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Abstract

Human capital is now widely recognized to confer numerous benefits, including higher incomes, lower incidence of unemployment, and better health, to those who invest in it. Yet, recent evidence suggests that it also produces larger, social (external) benefits, such as greater aggregate income and productivity as well as lower rates of crime and political corruption. This paper considers whether human capital also delivers external benefits via reduced mortality. That is, after conditioning on various individual-specific characteristics including income and education, do we observe lower rates of mortality in economies with higher average levels of education among the total population? Evidence from a sample of more than 200 U.S. metropolitan areas over the decade of the 1990s suggests that there are significant human capital externalities on health. After conditioning on a variety of city-specific characteristics, the findings suggest that a 5 percentage point decrease in the fraction of college graduates in the population corresponds to a 14 to 40 percent increase in the probability of (all-cause) death, on average. Although I am unable to identify the precise mechanism by which this relationship operates, it is certainly consistent with the idea that interactions with highly educated individuals - who tend to exhibit relatively healthy behaviors - encourage others to adopt similar behaviors. Evidence of a significant inverse relationship between aggregate human capital and smoking, conditional on personal characteristics, in a sample of 226 U.S. metropolitan areas provides additional support for this hypothesis.

JEL Classification: I10, I20, J10, R10
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1 Introduction

The benefits of human capital are numerous and wide-ranging in scope. Individuals who invest in greater levels of education tend to experience better labor market outcomes, including higher earnings and a lower incidence of unemployment, fewer social disorders, such as criminal behavior (Lochner and Moretti (2004)), and better health, measured either in terms of the incidence of disease (Marmot et al. (1978), Marmot et al. (1991), Grossman (2003), Marmot (2004)), or rates of mortality (Kitagawa and Hauser (1968), Christensen and Johnson (1995), Elo and Preston (1996), Marmot (2004), Lleras-Muney (2005)).[1]

Gains from education are not, however, limited to purely internal returns. Indeed, an intriguing literature that has developed over the past two decades suggests that there are also external, or social, benefits as well. Workers living in cities with larger fractions of college educated residents tend, for instance, to have higher labor earnings, even after having conditioned on their own personal characteristics and a variety of city-level covariates (Rauch (1993), Moretti (2004a)). Similarly, firms operating in cities with larger fractions of highly educated workers tend to exhibit significantly higher productivity, even after accounting for the education levels of their own employees (Moretti (2004b)). Education also increases civic participation and the extent to which voters are informed (Dee (2004), Milligan et al. (2004)) which may, in turn, help to reduce political corruption (Glaeser and Saks (2004)). Presumably, this effect benefits society as a whole.

Although the sources of these social benefits remain somewhat nebulous, many would agree that they derive, at least in part, from some sort of social interaction. Highly educated individuals may simply have a positive influence on those with whom they come into contact. For example, less-educated individuals may learn from their encounters at work with their more highly educated co-workers, making them more productive and boosting their earnings (e.g. Glaeser (1999)).[2]

[1] These literatures are vast. For an informative survey of some of the research on the health-education nexus, see Cutler and Lleras-Muney (2006). Marmot (2004) provides a useful survey of some of the relevant work from medicine and epidemiology.

[2] There is also a large literature on neighborhood effects that documents evidence consistent with the notion of peer influences in a variety of behaviors, including school attendance and employment status. See Durlauf (2004) for a survey.

Following the idea that individuals may learn from the highly-educated, this paper explores whether human capital also has an external benefit on the rate of adult mortality. Because mortality rates have been shown to depend crucially on a variety of behaviors, such as smoking, exercise, and choices about nutrition, it is possible that there is a social-learning aspect to the rate at which people die. The presence of large numbers of highly educated individuals, many of whom exhibit relatively healthy behaviors (Cutler and Lleras-Muney (2006)), may serve to increase the incidence of these behaviors among the general population within an economy, thus lowering the overall mortality rate.

While studies linking an individual's own education to his or her own health status (i.e. the 'internal' return) are quite numerous, those looking at the broader social influences of education on health are much less common. Indeed, although a variety of studies within epidemiology have examined the effect of environmental influences on health and mortality (e.g. Berkman and Glass (2000) and Kawachi and Berkman (2000) examine the impact of social networks; Stafford and McCarthy (2006) survey evidence on the effects of neighborhood housing characteristics, social cohesion, and economic conditions on health), little research has examined the explicit role of local human capital, particularly within the economics literature. This neglect is somewhat surprising given that existing evidence has shown education-related interactions to be quite important for influencing health among spouses (e.g. Bosma et al. (2005), Egeland et al. (2002), Monden et al. (2003)). Moreover, substantial evidence suggests that social interactions have an important role in explaining a variety of other social and economic phenomena including crime (Glaeser et al. (1996)), unemployment (Topa (2001)), and school performance (Sacerdote (2001)).

Besides offering evidence on a potentially significant determinant of mortality, an investigation of human capital externalities may also provide further evidence on *how* education influences health. Research on the internal return is often confounded by exogenous differences across individuals in terms of their health-related unobservables (e.g. patience, motivation, strength, innate susceptibility to disease) that may influence both education and health (e.g. Fuchs (1982, 2004)). This issue is not as problematic for investigations of external effects because it is unlikely that one individual's innate characteristics directly affect those of another. Finding evidence of positive human capital externalities on mortality,

therefore, may offer support for theories that appeal to learning about healthy behaviors, which are reasonably transferred from one person to another.[3]

To preview the results briefly, I find that, in a panel of more than 200 U.S. metropolitan areas observed in 1990 and 2000, there is a significantly negative association between mortality rates - conditional on age, race, gender, and education - and the fraction of the local population with at least a bachelor's degree. Although a wide range of point estimates are obtained, the magnitudes suggest that a 5 percentage point decrease in a city's college share may be accompanied by a 14 to 40 percent increase in the rate of death. This effect appears to hold for all demographic groups, but is especially pronounced for those who are relatively young (under 65) and highly educated (at least some post-secondary schooling). When I look at deaths by major cause, the evidence suggests that aggregate human capital lowers the rate of mortality from heart disease, cancer, and to lesser extent cerebrovascular diseases (e.g. stroke). There is considerably less evidence of reductions in mortality from either chronic lower respiratory diseases or unintentional injuries.

What is particularly interesting about the findings is that they show that, although increasing an individual's own education is probably among the most effective ways to improve his or her life expectancy, simply living near highly educated individuals may also have an important effect. Although the results certainly do not prove that a behavioral spillover is the underlying cause of this externality, cursory evidence on rates of smoking from a sample of more than 200 metropolitan areas lends some additional support to the idea. Research, of course, has long shown that the smoking is much more common among less educated individuals than among highly educated individuals (e.g. Zhu et al. (1996), Cutler and Lleras-Muney (2006)).

Using data from the 2006 Behavioral Risk Factor Surveillance System, I find that residents of high-human capital metropolitan areas are significantly less likely to smoke than those in low-human capital metropolitan areas, even after conditioning on numerous personal characteristics including an individual's own education. In particular, a 10 percentage point increase in the share of residents with a bachelor's degree or more tends to be associ-

[3]To be sure, there is an issue of geographic sorting: healthy individuals may simply choose to live near other healthy individuals. This, of course, is an illustration of Manski's (1993) 'reflection problem' in the identification of social influences. The analysis below attempts to address this matter.

ated with a 1.4 to 3 percentage point decline in the likelihood that a given worker smokes. Again, such findings are by no means intended to be definitive, only suggestive. At a minimum, they are consistent with the idea that good health behaviors, as practiced by the highly-educated, diffuse across individuals.

The remainder of the paper proceeds as follows. The next section surveys some theories linking education and mortality and describes what the existence of human capital externalities in mortality might imply. Section 3 provides a brief description of the data. Section 4 presents the empirical findings. Section 5 offers some concluding comments.

2 Explanations for the Effect of Education on Mortality

There are a variety of explanations for the observed decrease in the rate of mortality with an individual's own education. Besides those which appeal to the importance of income, wealth, or other measures of socio-economic status, all of which are directly correlated with education, theories of the 'gradient' typically fall into one of three broad categories.

First, there are behavioral explanations which posit that, through education, individuals learn to live in healthier ways.[4] Empirically, there is ample evidence suggesting that schooling teaches people to value certain types of behavior, such as maintaining a modest and balanced diet, exercising, making routine visits to physicians, and avoiding smoking or excessive alcohol consumption (Kenkel (1991), Cutler and Glaeser (2005), Cutler and Lleras-Muney (2006)). Education may also allow individuals to make better use of advances in medical technology (Goldman and Lakdawalla (2001), Glied and Lleras-Muney (2003)), thereby prolonging their lives.

Second, there are explanations that suggest that the gradient reflects exogenous differences between individuals with respect to their innate patience or durability (e.g. Fuchs (1982)). These differences may lead individuals to live longer, possibly because they make better behavioral decisions or because they are simply less susceptible to disease, and at the same time invest in greater amounts of schooling. By and large, this second class of expla-

[4]Grossman (1972), of course, was among the first to formalize the idea that education and investments in health capital are complementary, hence the acquisition of greater schooling goes hand-in-hand with the adoption of healthy behaviors.

nations holds that there is no causality running between education and mortality. Rather, innate differences between people explain both outcomes.

Third, some have argued that the gradient reflects health's effect on education. That is, we observe an education gradient in mortality because healthy individuals live longer and are better able to invest in education. Evidence reported by Miguel and Kremer (2004) on a de-worming program in Kenya, Bleakley (2007) on hookworm eradication in the United States, and Jayachandran and Lleras-Muney (2007) on Sri Lankan maternal mortality risk offers support for this view. In each case, improvements in health were associated with increases in human capital acquisition measured by school enrollment, attendance, and literacy.

Why, then, might education have an *external* effect on mortality? That is, why would the level of schooling that a particular individual achieves influence the rate at which his or her neighbors die? One possibility, quite simply, is that increases in aggregate human capital tend to boost income and earnings (e.g. Rauch (1993), Moretti (2004a)). Income, of course, tends to correlate significantly (and inversely) with mortality, possibly because it allows people to take advantage of better nutrition, healthier living conditions at home and work, and enhanced access to health care.[5].

Another is that, in highly educated cities, there is a demand for health-inducing amenities from which everyone can benefit. High-human capital metropolitan areas, for example, may have better medical facilities, easier access to outdoor recreation, restrictions on the quantities of various pollutants in the local environment (e.g. automobile exhaust, industrial waste, smoking), or even a greater availability of grocery stores selling healthy foods (Morland et al. (2002)).

A third possible explanation is a learning or imitation effect. With large numbers of highly educated people in an economy, large fractions of the resident population may be exposed to health-inducing behaviors, which they may then emulate. Consider, for example, the case of smoking. In 2005, the National Center for Health Statistics reported that 27

[5]Numerous studies find a negative association between income and mortality (e.g. Duleep (1986), Deaton and Paxson (1999), Smith (1999)). The paper by Snyder and Evans (2006) is a notable exception. Using the so-called 'notch' in the Social Security program as a source of exogenous variation in income, they find a *positive* association between income and mortality.

percent of high school graduates were smokers, as opposed to 9.1 percent of individuals with at least bachelor's degree.[6] As the fraction of college graduates in a city rises, therefore, the proportion of smokers in the local population tends to decline. As a result, there may be increased social pressure in cities with high levels of human capital for residents of all education levels to refrain from smoking.

