Human Capital Spillovers and Health:

Does Living Around College Graduates Lengthen Life?*

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Abstract

Equally educated people are healthier if they live in more educated places. Every 10 percent point increase in an area's share of adults with a college degree is associated with a decline in all-cause mortality by 7%, controlling for individual education, demographics, and area characteristics. Area human capital is also associated with lower disease prevalence and improvements in self-reported health. The association between area education and health increased greatly between 1990 and 2010. Spatial sorting does not drive these externalities; there is little evidence that sicker people move disproportionately into less educated areas. Differences in health-related amenities, ranging from hospital quality to pollution, explain no more than 17% of the area human capital spillovers on health. Over half of the correlation between area human capital and health is a result of the correlation between area human capital and smoking and obesity. More educated areas have stricter regulations regarding smoking and more negative beliefs about smoking. These have translated over time into a population that smokes noticeably less and that is less obese, leading to increasing divergence in health outcomes by area education.

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I. Introduction

The health of a region is closely tied to its education level. In data from Ezzati et al. (2008), county life expectancy rises by 1.3 years as the share of adults with a college degree increases by 10 percentage points. Much of this county-level correlation between education and health reflects the well-known individual-level relationship between years of schooling and mortality (Elo and Preston, 1996; Cutler and Lleras-Muney, 2006; Grossman, 2006; Grossman, 2008; Meara, Richards, and Cutler, 2008; Cutler, Lange, Meara, Richards-Shubik, and Ruhm, 2011; Cutler and Lleras-Muney, 2012; Grossman, 2015), but that may not be all of it. This paper asks whether human capital spillovers in health, akin to human capital spillovers in earnings (Rauch, 1993; Moretti, 2004a; Moretti, 2004b; Canton, 2007; Rosenthal and Strange, 2008; Iranzo and Peri, 2009), can help explain the relationship between area education and mortality, and why that might be.

The link between area education and health was particularly apparent during COVID. The number of COVID-related deaths declined by 35% for each 10 percentage-point increase in college graduates in an area, ten times larger than the difference expected due to compositional effects alone.[1] Factors linked with area education, such as share of workers able to work remotely, household and workplace crowding, public health regulations, adoption of protective behaviors such as masking and vaccination, and differences in underlying population morbidity, likely explain these human capital externalities.

The COVID-19 pandemic was but one example of a larger trend. Large and growing geographic disparities in health across the U.S are central aspects of American life (Murray et al., 2005; Murray et al., 2006; Krieger et al., 2008; Ezzati et al., 2008; Kulkarni et al., 2011; Chetty et al., 2016; Dwyer-Lindgren, 2017; Finkelstein, Gentzkow, and Williams, 2021). The life expectancy gap between counties in the 1st vs. 99th percentile increased from 8.3 years in 1980 to 10.7 years in 2014 (Dwyer-Lindgren, 2017). Experimental and quasi-experimental methods have established that place of residence causally impacts both physical and mental health, although there is little consensus about why place is so powerful (Katz, Kling, and Liebman, 2001; Kling, Liebman, and Katz, 2007; Doyle, 2011; Ludwig et al., 2011; Ludwig et al., 2012; Ludwig et al., 2013; Deryugina and Molitor, 2020; Finkelstein, Gentzkow, and Williams, 2021). The increasing variation in education levels over the past four decades (Berry and Glaeser, 2005; Moretti, 2013; Diamond, 2016) combined with human capital spillovers in health may help us understand widening geographic health disparities.

To estimate the association between area education and health, we combine U.S. Census and American Community Survey data for 1990, 2000, and 2010 with complete mortality records

[1] Authors' calculations. Case and Deaton (2023) report a COVID mortality rate of 57 per 100,000 for those with a college degree and 164 per 100,000 for those without.

containing cause of death information and individual education from the Multiple Cause Mortality Files; the U.S. Standard Certificate of Death only included information on the decedent's education after 1989. After adjusting for individual-level educational attainment, a 10-percentage point increase in the percentage of college graduates in an area – a move from roughly the 25th percentile to the 75th percentile –is associated with a 7% lower all-cause mortality rate. This correlation has strengthened over time.[2] It is present across all demographic groups but is strongest for Hispanics and people under 65 and for people residing in rural and less educated areas.

