educated men (women) had high cholesterol in 1971-1975 compared to 22 (27) percent in 1999-2004. Corresponding figures for the college-educated were 29 (28) and 19 (21) percent. Levels of the “good” HDL cholesterol also appear to have increased slightly for all groups, over the shorter time period for which data are available.

IV. Regression Estimates

Tables 3 and 5 display the results of alternative forms of the mortality risk model described in equation (1). In all cases, we estimate the models separately for men and women and using mortality follow-up data for NHANES I and III. The specification summarized in Table 3, controls for education, smoking and obesity status. We refer to this as “Model A” below and it is the most basic model because it does not allow for interactions between smoking and body weight, nor does it control for the additional health risk factors related to blood pressure or cholesterol. Since we are estimating hazard models, coefficients greater (less) than one indicate increased (decreased) mortality risk.

Several results stand out from the basic model. First, after controlling for smoking and body weight, the point estimates suggest that college educated males have lower predicted

mortality rates and that the differential has increased over time – the reduction in relative mortality risk is approximately 10 percent for the NHANES I cohort but is almost twice as large for NHANES III – however, the estimates are imprecise and we cannot reject the possibility of no education-related mortality gradient or change over time in it. The results for females also hint at a relative improvement in the status of the highly educated over time although, interestingly, college-educated women in the NHANES I cohort have an insignificantly greater predicted mortality risk.

Predicted mortality ratios are always smaller in models that do *not* control for smoking or body weight (not shown on the table) than when these are included: the coefficients are 0.80 and 1.03 for NHANES I men and women, and 0.74 and 0.79 for their NHANES III counterparts, compared to 0.90, 0.80, 1.06 and 0.94 in model A. This indicates that differences in the two health behaviors do explain a portion of the mortality gradients, although the estimates are often imprecise and this does not say much about whether the *trends* of the gradients are similarly explained.

The risk factors generally conform to the expected patterns. Current smoking yields the largest additional mortality risk, generally at least doubling the hazard rate, with larger increases for the NHANES III than NHANES I sample. Most estimates suggest that previous smoking also elevates the risk of death, but the increases are much smaller and statistically insignificant. Consistent with previous research (Flegal et al 2005; Willett et al. 2005) being underweight or severely obese are both associated with high mortality hazard rates; however, the predicted effects vary substantially by sex and time period and are frequently statistically insignificant. Mild obesity is also generally correlated with high mortality, although the estimated effects vary substantially, while overweight most commonly predicts statistically insignificant reductions in

the hazard rates. Although the coefficients are informative, it is useful to remember that substantial effects only occur if the risk factor has strong effects on the hazard rate and occurs with some frequency in the population. For example, being underweight is predicted to substantially increase male mortality in the NHANES I cohort but occurs sufficiently rarely (i.e. just 5.15 percent of NHANES I men are underweight) that it is unlikely to have much effect on the education gradients.

Estimates from the basic model assume that the risks related to smoking or obesity are additive, however, it is possible that the combined effects of multiple risk factors exceed the consequences of each individually. Table 4 provides evidence that this occurs. The models estimated include full interactions between smoking and body weight, with normal weight non-smokers constituting the reference group. What is noteworthy is that the mortality hazard rates of persons outside the health weight range (i.e. those who are overweight, mildly obese or severely obese) are much higher for current smokers than for non-smokers. The link between underweight and mortality risk may partially reflect advanced disease and deterioration that precede death but the increased hazard rates of severely obese smokers is profound. That said, it seems unlikely that the inclusion of smoking-obesity interactions will substantially affect the extent to which the risk factors are anticipated to change the education-mortality gradient. The evidence for this is that the coefficient on college education is only marginally changed when moving from the basic specification (model A) to the one that includes interactions (model B).

Our most fully specified model – model C shown in Table 5 – adds controls for risks related to hypertension and hypercholesterolemia. Hypertension is associated with substantially (but not always statistically significantly) higher mortality for females, with smaller effects for men. Pre-hypertension never has much effect and the estimates for high cholesterol suggest

somewhat elevated but imprecisely estimated effects on mortality. These additional controls generally have limited effect on the characteristics controlled for in the previous models. Most significantly, the college coefficients remain relatively unchanged, suggesting that the estimated effects on education-mortality gradients are unlikely to be strongly influenced by the choice of models. This expectation is confirmed in the next section.