Although the analysis below attempts to account for the first two explanations by including controls for personal income as well as a variety of metropolitan area-specific characteristics, I am, at this point, unable to completely distinguish between the latter two hypotheses. Doing so would require a much richer set of time-varying city-level data than I employ. The interpretation of the results, therefore, should acknowledge aspects of both conjectures.

3 Data

This paper combines mortality data from the Multiple Cause of Death (MCD) files for various years with Census public use samples. The MCD is assembled by the National Center for Health Statistics and reports basic information about all recorded deaths occurring in the United States during a given year, including the age, race, gender, education, and marital status of the deceased, as well as a detailed description of the cause of death. Crucially for this analysis, the geographic location of each death (occurrence and place of residence of the decedent) is also identified as long as the location is sufficiently large. Metropolitan areas with at least 100000 in population are reported.[7]

Although the Multiple Cause of Death series go back to 1968, the educational attainment of the deceased is first identified in the MCD series in 1988, limiting the scope of the study to the time since then. Studies of human capital externalities, of course, are based upon the estimation of the effect of aggregate education on an individual after controlling directly for his or her own level of education. Hence, this variable is crucial for carrying out the present analysis.

[6] These figures are based on the National Health Interview Survey and are available at www.cdc.gov/nchs/nhis.htm.

[7] The geographic unit of observation throughout the analysis is the metropolitan area. I use the terms 'city' and 'metropolitan area' interchangeably for expositional purposes.

Because I need to combine the MCD files with Census data in order to compute mortality rates, I focus on two years of data: 1990 and 2000. To calculate death rates, I estimate the total number of deaths within groups defined by age, race, gender, and education across a sample of more than 200 metropolitan areas in each year and merge them with estimated population counts for these groups derived from the 5 percent U.S. Census samples of the Integrated Public Use Microdata Series (IPUMS). Mortality rates simply follow as the ratio of deaths to population. In all, I compute death rates for groups defined by three age categories (25 to 44, 45 to 64, 65 and older), three racial groups (white, black, other), two genders (male, female), and five educational categories (no high school, some high school, high school graduate only, some college, bachelor's degree or more) in a total of 206 metropolitan areas in 1990, 234 in 2000. I also use the IPUMS samples to compute a number of group-level characteristics, including median household income, marital status, and labor force participation, to serve as control variables in the analysis.

To provide a sense of what the resulting mortality rates look like, Table 1A lists the resulting mean rates of death for the year 2000 for some of the major demographic groups considered. From them, a number of well-established results are apparent. Men, for example, tend to have higher rates of mortality than women; blacks have higher rates than whites; older individuals face a higher mortality risk than younger individuals; and there is a clear decrease in the likelihood of death as educational attainment rises.

Mortality rates are, however, noticeably lower for individuals whose race is described as 'other.' This category corresponds to a similar grouping in both the Census and Multiple Cause of Death files in the year 1990, where it includes American Indians, Native Alaskans, Chinese, Japanese, Other Asian and Pacific Islanders, and all other unclassified races. In 2000, however, the Census classification also includes individuals reporting *multiple* major races into this category, while the MCD does not do so explicitly. Although mortality rates do tend to be lower for Asians than for either whites or blacks, this features of the data may have created spuriously low rates of death for the 'other' category in 2000 by inflating the population counts in the Census relative to the number of deaths in the MCD.[8]

[8] In 1990, mortality rates for those classified as 'other' were still lower than those for whites and blacks, although the differences were smaller. For example, among women 45 to 64 in 1990, the mortality rates for whites, blacks, and other races were 6.5, 12, and 4.1 per 1000 people. For men, they were 11.5, 20.9, and

A further complication with the 'other' racial category may be the relatively small number of individuals covered. Numbers of deaths tend, in any year, to be somewhat small. Hence, with small populations of non-whites and non-blacks, mortality rates for this racial group may be particularly noisy. To address this matter, Tables A1 and A2 of the Appendix summarize the results obtained from replicating some of the basic estimation with this racial category dropped from the sample. Many of the results turn out to be quite similar to the main results presented below.

Summary statistics describing some of the metropolitan area-level characteristics used in the analysis (e.g. population, population density, industrial composition, aggregate human capital) appear in Table 1B for the year 2000. These are computed as unweighted statistics across the 234 metropolitan areas that appear in the sample for this year. Additional details about the data files used in the analysis are given in the Appendix.

4 Empirical Findings

4.1 Baseline Estimates

Consider the following statistical characterization of the log mortality rate for demographic group i of metropolitan area m in year t:

$$\log\left(\text{MORT}_{imt}\right) = \alpha + \delta_i + \delta_m + \delta_t + \beta X_{imt} + \theta Z_{mt} + \gamma H_{mt} + \epsilon_{imt} \tag{1}$$

where the three δ terms represent group, metropolitan area, and time fixed effects, X_{imt} is a vector of characteristics describing a particular group in a metro area in a given year, Z_{mt} denotes city-time varying characteristics (excluding aggregate education), and H_{mt} is the aggregate human capital of city m in year t. Following the existing literature on human capital externalities (e.g. Moretti (2004a, 2004b)), I measure H_{mt} by the fraction of the local population with at least a bachelor's degree. Groups, i, are defined by three age categories (25 to 44, 45 to 64, 65 or older), three racial categories (white only, black only, other), two genders, and five educational groups (no high school, some high school, high school, some

5.6 per 1000 people.

college, college or more). This categorization implies a potential of 90 demographic groups for each metropolitan area in each year.

The vector of group-city-time varying regressors, X_{imt}, includes three quantities: the logarithm of median household income, the fraction of households that are married, and the mean rate of labor force participation, all of which are derived from IPUMS samples. When considering mortality, income is usually of central interest because it may influence an individual's overall health by affecting the resources (health care, nutrition) he or she has as well as the environment to which he or she is exposed (e.g. neighborhood or place of work). Marital status may either directly affect, or at least capture certain characteristics not otherwise included that affect, mortality because marriage may be associated with more productive or healthy lifestyles. Labor force participation is included to proxy for a variety of characteristics that I am unable to control for directly, such as an individual's health and general level of activity. Individuals that work tend to be healthier (Marmot (2004)), both because illness makes work more difficult (if not infeasible) and possibly because employment boosts health directly by keeping people engaged in society (e.g. Snyder and Evans (2006)). High rates of participation may, therefore, indicate that a particular group within a metropolitan area at a given point in time has a lower rate of mortality.

The vector of city-level characteristics, Z_{mt}, includes a number of features that may influence health and mortality, including overall population, population density, six broad industry employment shares (manufacturing, agriculture-mining-construction, transportation-communications-utilities, wholesale-retail trade, finance-insurance-real estate, services), the unemployment rate, the number of physicians and surgeons per capita, and the number of hospitals per capita. In an effort to gauge the robustness of the estimated association between mortality and average human capital, $\hat{\gamma}$, I estimate several different specifications in which the composition of Z_{mt} is varied. In all cases, mortality rates are expressed in logarithmic form, which is a standard transformation in the analysis of rates.[9]

All other important city-level characteristics for understanding mortality I assume can

[9]Doing so is also, in part, motivated by measures of regression fit which tend to be better when the dependent variable is scaled logarithmically. It also accounts for the (obvious) non-normality of mortality expressed in levels. Estimation in logs, therefore, facilitates making proper inferences from standard statistical estimators.

be reasonably captured by the fixed effects, δ_m. These include any natural amenities that, for example, might be valued more highly by healthy individuals than unhealthy ones, say opportunities for outdoor recreation. Hence, to the extent that individuals of varying health status sort geographically according to temporally fixed city-level characteristics, valid inferences can be drawn from a model in which I account for metropolitan area-specific parameters.

Results from a very basic version of equation (1) appear in Table 2. Although I have controlled for time and metropolitan area effects, these results are based on controlling for group effects, δ_i, by means of dummies for race, gender, interacted race and gender, age group, and educational attainment. Admittedly, this approach is somewhat crude given the heterogeneity in mortality rates across individuals. These results are given primarily to demonstrate that some well-known associations between various characteristics, such as education, and rates of mortality are present in the data that I have constructed by merging the MCD files with the U.S. Census.

The findings clearly show some well-known associations: mortality rates tend to be significantly lower for women relative to men, whites relative to blacks, married individuals relative to those who are single or separated, and younger individuals (25 to 44 and 45 to 64) relative to older individuals (65 or more). The estimates also demonstrate that adults with high levels of schooling (i.e. at least some college) tend to experience lower rates of death than those with less education, which the literature on the internal return to education on mortality has longed stressed. Individuals with only 0 to 8 years of schooling completed, for example, face a 50 to 70 log point (i.e. 70 to 100 percent) higher probability of dying, all else held constant, than workers with at least some post-secondary education. These estimates appear to match reasonably well with the raw statistics reported in Table 1A.

Although income is often cited as a central determinant of health and mortality, I find only weak associations between the rate of mortality within a group and the log median household income of that group. To be sure, the point estimates are negative, suggesting that those with higher incomes face lower rates of death. However, statistically, I am not able to conclude that income has an important association with mortality.

Labor force participation, on the other hand, enters strongly and negatively in all cases,

suggesting that individuals who are able to work (or actively seek work) exhibit better health than those who are not. In part, this may reflect the fact that healthy individuals tend to participate in the labor force more extensively than unhealthy individuals. It may also, however, imply the reverse: work helps to keep individuals socially engaged, thus improving health (e.g. Snyder and Evans (2006)). Whatever the direction of the causation, the intent behind including this variable in the estimation is to provide some proxy for health into the regression to account for its influence on mortality.

As for the metropolitan area-level features, there are several that show significant associations with mortality. Population, most notably, shows a fairly robust negative association, indicating that, conditional on observable characteristics, the inhabitants of large cities are substantially less likely to die than those who live in small urban areas. This result stands in sharp contrast to the estimated effect of urbanization on mortality prior to 1940, which was strongly positive (Haines (2001)). Evidently, cities have become much healthier environments over the past century.