Area human capital is also strongly correlated with non-fatal health outcomes and self-reported health. Controlling for individual-level education, a 10-percentage point increase in the percentage of college graduates in an area is associated with a 9% reduction in lung disease, a 6% reduction in heart disease, and a 10% reduction in the number of days in poor physical or mental health. When examining human capital spillovers separately by cause of death, we find that area human capital spillovers exist across almost all causes of death but are increasing over time only for medically amenable causes of death (which includes deaths due to respiratory conditions and heart disease), deaths due to cancer, chronic respiratory disease, external causes, and drug overdoses.

We present an analytical framework with heterogeneous human capital across individuals, spatial sorting, and investment in health that suggests three potential reasons for the correlation between area-level education and health: i) spatial sorting, where innately healthier individuals move to high human capital areas; ii) area-level amenities that influence health and that are correlated with higher education, for example a better and safer environment, more and better quality medical care, etc.; and (iii) individuals in better-educated areas choose fewer health-harming behaviors.

We reject the spatial sorting hypothesis by using data from the Health and Retirement Study for individuals 51 years of age and older and the National Longitudinal Survey of Young Women and Men for younger individuals. Sicker people, as measured by predicted mortality, move to areas with approximately equal levels of human capital as healthier people.

In contrast, we find that differences across areas in health-related behaviors such as smoking and obesity explain approximately 60% of the correlation between area human capital and mortality, after controlling for individual education. This result is presaged by the strong and robust correlation between area human capital and smoking, obesity, and physical activity that we document. Using data from the Behavioral Risk Factor Surveillance System and the Current Population Survey, after controlling for individual-level education, we show that a 10 percentage point increase in area human capital is associated with a 13% decrease in the probability of smoking, a 7% decrease in the probability of having no physical activity, and a 12% decrease in the probability of being very obese. Parallel to our findings for mortality, we also find that the

[2] Human capital earnings externalities also appear to be increasing over time (Glaeser et al., 2004).

human capital spillovers on health-related behavior are slightly stronger for areas that are least educated and rural.

These changes also show up in smoking initiation and cessation. Young adults in more educated areas are less likely to begin smoking than equally educated young adults in low human capital areas. Furthermore, conditional on being a smoker, individuals in better educated areas are much more likely to quit smoking in their 30s and 40s than similarly educated individuals in low human capital areas. These findings are true cross-sectionally and in panel data, controlling for area and time fixed effects. Accumulated over several decades, lower smoking initiation and higher quit rates in more educated areas have led to a population that is increasingly characterized by never smokers and former smokers compared to current smokers, resulting in growing gaps in mortality across areas and the greater correlation between area education and longevity over time.

Other health-related amenities such as pollution, crime, access to medical care, and quality of medical care do not explain much of the gradient in health with area education. We estimate that at most 17% of the human capital externality on health is due to these external factors, driven largely by greater use of preventative care. As we cannot observe or even imagine the full range of omitted variables that could potentially impact health, we cannot reject the view that omitted variables are more important than what we estimate, but we have no evidence of any such variable significantly explaining health disparities across the U.S.

We examine two potential channels linking area human capital spillovers and smoking: smoking regulations and social norms. Smoking regulations can either be seen as an area-level omitted variable or as a form of human capital spillover that works through employer practices or the political system. In the Current Population Survey, we find that individuals living in areas with a 10 percentage point higher share of college graduates are 2% more likely to be employed at workplaces with a smoking ban in all work and public areas, even after controlling for individual-level education.

We examine differences in social norms through questions on peoples' knowledge of smoking's harms and attitudes towards smoking regulation. Analyzing data from the National Health Interview Survey (1987, 1992, and 2000), we find that controlling for individual education, a 10 percentage point increase in the percent of college graduates is associated with an 11% increase in the probability of agreeing with the statement that smoking is harmful to fetal development and a 15% increase in the probability of agreeing that most lung cancer deaths stem from smoking. It is also associated with an 8% increase in the probability of supporting smoking bans in bars, restaurants, and work areas. Directly controlling for smoking regulations and beliefs about smoking in regressions of smoking on area human capital and individual education suggests that regulations and beliefs can explain up about 15% (17%) of the correlation between area human capital and smoking rates (quitting rates) and almost *a quarter* of the correlation between area human capital and smoking initiation. Beliefs about smoking are not distributed randomly across

areas, so this is not necessarily a causal relationship. However, it shows a nexus between smoking beliefs, smoking behavior, and area mortality that helps understand the growing correlation between life expectancy and education across areas.