V. Observed and Predicted Mortality Gradients

We next use the hazard model results, described above, to estimate the extent to which trends in the education-mortality gradients do (or do not) result from changes in the risk factors related to smoking, body weight and, in later models, their interaction as well as cholesterol and blood pressure. Results from the basic model, corresponding to the estimates in Table 3, are summarized in Table 6. We examine both mortality ratios and absolute differentials. The college educated are the base group in both estimates, so that ratios above one or positive differences indicate higher mortality for the less educated.

Results displayed in bold refer to within-sample risk ratios or differentials (i.e. those that are based on the mortality equation for the NHANES sample over which subsequent mortality is observed). For instance, the first entry on Table 6 indicates that non-college educated NHANES I males had a 26 percent higher predicted five-year mortality rate than their counterparts who had attended college. The corresponding entry in the next column shows that the corresponding mortality differential for NHANES III men would have been 21 percent, given the risk profile of that group but using the mortality equation estimated for the NHANES I cohort. This shows that changes in the risk factors would be expected to have resulted in a (statistically insignificant) reduction in the mortality gradient between the two survey waves. Instead, as shown in the

second row of column 2, the ratio increased to 34 percent. (This 13 percentage point difference is statistically significant, with an associated t-statistic exceeding 3). Similarly, the hazard model estimates from NHANES III predicted that the mortality ratio for NHANES I would have been 39 percent, which substantially (and significantly) exceeds the 26 percent difference actually observed (shown in the second row of the first column). Thus, these estimates show that changes in risk factors fail to explain any of the secular increase in mortality risk ratios observed for less educated men. Instead, what appears to be important are changes in the effects of these factors.

A similar result is obtained when looking at the absolute death rate differentials of males. Less educated NHANES I men have a 0.192 percent higher mortality rate than their college-educated counterparts but would be predicted to have a 0.222 percent greater probability using the NHANES III mortality equation. This exceeds the 0.178 percent difference actually observed at the later date, confirming that changes in the risk factors would tend to lower the education gradient. However, none of these differences are statistically significant and it is noteworthy that the absolute mortality differential actually falls between NHANES I and III (from 0.192 to 0.178 percent). The reason that absolute differentials are falling while the ratios are increasing is because initial (NHANES I) mortality rates are so much higher for the less educated (6.3 versus 5.0 percent) so that a bigger absolute decline is not inconsistent with a smaller percentage reduction.¹

Changes in risk factors are somewhat more important for females. Interestingly, less educated women have lower mortality rates initially but a substantial, and statistically significant schooling-mortality gradient has emerged for the NHANES III cohort. As shown in the third and

¹ Note that the trend in predicted mortality presented here differs slightly from the trend suggested by the raw data. This occurs because our maximum likelihood estimation strategy, by construction, does not estimate the mean mortality rates. Using changes in the return to risk factors, we would predict a narrowing of absolute mortality gaps, but we know from table 1 that male mortality gaps widened both in absolute and relative terms.

fourth rows of Table 6, trends in smoking and body weight predicts a rise in the non-college vs. college mortality ratio from 0.95 to 1.01, using the NHANES I mortality equation and from 1.14 to 1.23 using the NHANES III equation. However, neither change is statistically significant (the associated t-statistics are 0.68 and 1.0) and the majority of the increased gradient remains unexplained. Similarly, the absolute mortality differential rises by 0.114 percentage points (from -.00022 to .00092) between NHANES I and III, with the risk factors being responsible for between 0.029 and 0.037 points, or between one-quarter and one-third of this change.

Table 7 investigates whether the findings just discussed are sensitive to the choice of mortality models estimated. The first two rows repeat the results presented in Table 6, while the next four present estimates for the model with smoking-body weight interactions (model B) and also with controls for health risks related to hypertension and high cholesterol. We focus on estimates of education gradients based on mortality ratios, since indicate changes in relative risks.

The pattern of findings is extremely robust to the choice of mortality models. Changes in the risk factors never account for any of the trend increase in male mortality gradients, with the estimated mortality ratios being essentially identical for all three specifications. The health risks do have some explanatory power for females but most of the trend in mortality ratios remains unaccounted for and the explained portion is particularly weak in the most comprehensive specification that includes both smoking-weight interactions and controls for hypertension and hypercholesterolemia.