A larger fraction of workers in high-wage (or high-status) sectors, such as finance-insurance-real estate, tends to be associated with lower mortality, whereas larger fractions in low-wage sectors such as services and trade (particularly retail) tend to be accompanied by higher mortality. The unemployment rate correlates negatively with the rate of mortality, which is consistent with Ruhm's (2000) evidence that periods of economic slowdown are associated with improvements in health. The results also indicate that metropolitan areas with larger numbers of physicians and surgeons per capita experience lower death rates overall, which may imply that the availability of medical services is an important determinant of health.[10]

As for evidence on human capital externalities, the findings reveal a strong, inverse relationship between mortality and the fraction of a metropolitan area's population with a bachelor's degree or more. In all four specifications, the resulting coefficients are negative and statistically significant, suggesting that greater numbers of highly educated individuals in a local economy may boost health broadly. To be sure, there is a fair amount of variability in what the point estimates suggest the magnitude of the association is, but all tend to be

[10] Admittedly, there is mixed evidence linking the increased use of medical services to health outcomes. See, for example, Manning et al. (1993).

fairly sizable. A 5 percentage point decrease in the college fraction (approximately one standard deviation in the cross section of cities), for example, may be accompanied by a 13 to 34 log point (14 to 40 percent) increase in the overall rate of mortality, conditional on all other covariates in the model.[11]

To put these figures in the context of some specific metropolitan areas, consider the implied difference between the mortality rates for observationally equivalent workers in cities at the low-end of the human capital distribution (e.g. Brownsville, TX; Yuma, AZ; Altoona, PA; and McAllen, TX) and those at the high-end (e.g. Boston, MA; Washington, D.C.; San Francisco, CA; and Madison, WI). In 2000, the difference in the college completion fractions between these two sets of cities was roughly 20 percentage points. According to the point estimates in Table 2, the implied mortality difference from specification IV is 1.03 log points. That is, the (conditional) mortality rate is on the order of 2.8 times higher in cities at the bottom of the human capital distribution that among those at the top.[12]

4.2 Estimation with Group-Specific Effects

A more complete way to account for the exogenous differences that exist between age-race-gender-education groups with respect to mortality, of course, is to specify a fixed effect for each one. Recall, given that there are three age categories, three race categories, two genders, and five education levels, there are a total of 90 demographic groups in the sample. Attempting to capture differences in the mortality rates across these groups by means of 11 indicators may be insufficient.

In this section, I estimate (1) by giving each group its own intercept. The results appear in Table 3. As before, I have reported estimates from four different specifications of the vector Z_{mt} in an attempt to demonstrate the sensitivity of the aggregate human capital coefficient to the presence (or lack) of the other metropolitan area-level characteristics.

[11] As demonstrated by Table 1A, the percentage-point change in mortality implied by these figures will vary depending on the group considered.

[12] Needless to say, this figure should be interpreted with respect to the standard error in Table 2, which is on the order of 25 percent of the estimate. Still, although the magnitude may seem rather large, it is not completely outlandish. The mortality rate for white high school graduates between 45 and 64 in Yuma, AZ, for example, was more than twice that in Madison, WI in 2000. Clearly, this is just an anecdote, but one that indicates that this estimated differential is not wholly implausible.

On the whole, the results are virtually identical to those from Table 2, suggesting that any possible misspecification in the modeling of personal characteristics given by the simple version of (1) in the previous section is largely unrelated to the metropolitan area-level features considered. As before, a stronger presence of employment in services and trade tends to increase mortality, while more populous cities as well as those with greater numbers of doctors per capita, higher rates of unemployment, and larger employment shares in finance-insurance-real estate or transportation-communications-utilities all display lower mortality.

Similarly, the coefficients on the city-wide college share are little changed from Table 2. In all cases, they are negative and statistically important, suggesting that human capital may indeed influence mortality in an external way.

4.3 Robustness: Two-Way Fixed Effects

Although I have attempted to specify a reasonably detailed set of parameters to capture exogenous mortality differentials between demographic groups (e.g. white male high school dropouts between 45 and 64), the specification still remains somewhat simplistic, particularly in light of the likely heterogeneity that exists in the death rates of individuals in different parts of the country (Chandra and Skinner (2003)). Mortality differs substantially from one part of the country to another, and some of these differences may not be adequately accounted for by simple demographic controls.

There may, for example, be important differences between the members of a given demographic group, i, in different cities, m, or differences in the trends in the mortality rates of groups, i, across time t. College-educated white men between the ages of 25 and 44 who live in Washington DC, for instance, may be quite different from those living in New Orleans. Indeed, the raw statistics certainly suggest that there are important differences. In the year 2000, the mortality rate for white male college graduates between 25 and 44 in Washington DC (a high-human capital city) was 5.9 per 10000 individuals. In New Orleans (a low-human capital city), the rate was more than twice as high: 12.2 per 10000 individuals. If these 'unobserved' differences are correlated with aggregate city-level

education, the estimated coefficient on the college fraction will be biased.[13]

To account for this possibility, this section considers the estimation of the following extension of (1):

$$\log\left(\text{MORT}_{imt}\right) = \alpha + \delta_i + \delta_m + \delta_t + \delta_{im} + \delta_{it} + \delta_{mt} + \beta X_{imt} + \theta Z_{mt} + \gamma H_{mt} + \epsilon_{imt} \quad (2)$$

Here, δ_{im} is a group-metropolitan area effect, δ_{it} is a group-year effect, and δ_{mt} is a metropolitan area-year effect. Unfortunately, it is not possible to estimate all of these parameters because, once we have accounted for a metropolitan area-time effect, δ_{mt}, we cannot identify the association between mortality and any of the time-varying metropolitan area characteristics, Z_{mt} and H_{mt}. Because the latter of these two terms is of fundamental interest in this paper, I assume that δ_{mt} is reasonably well-captured by the inclusion of Z_{mt} and H_{mt}.[14]

Dropping the metropolitan area-time effects and taking 10-year differences within group-metropolitan area observations (i.e. to difference out the δ_{im} terms), equation (2) becomes

$$\Delta\log\left(\text{MORT}_{imt}\right) = \Delta\delta_t + \Delta\delta_{it} + \beta\Delta X_{imt} + \theta\Delta Z_{mt} + \gamma\Delta H_{mt} + \Delta\epsilon_{imt} \quad (3)$$

Given that the sample time frame involves only a single decade, the estimation of (3) only requires a constant ($\Delta\delta_t$) and a set of group-specific fixed effects ($\Delta\delta_{it}$). Regardless of how the group effects are modeled, the resulting estimates of γ are similar. In an effort to be concise, I summarize only the estimated coefficients from the longest specification (IV) of the vector Z_{mt} (see Tables 2 and 3). I find a coefficient (standard error) of -2.88 (0.89) when I use age, race, gender, race-gender interactions, and education dummies to capture group

[13] Controlling for group-city effects should also further account for the sorting of individuals into cities by health status which may confound the identification of social effects (Manski (1993)). Not only does the analysis account for overall city effects, but specific group-city interactions to account for heterogeneous sorting on health across demographic groups (e.g. healthy high school dropouts may sort into healthy and unhealthy cities less than healthy college graduates).

[14] For this particular specification, a total of 201 metropolitan areas are identified in both 1990 and 2000 and, hence, used in the estimation.

effects (as in Table 2); and -2.85 (0.89) when I estimate all 90 group-specific parameters (as in Table 3).[15] Statistically, all are significant at conventional levels of confidence and, although the magnitudes are somewhat smaller than the corresponding values from specifications *IV* in Tables 2 and 3 (i.e. on the order of 55 to 60 percent of the previous estimates), all still suggest economically meaningful associations between aggregate education and mortality.

4.4 Instrumental Variables Estimates

Most studies involving the effects of education, whether internal or external, must confront the issue of endogeneity: unobserved features of individuals that tend to be strongly associated with both the decision to acquire various levels of schooling and an outcome of interest. In the literature examining the internal effect of education on mortality, for example, studies have attempted to account for the fact that individuals who choose to invest in high levels of schooling also tend to have healthier lifestyles, thereby reducing their rates of mortality. They must also address a reverse-causation form of endogeneity: healthier people are better able to go to school, which leads them to acquire greater amounts of education. Typically, studies attempt to circumvent these possibilities by finding an instrument for education which generates exogenous variation in the level of schooling an individual attains, such as the compulsory schooling age within an individual's state of birth (Adams (2002), Lleras-Muney (2005)) or the rate of unemployment in a worker's state during his or her teenage years (Arkes (2001)).

With human capital externalities, the issue is that certain unobserved features of a city may attract individuals who are both well-educated and relatively healthy. In such a case, there will be a negative correlation between a city's mortality rates and its aggregate level of schooling, but the correlation will not imply a *causal* relationship between the two. To some degree, I have already attempted to account for this possibility by including metropolitan area-specific fixed effects in the estimation. As noted previously, these should capture any permanent features or amenities that a location might have which influences the types of individuals who choose to live there. I have also accounted for time-invariant characteristics of particular demographic groups in each city to pick up exogenous differences in the health

[15] Dropping the 'other' racial category generates similar, although somewhat larger, parameter estimates (standard errors): -3.46 (0.92) and -3.47 (0.92).

of specific groups in different parts of the country. However, this approach may still be incomplete because time-invariant effects will not capture short run shocks to a city (e.g. periods of economic expansion or decline, changes in regulations regarding environmental pollutants or smoking) that may influence both the distribution of health and education among its residents.[16]

In order to address this potential problem, I turn to the use of instrumental variables estimation. To do so, I estimate the differenced version of the mortality-human capital relationship given by (3) where I instrument for the change in the college fraction ΔH_{mt} using two variables: (i) a predicted change in the college fraction based on a city's age distribution in 1980, and (ii) an indicator for the presence of a land grant college based on the Morrill Acts of 1862 and 1890. Both instruments were developed by Moretti (2004a) to estimate the effect of aggregate human capital on wages.

The first instrument is constructed from city-level data on the fraction of residents belonging to various age categories in the year 1980, combined with changes in the college fraction for each age category observed at the national level between 1990 and 2000. Hence, the instrument follows as

$$z_m = \sum_a \omega_{am} \Delta h_a \tag{4}$$

where ω_{am} is the fraction of individuals in metropolitan area m who fall into age category a in 1980, and Δh_a is the change in the fraction of college graduates within this age category between 1990 and 2000 in the U.S. as a whole.[17] So, for example, if a represents individuals between 25 and 29 years of age in 1980, Δh_a denotes the change in the college fraction between 1990 and 2000 for individuals who, in 1990, are between 35 and 39 years of age. Changes the fractions of these groups having completed a bachelor's degree is computed

[16] I have, of course, also attempted to control for certain time-varying features that may be associated with the prevalence for healthy people to locate in a particular city, such as numbers of doctors and hospitals per capita. Conditioning on these features should account for the geographic sorting of individuals according to these characteristics.

[17] I use 14 age categories: individuals in 1980 who are 15, 16, 17, 18, 19, 20, 21, 22-24, 25-29, 30-34, 35-44, 45-54, 55-59, 60 or more.

from the Census IPUMS samples.

The plausible exogeneity of this instrument, I argue, derives from the fact that its variation across cities comes entirely from differences in age distributions that exist 10 years prior to the start of the sample time frame. It is, therefore, unlikely to be influenced by shocks occurring between 1990 and 2000 that may have influenced the health distribution of a city (i.e. health after controlling for age-gender-race-education). In other words, I hold that it is unlikely that the age distribution of a metropolitan area in 1980 is, itself, affected by shocks occurring between 1990 and 2000, such as changes in the local economy or regulations on environmental pollutants, which may influence the general level of health in a city.

The second instrument is simply an indicator variable reflecting whether a metropolitan area has a land grant institution based on the Morrill Act of either 1862 or 1890. Here too, it is unlikely that the decision of where to situate a land-grant institution made more than a century ago was directly affected by a mortality-influencing shock occurring between 1990 and 2000.