Our conclusion is that the correlation between area-level education and beliefs about the harms of smoking is an important mediating factor contributing to area differences in health for equally educated individuals. These beliefs are associated with lower rates of smoking initiation and higher smoking quit rates, which over time drive increasing differences in health between areas with more and fewer educated individuals. Spillovers matter to a great extent because of what people internalize.

The paper is structured as follows. Section II discusses our data sources for mortality, non-fatal health outcomes, smoking, obesity, migration, and area characteristics. Section III establishes the baseline relationship between area human capital and mortality and examines variation in human capital spillovers by cause of death, demographic groups, and observable area characteristics. It also discusses the correlation between area human capital and disease prevalence and non-fatal health. Section IV presents a model of health-related behaviors and location choice that highlights the mechanisms behind human capital spillovers on health that we test empirically. In Section V, we empirically test the spatial sorting, health-related behaviors, and health-related amenities hypotheses. Section VI examines how human capital correlates with the cost of health-related behaviors across areas, as well as peer effects. Section VII concludes.

II. Mortality and Area Characteristics Data

In this section, we discuss our mortality and area-level data. All data sources used in our paper are summarized in Appendix A.

Mortality

We obtained microdata on all deaths of U.S. residents in 1990, 2000, and 2010 from the National Center for Health Statistics Multiple Cause Mortality Files (MCMF) restricted access files. MCMF data are compiled from death certificates and include the underlying cause of death as well as the age, sex, and educational attainment of the deceased (since 1988). Educational attainment on death certificates is typically reported by next-of-kin.[3] In the restricted access files, we also observe the deceased's county of residence.[4]

We aggregate total deaths into county-age-sex-race-education cells. We excluded the 3% of deaths that occurred among individuals younger than 25, as education is not reliably completed before

[3] Some concerns have been raised regarding the accuracy of such reporting relative to self-reports, particularly the overstatement of high school graduation rates (Shai and Rosenwaike, 1989; Sorlie and Johnson, 1996; Rosamond et al., 1997; Rostron et al., 2010).

[4] County of residence is suppressed for deceased individuals residing in counties with a population of less than 100,000.

that age. Cells were defined by 5-year age categories (25-29, 30-34, …, 85+), five levels of educational attainment based on completed years of school (<12, 12, 13-15, 16, 17+), gender (M, F), and race/ethnicity (White non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic).[5] We excluded deceased individuals with missing data on age (0.02%), county (0.16%), and education (10.5%) since we cannot match these deaths to a population denominator when calculating mortality rates. Most missing data on education occurred in 1990 for Louisiana, New York, Georgia, and Washington. These data exclusions mean that crude mortality rates in our sample are slightly lower than published estimates. This sample selection yielded 798,850 county-year-age-sex-race-education cells with non-zero deaths.

To ensure comparability of geographic units across years, we aggregated counties into consistent public use microdata areas (CONSPUMA), representing the most detailed geographic areas that can be consistently identified between 1980 and 2011.[6] This aggregation resulted in 309,970 area-year-age-sex-race-education with non-zero deaths.

To mitigate bias from the number of deaths without reported education, we excluded any area-year-age-sex-race cells where the percent of deaths with missing education was more than 25%, which eliminated 1.9% of adult deaths with non-missing age, race, area, and education. In robustness checks, we include only county-year-age-sex-race cells where the percentage of death certificates without reported education is 5% or less. Our regression analysis includes the percent of death certificates without education in each area-age-sex-race cell as a covariate.

Mortality rates were calculated by merging death counts for area-age-sex-race-education cells with corresponding population counts from the 1990 and 2000 U.S. Decennial Census (5% sample) and the pooled 2009-2011 American Community Survey (ACS) for 2010 (as in Wheeler, 2007).[7] Due to random sampling in the Census and ACS data, 21,746 area-year-age-sex-race-education cells with non-zero deaths could not be matched to population denominators.[8] We excluded these cells (containing 0.7% of deaths) from the analysis. There were 187 cells with death and population data, but where total deaths exceeded the estimated population, presumably due to sampling error. We censored mortality at 100 percent for these cells (reducing total deaths by 2,725 deaths).