VI. Alternative hypotheses for changing returns to risk factors

Why don't behaviors explain more of growing gradients

As shown for males and females in Table 2, smoking rates among the educated have declined substantially more rapidly than among the less educated. However, the observed changes in smoking rates for the full population do not tell the whole story. In order to understand changes in overall mortality rates, we need to focus on the distribution of risk factors among the elderly. And, if we compare those aged 50+ with those younger than 50, then important differences in the distribution of smoking rates emerge. Among males older than 50, the difference in smoking rate between the more and less educated males has in fact stayed roughly constant. In NHANES I, the difference in smoking rates between the less and the more educated was 13 %-points. For the NHANES III, the difference amounted to 12%-points and for NHANES IV, it had in fact fallen to 9%-points. Among older males, the differences in smoking rates have therefore declined, explaining why the overall divergence in smoking rates has not lead to a substantial additional widening of the education gradient of males. Among females older than 50, the smoking gradient has in fact risen from about 1%-point in NHANES I to about 10 %-points in NHANES IV. This partially explains why the education gradient due to risk factors has risen for females, but not for males. But this evidence presents a puzzle. If causes of death for which tobacco is a big risk factor contribute significantly to growing educational disparities in mortality, why doesn't smoking matter?

Increasing returns to smoke-free lifestyle

As treatment of cardiovascular disease has advanced, both the length and quality of life have improved among individuals with heart disease. This improvement implies that the health returns to limiting tobacco exposure (by not smoking and working and living in areas where environmental tobacco smoke is minimal) have improved over time. Where formally non-

smokers with other risk factors for cardiovascular disease may have still experienced significant risks of mortality, this risk has declined dramatically. To shed light on this issue, we show the rate of death for two broad categories of disease, cardiovascular disease and cancers. Table 8 shows 5-year mortality rates for these causes. Two things stand out. First, cardiovascular disease deaths have dropped dramatically, regardless of education group. The mortality ratios increase only slightly for women, and do not change for men. This result is consistent with the analyses of the Multiple Cause of Death files in Meara, Richards and Cutler (2008; see table A7) which show that among 45-64 years olds, cardiovascular disease deaths decline rapidly and they contribute very little to growing mortality differences by education. Second, deaths for cancer dropped dramatically and differentially for males. Male college-attendees experienced a large drop in cancer deaths from .015 to .005 between NHANES I and III. The less educated men also experienced improvements in cancer deaths, but they weren't nearly as dramatic.

We also estimated models similar to those in model A, but restricting to cardiovascular disease deaths and including hypertension and cholesterol as independent variables. Table 9 shows the results of these cardiovascular death models. There was no dramatic change in the return to smoking or obesity over this period. Finally, table 10 shows similar models, but for cancer. Here, one dramatic finding stands out. The relative mortality risk to being a smoker for men is much higher in the latter period than in NHANES I, but for males only. It is possible that men, among whom CVD deaths fell most dramatically, the competing risk of cancer among the smokers became much more important in the latter period, contributing to a rising disparity in deaths by education. (NEEDS MORE WORK TO EXPLAIN INTERPRET)

Why don't behaviors explain more? The role of environmental tobacco smoke (ETS)

Research conducted in a variety of settings since 2000 demonstrate that clean air regulations can have substantial effects on cardiovascular health, even among non-smokers.

Within the US, studies of Helena, Montana and Pueblo Colorado, two isolated communities that implemented smoke free legislation in public places, suggest substantial drops in admissions for AMI (Sargent et al. 2004; Bartecchi et al. 2006). Recent research from Scotland demonstrated that exposure to smoke dropped substantially among men, especially from lower SES groups, and this exposure coincided with dramatic 20% reductions in hospital admissions for acute coronary syndrome (Haw and Gruer 2007; Pell et al 2008). Reductions in exposure to smoke were concentrated among non-smokers, as one might expect given that rates of smoke exposure among individual living in households with at least one smoker did not change following the legislation. Thus, the health benefits seem to accrue mainly due to differences in exposure at work and other public places such as pubs and restaurants. A small study of workplace smoking bans in Wisconsin further implicates ETS as a potential determinant of growing educational gaps in cardiovascular outcomes because it confirms that smoking bans were more likely among higher SES workers. Thus, during earlier time periods, even if individuals quit smoking, the health benefits of doing so may have been muted because they still had high levels of exposure to ETS. As bans like those in the workplace began to proliferate, they tended to influence higher SES workers. Also, for individuals living with a smoker, the health benefits of clean air legislation or workplace bans were not fully realized. This last hypothesis has several testable implications. IF secondary smoke plays a role, one should observe bigger growth in gradients among non-smokers compared with smokers, since less educated non-smokers are more likely to work and live with other smokers, and thus have more ETS exposure.