Importantly, both instruments are highly relevant. A simple regression of the change in a metropolitan area's college fraction on the age distribution-predicted change yields a coefficient (standard error) of 3.95 (1.4). Regressing the change in a city's college fraction on the land grant dummy generates a coefficient (standard error) of 0.006 (0.002). Further tests of instrument relevance are described below.

Results appear in Table 4 for two broad specifications. In the first, the group-specific fixed effects are once again modeled using a set of simple indicators for age, race, gender, race-gender interactions, and education, just as in the estimation reported in Table 2. In the second, I use a set of 90 group dummies to capture these terms. Recall, given that the data are transformed to reflect 10-year differences within group-metropolitan area observations, this specification is similar to that of equations (2) and (3) which account for pairwise fixed effects in mortality.

In both sets of results, the IV estimates produce significantly negative coefficients on the city-wide college fraction. Although the estimate obtained from using the land grant college indicator is quite similar to what we have seen thus far in the OLS estimation, the

age distribution instrument produces a coefficient roughly five times larger in magnitude. Such a finding suggests that, between the two sets of IV results, those obtained from using the land grant indicator are probably more realistic. I base this conclusion not only on the plausibility of the estimated association between mortality and city-wide education, but also on the fact that, when the land grant variable is used, I obtain results similar to those shown in Tables 2 and 3, many of which reinforce those of existing research (e.g. population and doctors per capita enter negatively).[18] In addition, although marginal tests of instrument relevance, reported in the final row of Table 4, indicate that both instruments are highly relevant for explaining changing college shares, the land grant indicator appears to be the stronger of the two.

What is particularly important about these instrumental variables results is that they seem to indicate that there is little evidence of bias in the OLS estimates obtained in the analysis above. Again, the primary concern with the estimation of equations (1) and (3) is that there may be some unobserved component of mortality which is inversely related to education: stochastic elements associated with lower mortality may be correlated with living in a highly educated metropolitan area. Given that the magnitudes of the IV estimates in Table 4 are similar to, or even larger than, those of the OLS estimates in Tables 2 and 3, there appears to be little evidence that the OLS estimates are biased downward.[19]

4.5 Estimates by Age, Education, Gender, and Race

Research on the education gradient in morbidity and mortality suggests that the returns to education may be quite different across different demographic groups (Cutler and Lleras-Muney (2006)). This section extends the analysis to account for any potential heterogeneity in the association between aggregate human capital and the rate of mortality for groups defined by age, education, gender, and race. Different groups, for example, may see greater exposure to highly educated individuals in their neighborhoods or places of work than others, which may generate differences in the extent to which good health behaviors are transmitted.

[18] See, for example, Lleras-Muney (2005).

[19] It is not uncommon for instrumental variables estimates of the effect of education on health and mortality to be larger in magnitude than the corresponding OLS estimates. See Grossman (2004) for a brief survey.

To this end, I return to the estimation of equation (1), in which I use a full set of 90 age-gender-race-education dummies to account for group-specific fixed effects, but allow the coefficient on the city-wide college share to vary between age categories, educational groups, men and women, and races. Given the general lack of evidence from the instrumental variables estimation that the OLS results are strongly biased in any particular direction (especially downward), I have confined the estimation to OLS. The results are reported in Table 5.

Two broad features of the results are immediately apparent. First, across just about every group, the association between mortality and aggregate city-wide human capital is significantly negative. Only in one of the four specifications for whites (the shortest, I) is the estimate not statistically different from zero (although it is negative). This result suggests that human capital externalities may be quite relevant for a broad array of people, not just those belonging to certain demographic groups.

Second, however, the Wald test statistics reported throughout the table demonstrate that there appear to be significant differences in the estimated magnitudes of these associations across different groups. The one exception is the breakdown by gender, where there appears to be no significant difference between men and women in terms of how aggregate education relates to mortality. On the other hand, younger individuals tend to see greater benefits from aggregate education[20], as do racial minorities (especially those belonging to the 'other' category), and individuals with higher levels of schooling. Whatever mechanism underlies the connection between aggregate education and mortality, then, seems to benefit most individuals, but particularly those belonging to these specific groups. As suggested above, if young, well-educated minorities are especially likely to come into contact with college graduates, these findings could be consistent with a learning-based explanation for human capital externalities in health.

[20]This may be consistent with the finding that the education gradient in both mortality and health-related behaviors is strongest for young individuals (Christenson and Johnson (1995), Cutler and Lleras-Muney (2006)).

4.6 Analysis by Cause of Death

One of the advantages of using the Multiple Cause of Death data, in addition to its geographic coverage, is the detailed classification of why individuals die. In this section, I consider how human capital externalities influence deaths attributable to several different causes.

The causes considered are the five most common in the United States: heart disease, malignant neoplasms (cancer), cerebrovascular diseases (e.g. stroke), chronic lower respiratory diseases (e.g. bronchitis, emphysema, asthma), and unintentional injuries (accidents). Of the nearly 2.4 million deaths recorded in the U.S. in 2004, 652486 were attributed to heart disease, 553888 to malignant neoplasms, 150074 to cerebrovascular diseases, 121987 to chronic lower respiratory diseases, and 112012 to accidents (Minino et al. (2007)). Hence, the top five accounted for nearly two-thirds of all deaths in that year.

Other causes of death, by comparison, are rather infrequent. None beyond the top five was responsible for more than 75000 deaths nationwide in 2004, making the calculation of reliable death rates by demographic group at the metropolitan area level largely infeasible for most causes. For this reason, I focus on the top five. Additionally, certain causes of death beyond the top killers (e.g. Alzheimer's disease, influenza, and pneumonia) are problematic for the analysis because the formal classification of deaths via the International Classification of Diseases (ICD) changed between 1990 and 2000.[21]

Unfortunately, estimating mortality rates reliably by cause, even the top five, within groups defined by age, race, gender, and education across more than 200 metropolitan areas poses a significant challenge because the sample sizes are quite small. To look at deaths by type, therefore, I need to resort to another strategy, which proceeds as follows. I calculate for each metropolitan area in each year the total number of deaths from a particular cause for each of five education groups (no high school, some high school, high school only, some college, college or more), and use data from the Census files to translate these counts into rates. While this strategy accounts for the association between education and mortality, it

[21] See Anderson et al. (2001). Each of the top five causes of death has a comparability ratio (i.e. the number of deaths in 1996 classified by the 1990 system (ICD-9) divided by the number of deaths in 1996 classified by the 2000 system (ICD-10)) close to 1. Alzheimer's disease, influenza and pneumonia produce ratios that are substantially different from 1.

neglects the effects of age, race, and gender. To pick up these latter influences, I calculate the percentages of each education group in each city-year that are female, white, black, between 25 and 44 years of age, and between 45 and 64 years of age and include them in the following regression:

$$\log\left(\text{MORT}_{emt}\right) = \alpha + \delta_e + \delta_m + \delta_t + \beta X_{emt} + \theta Z_{mt} + \gamma H_{mt} + \epsilon_{emt} \tag{5}$$

where MORT_{emt} is the overall mortality rate due to a specific cause among the members of education category e in metropolitan area m at year t (e.g. the death rate from cancer among all high school graduates in St. Louis in 1990); X_{emt} is a vector of characteristics describing the members of this educational group (fractions female, white, black, married, 25 to 44, and 45 to 64); Z_{mt} is the same set of city-level characteristics used in the estimation of (1); H_{mt} is the overall college share; and the δ terms represent fixed effects for education groups, metropolitan areas, and years. Because the relationship between each of these elements and the rate of mortality may differ across the five causes considered, I estimate (5) separately for each death type.

The estimated coefficients on the metropolitan area-level college share appear in Table 6. Two columns of results appear: OLS and IV using the land grant indicator. Although the evidence on the endogeneity is of the city-wide college share from Section 4.4 is somewhat weak, mortality rates are constructed slightly differently in this case. As such, I have reported both the OLS and IV results, where the latter is confined to the use of the land grant variable which, as noted previously, generates more plausible results. For the sake of brevity, all other coefficients from the estimation of (5) have been suppressed. Most, as it turns out, have signs consistent with the results already presented (i.e., negative for women and younger individuals, positive for blacks and less educated individuals).

Beginning with the OLS results, we see that there is strong evidence of human capital externalities in three cases: heart disease, malignant neoplasms, and cerebrovascular diseases. We see less evidence of a significant association between aggregate human capital and deaths from either lower respiratory diseases or accidents, although the coefficients are negative for both. The instrumental variables results largely support these conclusions,

although they show the strongest results for cardiovascular disease and cancer.

Do these findings suggest anything about the mechanisms underlying the relationship between aggregate education and mortality? They might, quite simply, indicate that the presence of highly educated individuals largely influences behaviors related to heart disease, cancer, and stroke, such as smoking, diet, and exercise.[22] Behaviors related to external causes of death, particularly accidents, may be less affected by the local human capital stock. To be sure, such a conclusion is highly speculative, and more research is needed to determine the exact channels through which human capital may have an external influence on mortality.

4.7 Education and Health Behavior: The Example of Smoking

In an effort to pursue the 'learning' interpretation that I have given to explain the results so far, I turn to an exploration of tobacco use at this point. Specifically, I consider whether aggregate human capital shows any relationship with the propensity for individuals to smoke. Once again, it is well-documented that smoking tends to decrease dramatically with educational attainment. Greater fractions of college graduates within a local economy may, therefore, serve to deter people of all educational levels from smoking.[23]

Data on smoking behavior by metropolitan area is derived from the Behavioral Risk Factor Surveillance System (BRFSS). The BRFSS was begun in 1984 as a collaborative project of the Centers for Disease Control and Prevention and the states and territories of the US with the goal of collecting information on health and health-related behaviors for individuals 18 years of age or older. While only 15 states participated in 1984, all 50 (as well as the District of Columbia, Puerto Rico, Guam, and the Virgin Islands) were contributing to the survey by 2001. Currently, the BRFSS surveys more than 350,000 adults each year, and, critically for my purposes, reports the metropolitan area of residence for many of the

[22]The evidence documented in the next section on smoking may be consistent with this idea. Of the 420000 deaths attributed to smoking in the U.S. each year, the vast majority, nearly 300000, are formally linked to cancer, heart disease, and cerebrovascular disease (Guide to Clinical Preventive Services, Second Edition; Report of the U.S. Preventive Services Task Force).

[23]Evidence that smoking propensities carry a significant social component is given by Jones (1994), Meara (2001), and Monden et al. (2003).

respondents.

I take data from the 2006 survey, the most recent available as of the writing of this paper, and match them with the year 2000 metro area-level characteristics used in the mortality analysis above. To be sure, doing so clearly introduces a temporal discrepancy between the BRFSS data and the remainder of the covariates. However, metro area-level characteristics in 2006 are likely to be highly correlated with those from 2000. Moreover, using lagged values of the metropolitan area characteristics, such as human capital, may introduce fewer biases associated with endogeneity.