Table 1 shows summary statistics for the mortality data. Our final dataset, pooled across 1990, 2000, and 2010, contains 369,707 area-year-age-sex-race-education cells and covers 5,934,489

[5] Due to differences in data encoding over time in the MCMF data, in 1990 and 2000, we considered individuals with four years of high school as having completed high school, regardless of whether they were awarded a diploma. In 2010, we considered those with 12 years of education and no high school diploma as not having completed high school. Associate degrees were included in the 13-15 (some college) education category.

[6] For counties included in multiple CONSPUMAs, we use the CONSPUMA containing most of the county's population. Only 36 out of 3141 counties in 1990, 42 in 2000, and 44 in 2010 (out of 3,219) map to multiple CONSPUMAs.

[7] We use the 3-year ACS because the 2010 Decennial Census did not include data on individual education.

[8] This sampling issue is generally concentrated in the college-educated population aged 70 and older.

deaths across all years, which represents 84% percent of deaths for people aged 25 and older.[9] The death rate was 1,196 deaths per 100,000, roughly 1 percent per year.

Cause of death is also identified on death certificates. We classified causes of death as medically amenable, smoking-related, obesity-related, or due to external causes based on the literature (see Appendix B for details). A cause of death can fall into multiple categories; for example, heart disease is both smoking-related and obesity-related. Approximately 56% of deaths were due to causes classified as smoking-related, 41% as obesity-related, 41% as medically amenable, and 6% due to external causes.

Data on Non-Fatal Health Outcomes

We obtained individual-level data on health conditions such as cancer, lung disease, diabetes, heart disease, and stroke from the Health and Retirement Study (HRS), which provides a biennial, longitudinal survey of people aged 51 and older over the 1992-2008 period. We also used microdata on self-reported general health and number of days over the last 30 days where poor physical or mental health interfered with daily activities from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS).[10] Each respondent in the HRS and BRFSS data was mapped to an area using the same methodology as with the MCMF data, as discussed in Section IIA.[11] As with the mortality data, we restrict the BRFSS sample to individuals aged 25 years and older. All data sources also contained individual education and demographics.

Data on Migration

We also use the HRS to assess migration across counties among individuals aged 51 and older using questions about cross-country migration between survey waves. We also look at migration at younger ages, using data from the National Longitudinal Survey of Youth (NLSY). The NLSY sample was aged 26-38 in 1990 and 46-58 in 2010. Thus, the ages just precede the HRS. Young men were asked in 1969-1971 and 1976 whether they had moved to a different SMSA or county since the last interview, and young women were asked annually or every two years between 1968-2001 whether they had moved to a different standard metropolitan statistical area (SMSA) or county since the last interview.

Data on Health-Related Behaviors

[9] The largest drop in sample size is due to the exclusion of decedents with missing education. The biggest drop from the 798,850 county-year-age-sex-race-education cells to the 369,707 area-year-age-sex-race-education cells is that counties are combined when aggregating the data to the area level.

[10] The 1990 BRFSS does not contain data on self-reported health or number of days where poor health interfered with daily activities. It also does not have consistent geographic identifiers.

[11] We utilize a restricted-use HRS file with county identifiers. County identifiers are included in the BRFSS but are suppressed for areas with fewer than 50 respondents.

We used self-reported data on smoking status, body mass index (BMI), and physical activity from the abovementioned 1999-2001 and 2009-2011 BRFSS data. Since the BRFSS does not contain data on these measures from the 1990s, we supplement the BRFSS data with data on individual education, demographics, and smoking behavior from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015.[12] These data contain geographic information for counties with a population of 100,000 or greater. As with all prior data sources, we include only individuals 25 or older and match available counties to larger areas (CONSPUMAs).

Area Characteristics

We merged in several area-level attributes, summarized in Appendix A, to the health data. Area human capital was defined as the percent of area residents aged 25 years or older in a given year who had at least a college degree, using Census data from 1990 and 2000 and ACS data from 2009-2011. We also obtained area-level percent Black and Hispanic, and industry shares (proportion of workers who work in agriculture, forestry, fisheries, and mining; construction; manufacturing; transportation, communications, and other public utilities; trade; finance, insurance, and real estate; services; public administration; armed forces) from these data sources.