VII. Conclusions (MORE COMING SOON)

This paper examined trends in mortality and behavioral risk factors by education group between the 1970s and 2000 to try to quantify the importance of behavioral risk factors on growing gaps in mortality by education. We document several trends. First, as previous evidence suggests, risk factors are more prevalent among less educated groups. In some cases changing trends in risk factors, such as smoking, have favored better educated groups (especially among those under the age of 50). In contrast to smoking, obesity trends do not greatly favor the highly educated since obesity grows fastest among college attendees in recent time periods even as extreme obesity, still a rare event, grows more rapidly among the less educated.

Based on hazard models relating risk factors to mortality for historical and recent time periods, we simulated the deaths that would occur in each education group if only the risk profiles of each group, and not the return to these risk profiles, changed. The gaps in mortality due only to changes in obesity and smoking can explain none of the growing male gap in mortality by education and only some of the growing female gap in education. We hypothesize, and show suggestive evidence that gaps arise not because of differential trends in smoking behavior, but because of the interaction between higher rates of smoking among the less educated and the larger relative mortality risk for smoking compared with not smoking that develops over time.

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References (COMING SOON)

National Heart, Lung, and Blood Institute. 1998. *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report.* Washington D.C.: US Government Printing Office.

World Health Organization. 1997. *Obesity: Preventing and managing the global epidemic. report of a WHO consultation on obesity.* Geneva, Switzerland: World Health Organization.

TABLE 1: AGE ADJUSTED EDUCATION AND MORTALITY FROM NHANES I – IV

| Education | NHANES I (1971-1975) | NHANES II (1976-1980) | NHANES III (1988-94) | NHANES IV (1999-2004) |
|--|-------------------------|--------------------------|-------------------------|--------------------------|
| 5 year Mortality Rate by Education: Males | | | | |
| ≤12 Years | 6.3% | --- | 5.9% | --- |
| > 12 Years | 5.0% | --- | 3.7% | --- |
| Years of Education Completed: Males | | | | |
| < 12 years | 35% | 29% | 20% | 13% |
| 12 years | 32% | 34% | 31% | 27% |
| > 12 years | 33% | 37% | 49% | 60% |
| 5 year Mortality Rate: Females | | | | |
| ≤12 Years | 2.7% | --- | 2.6% | --- |
| > 12 Years | 3.4% | --- | 2.8% | --- |
| Years of Education Completed: Females | | | | |
| < 12 years | 33% | 29% | 18% | 12% |
| 12 years | 42% | 43% | 40% | 26% |
| > 12 years | 25% | 28% | 42% | 61% |

Note: All reported statistics are age-adjusted to the age distribution obtained from pooling the NHANES.

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TABLE 2: SUMMARY STATISTICS FOR AGE ADJUSTED RISK FACTORS, FROM NHANES I – IV