Smoking behavior is determined from the BRFSS variable that sorts individuals into one of four categories: everyday smoker, someday smoker, former smoker, and non-smoker. I define a binary indicator, y_{jm}, equal to 1 if the respondent j of metropolitan area m falls into either of the first two categories, 0 if the respondent falls into either of the latter two. I then consider the following statistical characterization of y:

$$\text{Prob}(y_{jm} = 1) = \Phi(\alpha + \beta X_{jm} + \theta Z_m + \gamma H_m) \tag{6}$$

where X_{jm} is a vector of personal characteristics of the individual (age, age squared, education, race, gender, marital status, indicators for 8 income categories); Z_m denotes a set of metropolitan area-level features (log population, log population density, employment shares for six major industry groupings, the unemployment rate, the numbers of doctors and hospitals per capita); H_m is the city-wide college completion fraction; and $\Phi(.)$ is some function, taken below to be the normal cumulative distribution function. Because I use a single year of data, I am unable to control for metropolitan area-level fixed effects. Doing so would not permit me to identify the key parameter of interest, γ. Nevertheless, because smoking behavior may exhibit significant (exogenous) geographic variation, I include in the analysis a set of state-level dummies based on an individual's place of residence.

After eliminating all observations for which any of the the key variables used in (6) could not be identified, I arrive at a final sample of 166,385 individuals across 226 metro areas. Probit estimates are reported in Table 7.[24] In an effort to gauge the robustness

[24]Observations from the BRFSS are weighted using the 'final weight' in the estimation.

of the estimates, I consider two specifications: one which serves as a baseline (I), and the other which controls for a host of additional metro area-level characteristics (II).

To begin, many of the results reinforce some well-known patterns. We see, for example, that racial minorities tend to smoke less than whites, women smoke less than men, and married individuals tend to smoke less than those without a spouse. There is also, in general, an inverse relationship between smoking and educational attainment, although the estimates suggest that those with 0 to 8 years of education are less likely to smoke than either high school dropouts or high school graduates. This result is similar to what Zhu et al. (1996) find using the National Health Interview Survey. Importantly, however, the results clearly demonstrate that college graduates exhibit the lowest incidence of smoking among all educational groups.

Interestingly, the results also suggest that education may have an external influence on smoking behavior. The estimated coefficient on the metropolitan area-level college fraction is significantly negative across both specifications, and the magnitude, although somewhat small, is far from negligible. In particular, the point estimates indicate that a 10 percentage point increase in a city's population with a bachelor's degree or more tends to be associated with a 1.4 to 3 percentage point decrease in the likelihood that a given individual smokes. This result, I should add, holds after accounting for some of the basic industry structure and population density of the local market, both of which may influence the extent to which people are permitted to smoke (e.g. higher density or larger fractions of office-based employment may be associated with greater limitations on smoking, thereby lowering its incidence in the population).

Are the results consistent across individuals with different levels of education? Based on the education-group specific estimates in Table 8 - found by estimating equation (6) with interactions between the metro area human capital share with five education group dummies - we can see that there are substantial differences across education groups. In particular, the estimated human capital externality on smoking tends to be the largest among the most highly educated. Looking at the longer of the two specifications, a 10 percentage point increase in the college fraction tends to be accompanied by a 4.2 percentage point decrease in the likelihood that a college graduate smokes, a 3 percentage point decrease

in the probability that someone with some post-secondary education smokes, and a 2.7 percentage point drop in the frequency with which a high school graduate smokes. For the bottom two education groups, the estimated associations are negative, although statistically negligible.

Two aspects of these findings are particularly interesting. First, the education group specific externalities on smoking mimic those on mortality, at least in the sense that they tend to be the greatest among individuals with the highest levels of schooling. For some reason, as the general level of education in a metropolitan area increases, smoking and mortality rates tend to decline, but do so most among college graduates. One possible explanation is that the most highly-educated may learn more readily about (and adopt) the healthy behaviors exhibited by those around them than the less-educated. This explanation, of course, is complementary to those suggesting that the internal education-mortality gradient may be related to the highly-educated adapting to new medical technologies better than the less-educated (e.g. Glied and Lleras-Muney (2003)).

Second, although largest among college graduates, the estimated human capital externality on smoking is statistically important for the vast majority of individuals. Given that nearly 86 percent of the U.S. population holds at least a high school diploma, the results suggest that increases in a metropolitan area's general level of education may be beneficial (in the context of smoking) to a substantial fraction of its residents.

5 Concluding Discussion

While many studies have shown that there appears to be a strong internal return to education on the probability of dying, this paper has shown that there also appears to be evidence of a social return. After controlling for numerous individual-specific characteristics, rates of mortality tend to be lower in highly-educated cities than in less-educated cities.

By and large, I have interpreted the results in terms of learning and social interactions, and the results are certainly consistent with such an interpretation. Again, because highly educated individuals tend to exhibit healthier behaviors, including lower rates of tobacco use, a larger presence of college graduates within a local market may reinforce these behaviors among the resident population.

The results are, however, also consistent with an alternative hypothesis. Particularly healthy individuals, of all demographic groups, may simply value living near highly educated individuals. That is, college graduates may themselves be an important amenity for people who are naturally predisposed to be healthier and live longer. It can certainly be argued that individuals tend to move in an effort to surround themselves with those who possess similar values.

To some degree, of course, I have attempted to account for this possibility by conditioning on both city and group-city fixed effects in mortality. However, the health composition of a given demographic group within a city may still change along with the aggregate human capital of that city. In this paper, I am unable to control for such a possibility, and doing so would be an important matter for future research to pursue.

Future work should also take better account of city-level characteristics that may be correlated with aggregate human capital. While I have attempted to control for the influence of certain time-varying features, such as industry composition, overall city scale, and the presence of medical services, a much more detailed set of covariates, including occupational shares, the presence of environmental hazards, and measures of the quality (rather than the raw quantity) of healthcare personnel and facilities could be considered.

In addition, because the extent to which an individual benefits from aggregate human capital may depend on how much interaction he or she has with the highly-educated, measures of segregation by education might be important. For example, if the external benefits of human capital are experienced primarily at the neighborhood level (e.g. peer effects in behavior may rely on interactions in a residential area; or healthy amenities, such as markets with fresh produce, may be concentrated near highly educated individuals), externalities are likely to be more widely dispersed in cities in which college graduates are spread out than they are in cities where college graduates tend to cluster in relatively few areas.

Interestingly, one trend that the results may cast some further light on is the rise in the education gradient in recent decades (e.g. Pappas et al. (1993), Crimmins and Saito (2001), Marmot (2004)). Many researchers have observed that the internal return to education in terms of reduced morbidity and mortality has become more pronounced over time. While there are a number of reasonable explanations, including rising financial returns to education

and an increase in the amount of education required to take advantage of certain medical technologies, it should be added that the geographic distribution of human capital may have played some role in this trend.

Indeed, research by Moretti (2004c), Berry and Glaeser (2005), and Wheeler (2006) finds that college graduates have become increasingly concentrated within relatively few metropolitan areas. Using a sample of 188 metropolitan areas identified in the IPUMS Census samples, Wheeler (2006) finds that the standard deviation of the city-level college completion fraction rose from 0.043 to 0.064 between 1980 and 2000. This increase was generated by a general spreading out of the distribution: over this period, the increase in the college completion rate at the 90th percentile of distribution of city-level college shares outpaced that of the median (10 percentage points versus 7 percentage points), which itself was faster than that of the 10th percentile (5 percentage points).

Not only does the evidence from Table 5 suggest that the most highly educated benefit the most from human capital externalities, but the divergence in human capital across cities in recent decades implies that college graduates have become increasingly exposed to such externalities. As a result, it is perhaps not surprising that we have seen a growing disparity between the mortality rates of the less-educated and the highly-educated in recent decades.

Table 1A: Summary Statistics - Mortality Rates, 2000

Group	Age 25 to 44		Age 45 to 64		Age 65 or Older	
	Men	Women	Men	Women	Men	Women
White	2.2 (1.2)	1.2 (0.7)	9.1 (7.3)	5.6 (4.2)	64.2 (49.4)	55.7 (43.6)
Black	3.9 (2.9)	2.8 (3.7)	15.2 (12.7)	10.4 (9.3)	82.3 (84.7)	60.9 (48.7)
Other	0.7 (0.8)	0.5 (0.5)	3 (3.2)	2 (2.6)	19.7 (22.4)	15.4 (16.8)
No High School	4.3 (4.4)	3.1 (4.4)	16.4 (10.9)	11.2 (8.9)	86.9 (45.6)	80.1 (55.6)
Some High School	5 (5.9)	3 (2.7)	16.8 (11.2)	10.2 (9.1)	65.4 (63.5)	49 (32.1)
High School	3 (1.7)	1.6 (1.1)	12.3 (9.9)	7 (5.1)	66.6 (53.7)	51 (42.9)
Some College	1.2 (0.7)	0.7 (0.5)	5.2 (4.3)	3.4 (2.7)	38.5 (29.9)	36.6 (41.8)
College	1 (0.7)	0.7 (0.6)	5.2 (8)	3.3 (4.7)	49 (110.8)	43.6 (57)
White-No High School	7.1 (11.5)	4.5 (5.5)	19.9 (10.6)	14.9 (9.4)	95.7 (44.1)	91.6 (65.7)
White-Some High School	5.5 (5.1)	3.3 (2.4)	17.9 (10.7)	11.1 (10.5)	69.1 (63.4)	51.6 (32.3)
White-High School	3.1 (1.8)	1.6 (1.1)	12.5 (9.4)	6.9 (4.7)	67.1 (51)	51.8 (43.6)
White-Some College	1.2 (0.6)	0.7 (0.4)	5.3 (4.6)	3.3 (2.7)	39 (30.6)	37.4 (43.4)
White-College	1 (0.7)	0.6 (0.5)	5.2 (7.6)	3.1 (4.1)	50 (11.7)	44.2 (55.8)
Black-No High School	10.8 (11.9)	8 (9.3)	29 (41.8)	19.9 (2.3)	92.7 (89)	70 (51.4)
Black-Some High School	8.9 (17.5)	6.9 (15.1)	28.4 (33.9)	15.6 (17.3)	75.7 (102.9)	50.8 (47.6)
Black-High School	5.1 (5.3)	3.9 (5.6)	21.1 (23.9)	12.6 (11.6)	96.8 (185.9)	67.9 (72.7)
Black-Some College	2.4 (1.7)	2 (4.1)	8.3 (8)	6.3 (5.2)	56.6 (66.9)	49.7 (75.7)
Black-College	4.3 (6.3)	3 (5.4)	12.2 (15.3)	11.3 (3.3)	59.7 (65.5)	59 (89.4)
Other-No High School	0.8 (1.2)	1.8 (3.3)	6.1 (10.8)	4.8 (7.2)	22.9 (33.3)	18.8 (19.3)
Other-Some High School	2.9 (7.2)	2.3 (3)	6.8 (8.3)	7.9 (17.3)	20.8 (17.2)	23.4 (70.4)
Other-High School	1.8 (2.8)	1 (1.1)	5.9 (8.7)	3.7 (5.6)	29.2 (25.7)	17.6 (17.6)
Other-Some College	1 (1.6)	0.7 (1.1)	5.7 (16.8)	3 (6)	28.3 (40.1)	19.4 (30.8)
Other-College	0.9 (1)	1.1 (1.7)	4.4 (4.7)	3.5 (8.4)	30.6 (35.7)	20.5 (16.6)

Note: Mean death rates (standard deviations), reported in deaths per 1000 individuals, calculated as ratios of deaths to Census population counts.