| Risk Factor | Education (Years) | Males | | | | Females | | | |
|-----------------------|----------------------|-------------|--------------|---------------|--------------|-------------|--------------|---------------|--------------|
| | | NHANES I | NHANES II | NHANES III | NHANES IV | NHANES I | NHANES II | NHANES III | NHANES IV |
| Current Smoker | ≤ 12 | 48% | 45% | 43% | 40% | 36% | 38% | 35% | 39% |
| Never Smoked | | 21% | 20% | 22% | 28% | 51% | 48% | 43% | 41% |
| Current Smoker | > 12 | 34% | 33% | 19% | 19% | 29% | 25% | 19% | 17% |
| Never Smoked | | 36% | 32% | 44% | 50% | 52% | 51% | 54% | 57% |
| BMI | ≤ 12 | 25.9 | 26.0 | 28.0 | 29.3 | 26.6 | 26.4 | 28.2 | 29.7 |
| Obesity: Class 1 | | 12% | 12% | 25% | 24% | 18% | 12% | 18% | 25% |
| Obesity: Class 2/3 | | 2% | 2% | 6% | 14% | 7% | 8% | 15% | 18% |
| BMI | > 12 | 25.4 | 25.9 | 27.0 | 28.8 | 25.1 | 25.5 | 26.6 | 28.2 |
| Obesity: Class 1 | | 8% | 10% | 17% | 23% | 10% | 12% | 15% | 17% |
| Obesity: Class 2/3 | | 1% | 2% | 5% | 11% | 4% | 4% | 8% | 15% |
| Systolic / Diastolic | ≤ 12 | 132 / 85 | 132 / 84 | 126 / 77 | 124 / 75 | 129 / 81 | 127 / 81 | 120 / 73 | 120 / 71 |
| Hypertension: Stage 1 | | 31% | 28% | 18% | 15% | 21% | 20% | 11% | 13% |
| Hypertension: Stage 2 | | 16% | 18% | 4% | 6% | 14% | 15% | 3% | 5% |
| Systolic / Diastolic | > 12 | 131 / 84 | 130 / 84 | 124 / 78 | 122 / 75 | 126 / 80 | 123 / 21 | 119 / 72 | 119 / 71 |
| Hypertension: Stage 1 | | 25% | 28% | 15% | 12% | 18% | 22% | 10% | 10% |
| Hypertension: Stage 2 | | 16% | 17% | 5% | 5% | 12% | 9% | 4% | 4% |
| Total Cholesterol | ≤ 12 | 218 | 217 | 213 | 206 | 224 | 221 | 216 | 210 |
| HDL | | - | 43 | 44 | 45 | - | 53 | 53 | 55 |
| High Cholesterol | | 29% | 28% | 27% | 22% | 35% | 33% | 32% | 27% |
| Total | > 12 | 217 | 218 | 210 | 203 | 216 | 218 | 214 | 206 |
| HDL | | - | 45 | 46 | 47 | - | 56 | 59 | 60 |
| High Cholesterol | | 29% | 31% | 23% | 19% | 28% | 30% | 29% | 21% |

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Note: All reported statistics are age-adjusted to the age distribution obtained from pooling the NHANES. Class 1 obesity refers to individuals with a BMI of 30 to 34.9. Class 2/3 obesity refer to persons with a BMI of 35 or greater. Stage 1 and 2 hypertension are defined in text. High cholesterol refers to >240 mmHg.

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TABLE 3: MORTALITY MODEL WITH SMOKING AND OBESITY ONLY (Model A)

| Characteristic | (1) Nhanes I - Males | (2) Nhanes III - Males | (3) Nhanes I - Females | (4) Nhanes III - Females |
|-------------------|-------------------------|------------------------------|------------------------------|--------------------------------|
| Some College | 0.90 [0.14] | 0.80 [0.13] | 1.06 [0.22] | 0.94 [0.19] |
| Former Smoker | 1.01 [0.17] | 1.44+ [0.31] | 1.29 [0.29] | 1.16 [0.23] |
| Current Smoker | 1.77** [0.30] | 2.02** [0.52] | 1.66** [0.32] | 2.59** [0.55] |
| Underweight | 2.13** [0.46] | 2.08* [0.69] | 1.07 [0.27] | 1.16 [0.38] |
| Overweight | 0.84 [0.12] | 0.77 [0.13] | 0.73 [0.14] | 1.16 [0.25] |
| Class 1 Obesity | 1.64** [0.32] | 0.67+ [0.16] | 1.31 [0.29] | 1.10 [0.31] |
| Class 2/3 Obesity | 1.35 [0.60] | 1.46 [0.48] | 1.58 [0.44] | 2.37** [0.64] |
| Observations | 2632 | 2211 | 3038 | 2508 |

Note: Table displays hazard rates from a model with log-linear age trend in mortality. The omitted category comprises non-smokers with normal weight (BMI 20-25). Robust standard errors are shown in brackets. + significant at 10%; * significant at 5%; ** significant at 1%

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TABLE 4: MORTALITY MODEL WITH SMOKING AND OBESITY, INTERACTIONS (Model B)

| Characteristic/Interaction | | (1) Nhanes I - Males | (2) Nhanes III - Males | (3) Nhanes I - Females | (4) Nhanes III - Females |
|----------------------------|-------------------|-------------------------|---------------------------|---------------------------|-----------------------------|
| Some College | | 0.9 [0.14] | 0.79 [0.13] | 1.09 [0.22] | 0.95 [0.20] |
| Smoking Status | Weight-Class | | | | |
| Non-Smoker | Underweight | 1.05 [0.93] | 0.01** [0.00] | 1.49 [0.54] | 0.62 [0.50] |
| | Overweight | 0.75 [0.24] | 0.56 [0.23] | 0.59+ [0.16] | 0.98 [0.33] |
| | Class 1 Obesity | 1.96+ [0.72] | 0.30* [0.18] | 1.35 [0.36] | 0.78 [0.35] |
| | Class 2/3 Obesity | 1.51 [1.42] | 0.26 [0.27] | 1.37 [0.60] | 1.92 [0.77] |
| Former Smoker | Underweight | 2.29+ [1.06] | 3.29* [1.98] | 1.22 [0.79] | 1.68 [1.32] |
| | Normal | 1.1 [0.33] | 0.97 [0.36] | 0.83 [0.29] | 1.19 [0.43] |
| | Overweight | 0.82 [0.24] | 0.79 [0.28] | 1.18 [0.44] | 1.21 [0.43] |
| | Class 1 Obesity | 1.2 [0.40] | 0.6 [0.25] | 2.25 [1.15] | 0.89 [0.37] |
| | Class 2/3 Obesity | 0.98 [0.72] | 1.4 [0.68] | 2.05 [0.95] | 1.68 [0.77] |
| Current Smoker | Underweight | 3.77** [1.26] | 3.15* [1.60] | 1.28 [0.48] | 2.64* [1.15] |
| | Normal | 1.56 [0.44] | 1.17 [0.47] | 1.77* [0.50] | 1.55 [0.55] |
| | Overweight | 1.5 [0.44] | 0.97 [0.39] | 1.51 [0.48] | 2.43* [0.94] |
| | Class 1 Obesity | 3.15** [1.22] | 1.37 [0.65] | 0.93 [0.52] | 3.30* [1.56] |
| | Class 2/3 Obesity | 2.8 [2.04] | 5.56** [3.30] | 2.99* [1.40] | 7.94** [3.77] |
| Observations | | 2632 | 2211 | 3038 | 2508 |

Note: Table displays hazard rates from a model with log-linear age trend in mortality. The omitted category comprises non-smokers with normal weight (BMI 20-25). Robust standard errors are shown in brackets. + significant at 10%; * significant at 5%;

** significant at 1%

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Table 5: Mortality Model with Smoking and Obesity Interactions plus Hypertension and Cholesterol (Model C)

| Characteristic/Interaction | | (1) Nhanes I - Males | (2) Nhanes III - Males | (3) Nhanes I - Females | (4) Nhanes III - Females |
|----------------------------|-------------------|-------------------------|---------------------------|---------------------------|-----------------------------|
| Some College | | 0.89 [0.14] | 0.82 [0.13] | 1.1 [0.23] | 0.96 [0.20] |
| Smoking Status | Weight Class | | | | |
| Non-Smoker | Underweight | 0.98 [0.88] | 0.01** [0.01] | 1.53 [0.57] | 0.69 [0.56] |
| | Overweight | 0.77 [0.25] | 0.56 [0.24] | 0.57* [0.15] | 0.92 [0.31] |
| | Class 1 Obesity | 1.98+ [0.73] | 0.31* [0.18] | 1.25 [0.34] | 0.71 [0.32] |
| | Class 2/3 Obesity | 1.32 [1.24] | 0.25 [0.26] | 1.21 [0.52] | 1.7 [0.70] |
| Former Smoker | Underweight | 2.25+ [1.06] | 3.60* [2.11] | 1.27 [0.81] | 1.97 [1.56] |
| | Normal | 1.16 [0.35] | 0.98 [0.36] | 0.86 [0.31] | 1.16 [0.42] |
| | Overweight | 0.82 [0.24] | 0.78 [0.28] | 1.22 [0.46] | 1.12 [0.41] |
| | Class 1 Obesity | 1.18 [0.40] | 0.57 [0.25] | 2.12 [1.09] | 0.8 [0.34] |
| | Class 2/3 Obesity | 0.93 [0.70] | 1.32 [0.65] | 1.78 [0.81] | 1.46 [0.68] |
| Current Smoker | Underweight | 3.67** [1.23] | 3.07* [1.58] | 1.32 [0.49] | 2.82* [1.25] |
| | Normal | 1.57 [0.44] | 1.16 [0.47] | 1.87* [0.53] | 1.56 [0.56] |
| | Overweight | 1.51 [0.45] | 0.94 [0.39] | 1.58 [0.51] | 2.45* [0.95] |
| | Class 1 Obesity | 2.97** [1.17] | 1.24 [0.62] | 0.93 [0.51] | 2.99* [1.47] |
| | Class 2/3 Obesity | 2.59 [1.87] | 4.96* [3.19] | 2.72* [1.26] | 7.39** [3.44] |