Table 1B: Summary Statistics - Metropolitan Areas, 2000

Variable	Mean	Standard Deviation	Minimum	Maximum
Population	948616.3	2056611	104646	19397717
Density	577.1	1138.7	29	15273.6
% College	16.3	4.8	6.6	29
% Manufacturing	8.6	4	1.7	25.8
% Ag.-Min.-Con.	5.8	1.6	3.2	12.3
% Tran.-Com.-Util.	3.8	0.9	1.8	7.4
% Trade	13.4	1.4	10.8	18.2
% FIRE	3.4	1.3	1.5	12.4
% Services	21.7	3.5	13.8	34.1
Unemployment Rate	0.042	0.016	0.015	0.115
Doctors Per Capita	0.003	0.001	0.0007	0.016
Hospitals Per Capita	0.00004	0.00001	0.000006	0.00006

Note: Unweighted statistics taken over 234 metropolitan areas.

Table 2: Parameter Estimates - Basic Specification

	Specification			
Variable	*I*	*II*	*III*	*IV*
Female	-0.58* (0.02)	-0.58* (0.02)	-0.58* (0.02)	-0.58* (0.02)
Black	0.31* (0.02)	0.31* (0.02)	0.31* (0.02)	0.31* (0.02)
Other	-0.86* (0.03)	-0.86* (0.03)	-0.86* (0.03)	-0.86* (0.03)
Female*Black	0.07* (0.02)	0.07* (0.02)	0.07* (0.02)	0.07* (0.02)
Female*Other	0.06* (0.035)	0.06* (0.035)	0.06* (0.035)	0.06* (0.035)
Age 25-44	-2.68* (0.03)	-2.67* (0.03)	-2.67* (0.03)	-2.67* (0.03)
Age 45-64	-1.35* (0.03)	-1.35* (0.03)	-1.35* (0.03)	-1.34* (0.03)
Some High School	-0.07* (0.02)	-0.07* (0.02)	-0.07* (0.02)	-0.07* (0.02)
High School	-0.14* (0.02)	-0.14* (0.02)	-0.14* (0.02)	-0.14* (0.02)
Some College	-0.7* (0.02)	-0.71* (0.02)	-0.71* (0.02)	-0.71* (0.02)
College	-0.52* (0.03)	-0.53* (0.03)	-0.53* (0.03)	-0.53* (0.03)
% Married	-0.38* (0.04)	-0.37* (0.04)	-0.37* (0.04)	-0.37* (0.04)
Log Median Income	-0.02 (0.02)	-0.01 (0.02)	-0.003 (0.02)	-0.003 (0.02)
Labor Force Participation	-0.54* (0.05)	-0.56* (0.05)	-0.56* (0.05)	-0.56* (0.05)
Log Population	–	-1.01* (0.12)	-0.75* (0.13)	-0.65* (0.15)
Log Density	–	-0.05 (0.13)	0.01 (0.14)	0.09 (0.14)
% Manufacturing	–	–	0.81 (1.01)	-0.44 (1.01)
% Ag.-Min.-Con.	–	–	-5.35* (1.76)	-5.6* (1.8)
% Tran.-Com.-Util.	–	–	-23.9* (3.1)	-22.7* (3.1)
% Trade	–	–	8.6* (1.4)	8.4* (1.4)
% FIRE	–	–	-9.9* (2.4)	-11* (2.4)
% Services	–	–	10.6* (1.1)	10.6* (1.2)
Unemployment Rate	–	–	-5.1* (1.2)	-4.9* (1.2)
Doctors Per Capita	–	–	–	-66.9* (14.8)
Hospitals Per Capita	–	–	–	12604.5* (1762.2)
% College	-2.56* (0.76)	-3* (0.79)	-6.75* (0.99)	-5.13* (1.03)
R^2	0.77	0.77	0.78	0.78

Note: Dependent variable is log mortality rate. 25692 observations. All specifications include a time effect and metropolitan area fixed effects. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table 3: Results with Group-Level Effects

	Specification			
Variable	*I*	*II*	*III*	*IV*
% Married	-0.02 (0.05)	-0.005 (0.05)	0.004 (0.05)	0.0002 (0.05)
Log Median Income	-0.03 (0.02)	-0.02 (0.02)	-0.02 (0.02)	-0.02 (0.02)
Labor Force Participation	-0.05 (0.05)	-0.07 (0.06)	-0.07 (0.06)	-0.07 (0.06)
Log Population	–	-0.99* (0.12)	-0.74* (0.12)	-0.66* (0.14)
Log Density	–	-0.08 (0.12)	-0.02 (0.13)	0.06 (0.14)
% Manufacturing	–	–	0.53 (0.95)	-0.67 (0.95)
% Ag.-Min.-Con.	–	–	-5.3* (1.6)	-5.5* (1.7)
% Tran.-Com.-Util.	–	–	-23.9* (2.9)	-22.7* (2.9)
% Trade	–	–	8.7* (1.3)	8.4* (1.3)
% FIRE	–	–	-10.7* (2.3)	-11.8* (2.3)
% Services	–	–	10.7* (1.05)	10.7* (1.1)
Unemployment Rate	–	–	-5.03* (1.1)	-4.8* (1.1)
Doctors Per Capita	–	–	–	-66.2* (13.8)
Hospitals Per Capita	–	–	–	12096.1* (1685.1)
% College	-2.53* (0.7)	-3.01* (0.73)	-6.7* (0.93)	-5.16* (0.97)
R^2	0.8	0.8	0.81	0.81

Note: Dependent variable is log mortality rate. 25692 observations. All specifications include a time effect, metropolitan area fixed effects, and group effects for 90 age-gender-race-education categories. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table 4: Instrumental Variables Results

	Simple Indicators		Complete Group Effects	
Variable	Land Grant Instrument	Age Distribution Instrument	Land Grant Instrument	Age Distribution Instrument
Female	0.09* (0.02)	0.09* (0.02)	–	–
Black	-0.22* (0.03)	-0.19* (0.03)	–	–
Other	-0.61* (0.04)	-0.59* (0.04)	–	–
Female*Black	0.04 (0.04)	0.04 (0.04)	–	–
Female*Other	-0.03 (0.05)	-0.03 (0.05)	–	–
Age 25-44	-0.07* (0.02)	-0.06* (0.02)	–	–
Age 45-64	-0.04* (0.02)	-0.04* (0.02)	–	–
Some High School	0.06* (0.03)	0.06* (0.03)	–	–
High School	0.02 (0.03)	0.02 (0.03)	–	–
Some College	-0.08* (0.03)	-0.08* (0.03)	–	–
College	-0.17* (0.03)	-0.16* (0.03)	–	–
Δ % Married	-0.03 (0.07)	-0.03 (0.07)	0.02 (0.07)	0.02 (0.07)
Δ Log Median Income	0.02 (0.03)	0.02 (0.03)	-0.005 (0.03)	-0.006 (0.03)
Δ Labor Force Participation	-0.02 (0.06)	0.01 (0.06)	0.03 (0.06)	0.06 (0.06)
Δ Log Population	-0.35* (0.12)	0.4* (0.18)	-0.32* (0.12)	0.4* (0.18)
Δ Log Density	-0.1 (0.12)	-0.6* (0.14)	-0.11 (0.12)	-0.63* (0.14)
Δ % Manufacturing	-1.95* (0.75)	-2.8* (0.79)	-1.94* (0.74)	-2.74* (0.78)
Δ % Ag.-Min.-Con.	-4.1* (1.3)	-4.9* (1.4)	-3.8* (1.3)	-4.63* (1.4)
Δ % Tran.-Com.-Util.	-17.6* (2.9)	-30.6* (3.7)	-17.2* (2.9)	-29.9* (3.7)
Δ % Trade	5.2* (1.2)	0.41 (1.3)	5.2* (1.2)	0.54 (1.3)
Δ % FIRE	-13.8* (2.8)	2.2 (3.4)	-13.7* (2.8)	1.9 (3.3)
Δ % Services	8.7* (1.7)	19.7* (2.3)	8.8* (1.7)	19.6* (2.3)
Δ Unemployment Rate	-2.8* (0.9)	-6.1* (1.2)	-3.04* (0.9)	-6.2* (1.2)
Δ Doctors Per Capita	-53.6* (16)	35.7* (19.8)	-52.6* (15.9)	34.6* (19.6)
Δ Hospitals Per Capita	8144.5* (1573.1)	4777.4* (1473.6)	8313.2* (1555.7)	5014.2* (1459.7)
Δ % College	-5.53* (3.04)	-27.4* (4.2)	-5.48* (3.02)	-26.9* (4.2)
F-test statistic	1299.3 (0)	719.3 (0)	1294.4 (0)	717 (0)

Note: Dependent variable is the change in the log mortality rate between 1990 and 2000. 12573 observations. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better. 'F-test statistic' reports test of marginal significance of the instrument in regressions of the change in the college fraction on all variables.