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(Table 5 continued)

| | Nhances I - Males | Nhances III - Males | Nhances I - Females | Nhances III - Females |
|-----------------------|-------------------|---------------------|---------------------|-----------------------|
| Pre-Hypertension | 0.76 [0.16] | 1 [0.20] | 0.96 [0.27] | 1.17 [0.29] |
| Stage I Hypertension | 0.82 [0.16] | 1.19 [0.28] | 1.36 [0.37] | 1.62+ [0.45] |
| Stage II Hypertension | 1.11 [0.22] | 1.35 [0.37] | 1.64+ [0.45] | 1.91+ [0.67] |
| High Cholesterol | 1 [0.13] | 1.24 [0.21] | 1.13 [0.19] | 1.16 [0.21] |
| Observations | 2632 | 2211 | 3038 | 2508 |

Note: Table displays hazard rates from a model with log-linear age trend in mortality. The omitted category comprises non-smokers with normal weight (BMI 20-25). Robust standard errors in brackets. + significant at 10%; * significant at 5%; ** significant at 1%

TABLE 6: OBSERVED AND PREDICTED MORTALITY RATIOS AND DIFFERENTIALS

| Model Estimated On: | Distribution of Risk Factors: | | | |
|---------------------|-------------------------------|-----------------------|----------------------------|---------------------------|
| | Mortality Ratio | | Mortality Differential | |
| | NHANES 1 | NHANES 3 | NHANES 1 | NHANES 3 |
| | (1) | (2) | (3) | (4) |
| Males | | | | |
| NHANES 1 | 1.26 [0.08] | 1.21 [0.08] | .00192 [.00050] | .00150 [.00050] |
| NHANES 3 | 1.39 [0.09] | 1.34 [0.08] | .00222 [.00039] | .00178 [.00037] |
| Females | | | | |
| NHANES 1 | 0.95 [0.06] | 1.01 [0.07] | -.00022 [.00029] | .00007 [.00031] |
| NHANES 3 | 1.14 [0.07] | 1.23 [0.08] | .00055 [.00024] | .00092 [.00026] |

Note: Mortality ratios refer to the ratio of predicted mortality between those without and with college educations. Differentials refer to the absolute differences between these two groups. Ratios greater than one or positive differentials imply greater mortality for less educated individuals. In-sample risk ratios or differentials are shown in bold. Counterfactual risk ratios or differentials are based on the estimates from model A, which excludes obesity-smoking interactions and controls for high blood pressure or cholesterol, but for a different survey wave. Standard errors are in brackets.

TABLE 7: OBSERVED AND PREDICTED MORTALITY RATIOS FROM ALTERNATIVE MODELS

| Mortality Model | Model Estimated On: | Distribution of Risk Factors: | | | |
|--|--------------------------------|--------------------------------------|-----------------|-----------------|-----------------|
| | | Males | | Females | |
| | | NHANES 1 | NHANES 3 | NHANES 1 | NHANES 3 |
| | | (1) | (2) | (3) | (4) |
| | | | | | |
| <i>Smoking and Obesity (no interactions)</i> | NHANES 1 | 1.26 | 1.21 | 0.95 | 1.01 |
| | NHANES 3 | 1.39 | 1.34 | 1.14 | 1.23 |
| <i>Smoking and Obesity with interactions</i> | NHANES 1 | 1.26 | 1.20 | 0.95 | 1.00 |
| | NHANES 3 | 1.39 | 1.36 | 1.13 | 1.23 |
| <i>Smoking, Obesity (with interactions), hypertension, and cholesterol</i> | NHANES 1 | 1.27 | 1.20 | 0.97 | 0.96 |
| | NHANES 3 | 1.37 | 1.34 | 1.164 | 1.22 |

Note: Mortality ratios refer to the ratio of actual or predicted mortality between those without and with college educations; ratios greater than one or positive differentials imply greater mortality for less educated individuals. Counterfactual risk ratios are based on the estimates from model A, B or C, as described in the first column.