Table 5: Estimates by Age, Education, Gender, and Race

	Specification			
Variable	*I*	*II*	*III*	*IV*
% College*Age 25-44	-3* (0.73)	-3.5* (0.75)	-7.24* (0.96)	-5.67* (0.99)
% College*Age 45-64	-2.98* (0.72)	-3.45* (0.74)	-7.2* (0.94)	-5.6* (0.98)
% College*Age 65 or more	-1.72* (0.7)	-2.2* (0.73)	-5.96* (0.93)	-4.36* (0.97)
Wald test statistic	22.04 (0)	21.6 (0)	21.7 (0)	22.2 (0)
% College*No High School	-1.9* (0.7)	-2.35* (0.75)	-6.06* (0.95)	-4.5* (0.99)
% College*Some High School	-1.6* (0.7)	-2.06* (0.75)	-5.78* (0.95)	-4.2* (0.99)
% College*High School	-1.86* (0.7)	-2.37* (0.74)	-6.09* (0.94)	-4.53* (0.98)
% College*Some College	-2.45* (0.7)	-2.9* (0.75)	-6.61* (0.95)	-5.04* (0.98)
% College*College	-4.6* (0.7)	-5.1* (0.75)	-8.79* (0.95)	-7.22* (0.99)
Wald test statistic	34.4 (0)	34.9 (0)	34.3 (0)	34.2 (0)
% College*Female	-2.45* (0.7)	-2.94* (0.7)	-6.68* (0.94)	-5.09* (0.98)
% College*Male	-2.6* (0.7)	-3.07* (0.7)	-6.81* (0.94)	-5.23* (0.98)
Wald test statistic	0.7 (0.39)	0.5 (0.48)	0.52 (0.47)	0.56 (0.45)
% College*White	-1.06 (0.7)	-1.59* (0.73)	-5.2* (0.93)	-3.61* (0.97)
% College*Black	-2.7* (0.72)	-3.2* (0.74)	-6.7* (0.94)	-5.2* (0.98)
% College*Other	-4.7* (0.8)	-5.05* (0.82)	-8.5* (1)	-6.9* (1.03)
Wald test statistic	66.2 (0)	61.5 (0)	58.1 (0)	57.2 (0)

Note: Coefficients on interations between age, education, gender, and race indicators and metropolitan area college shares. Dependent variable is log mortality rate. Specifications *I* - *IV* refer to those in Tables 2 and 3. All include a time effect, metropolitan area fixed effects, and group effects for 90 age-gender-race-education categories. 'Wald test statistic' reports test of null that all coefficients within each set are equal (p-value under null in parentheses). Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table 6: Estimates by Cause of Death

Cause	OLS	IV (Land Grant)
Heart Disease	-13.3*	-25.2*
	(2.5)	(11)
Malignant Neoplasms	-10.9*	-19.6*
	(2.6)	(11.6)
Lower Respiratory Diseases	-0.87	-8.8
	(2.5)	(8.6)
Cerebrovascular Diseases	-4.6*	-12.5
	(2.5)	(9.3)
Accidents	-0.6	4.4
	(2.3)	(9.8)

Note: Coefficients on the city-wide college share. OLS specification is estimated in levels with fixed effects for time and metropolitan areas. IV estimation is performed in 10-year differences of all variables. Regressions also include shares of each group that are female, white, black, between 25 and 44, between 45 and 64, and indicators for education group. All city-level characteristics from specification IV from Tables 2 and 3 are included. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table 7: Correlates of Smoking

Variable	*I*	*II*
Age	0.016* (0.001)	0.016* (0.001)
Age squared	-0.02* (0.001)	-0.02* (0.001)
Some High School	0.11* (0.02)	0.11* (0.02)
High School	0.036* (0.017)	0.036* (0.017)
Some College	-0.01 (0.01)	-0.01 (0.01)
College	-0.1* (0.01)	-0.1* (0.01)
Married	-0.1* (0.005)	-0.1* (0.005)
Black	-0.06* (0.005)	-0.06* (0.005)
Hispanic	-0.08* (0.007)	-0.08* (0.007)
Other Race	-0.02* (0.01)	-0.02* (0.01)
Female	-0.05* (0.004)	-0.05* (0.004)
Log Population	–	0.006 (0.006)
Log Density	–	-0.001 (0.007)
% Manufacturing	–	-0.01 (0.09)
% Ag.-Min.-Con.	–	0.13 (0.25)
% Tran.-Com.-Util.	–	-0.1 (0.25)
% Trade	–	-0.1 (0.25)
% FIRE	–	0.17 (0.2)
% Services	–	0.11 (0.14)
Unemployment Rate	–	-0.7* (0.33)
Doctors Per Capita	–	-0.68 (2.8)
Hospitals Per Capita	–	361.5 (353)
% College	-0.14* (0.04)	-0.3* (0.1)
Log Likelihood	-72743.3	-72724.5

Note: Dependent variable is indicator equal to 1 if respondent reports smoking at all, 0 otherwise. Both specifications also include indicators for 8 income categories and fixed effects for state of residence. Probit estimates are reported as estimated marginal associations at the mean values of the variables. 166385 observations. Coefficients and standard errors on age squared have been multiplied by 100. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table 8: Correlates of Smoking - By Education Group

Variable	*I*	*II*
% College*No High School	0.07 (0.2)	-0.1 (0.22)
% College*Some High School	0.13 (0.14)	-0.03 (0.17)
% College*High School	-0.12* (0.06)	-0.27* (0.11)
% College*Some College	-0.16* (0.06)	-0.3* (0.11)
% College*College	-0.28* (0.07)	-0.42* (0.11)

Note: Dependent variable is indicator equal to 1 if respondent reports smoking at all, 0 otherwise. Models are the same as those reported in Table 7. Probit estimates are reported as estimated marginal associations at the mean values of the variables. 166385 observations. Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

A Appendix

A.1 Additional Data Details

There are two primary data sources used in the analysis of death rates. The Multiple Cause of Death (MCD) files, prepared by the National Center for Health Statistics, records all deaths occurring in the United States each year. The series began in 1968, but the educational attainment of the decedent was only recorded beginning with the introduction of a revised U.S. Standard Certificate of Death for use by the states in 1989. The 1990 MCD file consists of 2151890 recorded deaths, 1593438 of which took place in a metropolitan area that could be identified in the data. Of these, the numbers of deaths by cause were 526861 (heart disease), 376741 (malignant neoplasms), 62739 (chronic lower respiratory diseases), 102558 (cerebrovascular diseases), 45881 (accidents). For 2000, there were 2407193 recorded deaths, 1828346 of which were in an identifiable metropolitan area. Deaths by cause in 2000 were 538634 (heart disease), 424257 (malignant neoplasms), 91160 (chronic lower respiratory diseases), 125334 (cerebrovascular diseases), 70152 (accidents). Total numbers of deaths per age-race-gender-education group across the metropolitan areas in the sample averaged 82.7 (minimum = 1, maximum = 13422) in 1990, 91.5 (minimum = 1, maximum = 29914) in 2000. Total numbers of deaths by education group and cause across all metro areas in the sample averaged 133.3 (minimum = 1, maximum = 12372) in 1990, 160.6 (minimum = 1, maximum = 28117) in 2000.

The 5 Percent samples of the 1990 and 2000 Census of Population and Housing are obtained from the Integrated Public Use Microdata Series (IPUMS) at www.ipums.org (Ruggles et al. (2004)). The 1990 sample consists of 12501046 person records, 7983731 of which are individuals who are 25 years of age or older and reside in an identified metropolitan area. The 2000 file has 14081466 person records and 9144986 adults in a metropolitan area. These files are used to compute populations of individuals in each age-race-gender-education group in each city in the construction of death rates. Income used in the analysis is given by the median household income of the relevant demographic group in the corresponding metropolitan area, expressed in year 2000 dollars using the Personal Consumption Expenditure Chain-Type Price Index from the National Income and Product Accounts. Because I take medians, I do not transform the top- and bottom-coded income values reported in the Census.

Population density is calculated for each metropolitan area as a weighted average of county-level population densities, where the weights are the fraction of each metro area's population residing in each constituent county. Land area at the county level is derived from the USA Counties data files of the U.S. Census Bureau. The number of doctors in each metro area is derived from the IPUMS samples based on the reported occupational data, and the number of hospitals comes from County Business Patterns for the years 1990 and 2000 (U.S. Census Bureau). Unemployment rates are based on county-level unemployment estimates from the Local Area Unemployment Statistics program of the Bureau of Labor Statistics.

A.2 Geographic Areas

Metropolitan areas (or 'cities') are defined as either metropolitan statistical areas (MSAs) or consolidated metropolitan statistical areas (CMSAs) using definitions from 1993. Although CMSAs are extremely large geographically, and therefore may seem inappropriate when discussing interactions and externalities among individuals, their use greatly facilitates the creation of metropolitan areas with (reasonably) consistent definitions between 1990 and 2000. For example, individuals assigned to certain metropolitan areas within a CMSA in one year are sometimes assigned to another in the next year, simply based on changing geographic definitions. Aggregating metro areas within CMSAs (i.e. primary MSAs) to the CMSA level circumvents this problem.

Table A1: Estimates with Whites and Blacks Only

	Specification			
Group Effects	*I*	*II*	*III*	*IV*
Simple Indicators	-3.4* (0.72)	-3.9* (0.75)	-7.7* (0.95)	-5.99* (0.99)
Indicators for 90 Groups	-3.3* (0.67)	-3.82* (0.7)	-7.5* (0.9)	-5.89* (0.93)

Note: Estimated coefficients on the city-wide college fraction from the estimation of (1). Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

Table A2: Estimates by Age, Education, Gender, and Race

Whites and Blacks Only

	Specification			
Variable	*I*	*II*	*III*	*IV*
% College*Age 24-44	-3.6* (0.7)	-4.1* (0.71)	-7.8* (0.91)	-6.2* (0.95)
% College*Age 45-64	-3.7* (0.68)	-4.2* (0.7)	-7.9* (0.9)	-6.3* (0.94)
% College*Age 65 or more	-2.67* (0.67)	-3.2* (0.7)	-6.9* (0.89)	-5.3* (0.9)
Wald test statistic	14.2 (0)	14.1 (0)	13.8 (0)	14.2 (0)
% College*No High School	-2.84* (0.7)	-3.3* (0.7)	-7.03* (0.9)	-5.4* (0.95)
% College*Some High School	-2.3* (0.7)	-2.8* (0.7)	-6.5* (0.9)	-4.86* (0.94)
% College*High School	-2.77* (0.68)	-3.3* (0.7)	-7.06* (0.9)	-5.43* (0.94)
% College*Some College	-3.2* (0.7)	-3.7* (0.7)	-7.4* (0.9)	-5.74* (0.94)
% College*College	-5.3* (0.7)	-5.8* (0.7)	-9.5* (0.9)	-7.91* (0.95)
Wald test statistic	34.5 (0)	35.1 (0)	34.7 (0)	34.7 (0)
% College*Female	-3.2* (0.67)	-3.74* (0.7)	-7.46* (0.9)	-5.82* (0.93)
% College*Male	-3.39* (0.68)	-3.89* (0.7)	-7.61* (0.9)	-5.98* (0.93)
Wald test statistic	1 (0.31)	0.67 (0.41)	0.73 (0.39)	0.77 (0.38)
% College*White	-2.48* (0.67)	-3.01* (0.7)	-6.7* (0.9)	-5.05* (0.93)
% College*Black	-4.23* (0.68)	-4.75* (0.71)	-8.4* (0.9)	-6.74* (0.94)
Wald test statistic	46.6 (0)	48.2 (0)	65.8 (0)	51.8 (0)

Note: Coefficients on interactions between age, education, gender, and race indicators and metropolitan area college shares. Dependent variable is log mortality rate. Specifications *I* - *IV* refer to those in Tables 2 and 3. All include a time effect, metropolitan area fixed effects, and group effects for 90 age-gender-race-education categories. 'Wald test statistic' reports test of null that all coefficients within each set are equal (p-value under null in parentheses). Heteroskedasticity-consistent standard errors are reported in parentheses. An asterisk (*) denotes statistical significance at 10 percent or better.

References

Adams, S. (2002) "Educational Attainment and Health: Evidence from a Sample of Older Adults." *Education Economics*. 10, 97-109.

Anderson, R., A. Minino, D. Hoyert, and H. Rosenberg. (2001) "Comparability of Cause of Death Between ICD-9 and ICD-10: Preliminary Estimates." *National Vital Statistics Reports*. 49 (2). 1-32.

Arkes, J. (2001) "Does Schooling Improve Adult Health?" RAND Working Paper.

Berkman, L. and T. Glass. (2000) "Social Integration, Social Networks, Social Support, and Health." In *Social Epidemiology*. L. Berkman and I. Kawachi eds. New York: Oxford University Press. 137-173.

Berry, C. and E. Glaeser. (2005) "The Divergence of Human Capital Levels Across Cities." *Papers in Regional Science*. 84, 407-444.

Bleakley, H. (2007) "Disease and Development: Evidence from Hookwork Eradication in the American South." *Quarterly Journal of Economics*. 122 (1), 73-117.

Bosma, H., A. Appels, F. Sturmans, V. Grabauskas, A. Gostautas. (1995) "Educational Levels of Spouses and Risk of Mortality." *International Journal of Epidemiology*. 24, 119-126.

Chandra, A. and J. Skinner. (2003) "Geography and Racial Health Disparities." NBER Working Paper No. 9513.

Christensen, B. and N. Johnson. (1995) "Educational Inequality in Adult Mortality: An Assessment with Death Certificate Data from Michigan." *Demography*. 32 (2), 215-229.

Crimmins, E. M. and Y. Saito. (2001) "Trends in Healthy Life Expectancy in the United States, 1970-1990: Gender, Racial, and Educational Differences." *Social Science and Medecine*. 42, 47-57.

Cutler, D. and A. Lleras-Muney. (2006) "Education and Health: Evaluating Theories and Evidence." NBER Working Paper No. 12352.

Cutler, D. and E. Glaeser (2005) "What Explains Differences in Smoking, Drinking and Other Health-Related Behaviors." NBER Working Paper No. 11100.

Deaton, A. and C. Paxson. (1999) "Mortality, Education, Income, and Inequality among American Cohorts." NBER Working Paper No. 7140.

Dee, T. (2004) "Are There Civic Returns to Education?" *Journal of Public Economics*. 88, 1697-1720.

Duleep, H. (1986) "Measuring the Effect of Income on Adult Mortality Using Longitudinal Adminstrative Record Data." *Journal of Human Resources*. 21 (2), 238-251.

Durlauf, S. (2004) "Neighborhood Effects." In *Handbook of Regional and Urban Economics*. Vol. 4. J. Henderson and J. Thisse eds. New York: Elsevier.

Egeland, G., A. Tverdal, H. Meyer, and R. Selmer. (2002) "A Man's Heart and a Wife's Education: A 12-Year Coronary Heart Disease Mortality Follow-Up in Norwegian Men." *International Journal of Epidemiology*. 31, 799-805.

Elo, I. and S. Preston. (1996) "Educational Differentials in Mortality: United States, 1979-85." *Social Science and Medicine*. 42 (1), 47-57.

Evans, W., M. Farrelly, and E. Montgomery. (1999) "Do Workplace Smoking Bans Reduce Smoking?" *American Economic Review*. 89 (4), 728-747.

Fuchs, V. (1982) "Time Preference and Health: An Exploratory Study." In *Economic Aspects of Health*. V. Fuchs, ed. Chicago: University of Chicago Press. 93-120.

Fuchs, V. (2004) "Reflections on the Socio-economic Correlates of Health." *Journal of Health Economics*. 23, 653-661.

Glaeser, E. (1999) "Learning in Cities." *Journal of Urban Economics*. 46, 254-277.

Glaeser, E., B. Sacerdote, and J. Scheinkman. (1996) "Crime and Social Interactions." *Quarterly Journal of Economics*. 111 (2), 507-548.

Glaeser, E. and R. Saks. (2004) "Corruption in America." Harvard Institute of Economic Research Discussion Paper No. 2043.

Glied, S. and A. Lleras-Muney. (2003) "Health Inequality, Education and Medical Innovation." NBER Working Paper No. 9738.

Goldman, D. and D. Lakdawalla. (2001) "Understanding Health Disparities Across Education Groups." NBER Working Paper No. 8328.

Grossman, M. (1972) "On the Concept of Health Capital and the Demand for Health." *Journal of Political Economy*. 80 (2), 223-255.

Grossman, M. (2003) "Education and Nonmarket Outcomes." In *Handbook of the Economics of Education*. E. Hanushek and F. Welch, Eds. Amsterdam: North-Holland.

Grossman, M. (2004) "The demand for health, 30 years later: a very personal retrospective and prospective reflection." *Journal of Health Economics*. 23, 629-636.

Jayachandran, S. and A. Lleras-Muney. (2007) "Life expectancy and human capital investments: Evidence from maternal mortality declines." Princeton University Working Paper.

Haines, M. (2001) "The Urban Mortality Transition in the United States, 1800-1940." NBER Historical Paper No. 134.

Jones, A. (1994) "Health, Addiction, Social Interaction, and the Decision to Quit Smoking." *Journal of Health Economics*. 13 (1), 93-110.

Kawachi, I. and L. Berkman. (2000) "Social Cohesion, Social Capital, and Health." In *Social Epidemiology*. L. Berkman and I. Kawachi eds. New York: Oxford University Press. 174-190.

Kenkel, D. (1991) "Health Behavior, Health Knowledge, and Schooling." *Journal of Political Economy*. 99 (2), 287-305.

Kitagawa, E. and P. Hauser. (1968) "Education Differentials in Mortality by Cause of Death: United States, 1960." *Demography*. 5 (1), 318-353.

Lleras-Muney, A. (2005) "The Relationship Between Education and Adult Mortality in the United States." *Review of Economic Studies*. 72, 189-221.

Lochner, L. and E. Moretti (2004) "The Effect of Education on Criminal Activity: Evidence from Prison Inmates, Arrests, and Self-Reports." *American Economic Review*. 94, 155-189.

Manning, W., J. Newhouse, N. Duan, E. Keeler, A. Leibowitz, and M.S. Marquis. (1993) "Health Insurance and the Demand for Medical Care: Evidence from a Randomized Experiment." *American Economic Review*. 77 (3), 251-277.

Manski, C. (1993) "Identification of Endogenous Social Effects: The Reflection Problem." *Review of Economic Studies*. 60, 531-542.

Marmot, M., G. Rose, M. Shipley, and P. Hamilton. (1978) "Employment Grade and Coronary Heart Disease in British Civil Servants." *Journal of Epidemiology and Community Health*. 32, 244-249.

Marmot, M., G. Smith, S. Stansfeld, C. Patel, F. North, J. Head, I. White, E. Brunner, and A. Feeney. (1991) "Health Inequalities Among British Civil Servants: The Whitehall II Study." *The Lancet*. 337, 1387-1393.

Marmot, M. (2004) *The Status Syndrome*. New York: Henry Holt.

Meara, E. (2001) "Why is Health Related to Socioeconomic Status? The Case of Pregnancy and Low Birth Weight." NBER Working Paper No. 8231.

Miguel, E. and M. Kremer. (2004) "Worms: Identifying Impacts on Education and Health in the Presence of Treatment Externalities." *Econometrica*. 72, 159-217.

Milligan, K., E. Moretti, and P. Oreopoulos. (2004) "Does Education Improve Citizenship? Evidence from the U.S. and U.K." *Journal of Public Economics*. 88, 1667-1695.

Minino, A, M. Heron, S. Murphy, and K. Kochanek. (2007) "Deaths: Final Data for 2004." *National Vital Statistics Reports*. 55 (19).

Monden, C., F. Van Lenthe, N. de Graaf, and G. Kraaykamp. (2003) "Partner's and own education: Does who you live with matter for self-assessed health, smoking and excessive alcohol consumption?" *Social Science and Medicine*. 57 (10), 1901-1912.

Moretti E. (2004a) "Estimating the social return to higher education: evidence from longitudinal and repeated cross-sectional data." *Journal of Econometrics*. 121, 175-212.

Moretti E. (2004b) "Workers' education, spillovers, and productivity: evidence from plant-level production functions." *American Economic Review*. 94, 656-690.

Moretti, E. (2004c) "Human Capital Externalities in Cities." In *Handbook of Regional and Urban Economics*. Vol. 4, J. V. Henderson and J.-F. Thisse eds. New York: Elsevier, 2243-2291.

Morland, K., S. Wing, A. Diez Roux, and C. Poole. (2002) "Neighborhood Characteristics Associated with the Location of Food Stores and Food Service Places." *American Journal of Preventive Medicine*. 22 (1), 23-29.

Pappas, G., S. Queen, W. Hadden, and G. Fisher. (1993) "The increasing disparity in mortality between socio-economic groups in the United States, 1960 and 1986." *New England Journal of Medicine*. 329, 103-109.

Rauch, J. (1993) "Productivity Gains from Geographic Concentration of Human Capital: Evidence from the Cities." *Journal of Urban Economics*. 34, 380-400.

Ruggles, S., M. Sobek, T. Alexander, C. Fitch, R. Goeken, P. Hall, M. King, C. Ronnander. (2004) *Integrated Public Use Microdata Series: Version 3.0*. Minnesota Population Center [producer and distributor], Minneapolis.

Ruhm, C. (2000) "Are Recessions Good for Your Health?" *Quarterly Journal of Economics*. 115 (2), 617-650.

Sacerdote, B. (2001) "Peer Effects with Random Assignment: Results for Dartmouth Roommates." *Quarterly Journal of Economics*. 116 (2), 681-704.

Smith, J. (1999) "Healthy Bodies and Thick Wallets: The Dual Relation between Health and Economic Status." *Journal of Economic Perspectives*. 13 (2), 145-166.

Snyder, S. and W. Evans. (2006) "The Effect of Income on Mortality: Evidence from the Social Security Notch." *Review of Economics and Statistics*. 88 (3), 482-495.

Stafford, M. and M. McCarthy. (2006) "Neighbourhoods, Housing, and Health." In *Social Determinants of Health*. M. Marmot and R. Wilkinson eds. New York: Oxford University Press. 297-317.

Topa, G. (2001) "Social Interactions, Local Spillovers, and Unemployment." *Review of Economic Studies*. 68 (2), 261-295.

U.S. Bureau of the Census. (1999) *USA Counties 1998 on CD-ROM*. [machine readable data file]. Washington, D.C.: The Bureau.

U.S. Department of Health and Human Services, National Center for Health Statistics. (1993) *Multiple Cause of Death, 1990*. [Computer file]. Hyattsville, MD: U.S. Department of Health and Human Services, National Center for Health Statistics [producer]. Ann Arbor, MI: ICPSR [distributor].

U.S. Department of Health and Human Services, National Center for Health Statistics. (2002) *Multiple Cause of Death, 2000-2002*. [Computer file]. Hyattsville, MD: U.S. Department of Health and Human Services, National Center for Health Statistics [producer]. Ann Arbor, MI: ICPSR [distributor].

Wheeler, C. (2006) "Human Capital Growth in a Cross Section of U.S. Metropolitan Areas." *Federal Reserve Bank of St. Louis Review*. 88 (2), 113-132.

Zhu, B., G. Giovino, P. Mowery, and M. Eriksen. (1996) "The Relationship between Cigarette Smoking and Education Revisited: Implications for Categorizing Persons' Educational Status." *American Journal of Public Health*. 86 (11), 1582-1589.