Table 8: mortality for cardiovascular disease and cancer

Raw cardiovascular 5 year mortality (age-adjusted)

| | <i>Nhances I</i> | | | <i>Nhances III</i> | | |
|--------|------------------|-------|-------|--------------------|-------|-------|
| | All | <=12 | >12 | All | <=12 | >12 |
| Male | 0.029 | 0.034 | 0.024 | 0.017 | 0.020 | 0.015 |
| Female | 0.012 | 0.012 | 0.011 | 0.011 | 0.012 | 0.010 |

Raw cancer 5-year mortality rates (age-adjusted)

| | <i>Nhances I</i> | | | <i>Nhances III</i> | | |
|--------|------------------|-------|-------|--------------------|-------|-------|
| | All | <=12 | >12 | All | <=12 | >12 |
| Male | 0.016 | 0.017 | 0.015 | 0.010 | 0.014 | 0.005 |
| Female | 0.011 | 0.010 | 0.013 | 0.009 | 0.008 | 0.010 |

TABLE 9: MORTALITY MODEL FOR CARDIOVASCULAR DISEASE WITH SMOKING AND OBESITY ONLY (Model A)

| Characteristic | (1) Nhanes I - Males | (2) Nhanes III - Males | (3) Nhanes I - Females | (4) Nhanes III - Females |
|-------------------|-------------------------|------------------------------|------------------------------|--------------------------------|
| Some College | 0.83 [0.18] | 0.82 [0.20] | 1.00 [0.35] | 0.79 [0.28] |
| Former Smoker | 1.05 [0.24] | 1.16 [0.35] | 1.31 [0.45] | 1.08 [0.34] |
| Current Smoker | 1.64* [0.38] | 1.45 [0.55] | 3.05** [0.69] | 2.87** [1.00] |
| Underweight | 1.03 [0.30] | .96 [0.53] | .50 [0.21] | .95 [0.57] |
| Overweight | 0.75 [0.14] | 0.79 [0.13] | 0.54* [0.16] | .75 [0.26] |
| Class 1 Obesity | 0.91 [0.27] | 1.04 [0.16] | 1.21 [0.29] | .70 [0.30] |
| Class 2/3 Obesity | 1.56 [0.44] | 1.34 [0.48] | 1.35 [0.40] | 1.91 [0.79] |
| Observations | 3147 | 2211 | 2701 | 2508 |

Note: Table displays hazard rates from a model with log-linear age trend in mortality. The omitted category comprises non-smokers with normal weight (BMI 20-25). Robust standard errors are shown in brackets. + significant at 10%; * significant at 5%; ** significant at 1%

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TABLE 10: MORTALITY FROM CANCERS, MODEL WITH SMOKING AND OBESITY ONLY (Model A)

| Characteristic | (1) Nhanes I - Males | (2) Nhanes III - Males | (3) Nhanes I - Females | (4) Nhanes III - Females |
|-------------------|-------------------------|------------------------------|------------------------------|--------------------------------|
| Some College | 1.08 [0.30] | 0.55* [0.16] | 1.02 [0.31] | 0.96 [0.29] |
| Former Smoker | .99 [0.31] | 3.30** [0.31] | 1.29 [0.44] | 1.14 [0.36] |
| Current Smoker | 1.73** [0.49] | 6.35** [3.05] | .68 [0.25] | 2.07** [0.72] |
| Underweight | 1.31** [0.48] | 1.35 [0.80] | .85 [0.57] | .78 [0.48] |
| Overweight | 0.86 [0.20] | 0.90 [0.28] | 1.22 [0.38] | 1.58 [0.55] |
| Class 1 Obesity | 1.14** [0.42] | 0.91 [0.46] | 1.43 [0.61] | 1.00 [0.44] |
| Class 2/3 Obesity | 2.65 [1.98] | 1.71 [1.13] | 1.87 [1.06] | 1.56 [0.80] |
| Observations | 2632 | 2211 | 2701 | 2212 |

Note: Table displays hazard rates from a model with log-linear age trend in mortality. The omitted category comprises non-smokers with normal weight (BMI 20-25). Robust standard errors are shown in brackets. + significant at 10%; * significant at 5%; ** significant at 1%