Data on area population size and land area were obtained from the Area Resource Files provided by the Bureau of Health Workforce for 1990, 2000, and 2010, and we used these data to compute population density. We also obtained data on numbers of non-federal physicians and hospital beds at the county level for 1990, 2000, and 2010 from the Area Resource Files, which we analyzed on a "per 1,000 population" basis.

From the Dartmouth Health Atlas, we obtained county-level data on two population-based measures of access to primary health care: the average annual percent of Medicare enrollees having at least one annual ambulatory visit to a primary care clinician and the average percent of female Medicare enrollees aged 67-69 having at least one mammogram over a two-year period for years 2003-2015. We aggregated as discussed above.

County-level reported homicides were obtained from the Uniform Crime Reports. For each of 1990, 2000, and 2010, we averaged reported homicides in the three years centered around the decade (e.g., 1989-91 for 1990) to improve precision. We aggregate these data into areas and express them as rates per 100,000 individuals.

Satellite data on air pollution for 1999-2001 and 2009-2011 are from van Donkelaar (2019) and capture the concentration of suspended particulate matter of diameter 2.5μm or less (PM-2.5). For 1989-1991, we obtain data on PM-10 measurements from the Environmental Protection Agency for counties with particulate matter monitoring agencies. We follow the methodology from Meng

[12] The CPS data does not ask about height and weight and thus we cannot calculate BMI in this data set.

et al. (2019) to generate predicted PM-2.5 measurements for 1989-1991 using the PM-10 and PM-2.5 data.

Hospital quality data comes from the Hospital Compare Database provided by the Centers for Medicare and Medicaid Services for 2003-2008. The database contains information on process-of-care indicators for pneumonia, congestive heart failure, and acute myocardial infarction. These quality measures typically reflect the usage of inexpensive, easy-to-implement practices that represent the standard of care for patients presenting with these conditions.[13] For each hospital, quality scores were first averaged at the condition level, using condition-specific z-scores. We then average these z-scores for the three conditions, which provides a single hospital-specific metric for 3,879 hospitals, which we treat as roughly representing hospital quality for 2010. Finally, we calculate area-level hospital quality scores, weighting the hospital quality of all hospitals in the area by the number of discharges per hospital.

III. Area Human Capital and Mortality

We begin with an examination of the empirical relationship between area human capital and mortality. Figure 1 shows ventiles of the relationship between area human capital and mortality across area-age-sex-race-education cells for each of 1990, 2000, and 2010. In forming these ventiles, we control for age and sex but not for individual education.[14] The figure shows a negative relationship between education and mortality which is increasing over time. In 2010, a 10 percentage point increase in the area-level share of adults with a college degree – e.g., moving from the bottom quartile to the top quartile of the 2010 distribution of area human capital – was associated with a decline of 97 deaths per 100,000 ($p < 0.01$), a 8.2% reduction in all-cause mortality.

The major issue with interpreting these coefficients is that area education is clearly correlated with individual education, and individual education is clearly related to health.[15] Table 2 shows a variety of analyses separating individual and area-level education. Each column of the table reports the results of a regression model relating cell-level mortality rates to cell and area characteristics, using data for all area-year-age-sex-race-education cells. We limit controls to demographic and geographic characteristics that are unlikely to be part of the causal pathway between area human capital and health: in addition to the controls shown in Table 2, we include controls for 5-year age-sex-race/ethnicity interactions, as well as year. We also control for the percent of death certificates

[13] For example, one measure is the percent of patients presenting with an acute myocardial infarction who are given aspirin upon arrival.

[14] Since we do not control for individual education, data in this figure includes deaths with missing education. Excluding deaths with missing education yields similar results (see Appendix Figure C1).

[15] The literature on the relationship between individual education and health is vast. For a comprehensive review of the theoretical background, as well as descriptive and quasi-experimental evidence on the relationship between education and health, see, for instance, Grossman (2006), Cutler and Lleras-Muney (2006), Grossman (2008), Cutler and Lleras-Muney (2012), Grossman (2015), and Galama et al. (2017).

in the cell with missing education data, population and population density (both log-transformed), and employment shares by industry at the area level.

Column 1 of the table examines the effect of individual education alone. Controlling for other cell-level and area-level covariates, the correlation between individual education and mortality is enormous. Individuals without a high school degree experience 730 additional deaths per 100,000 relative to individuals with graduate education. Mortality risk declines with each additional level of educational attainment. The second column shows the relationship between mortality and area human capital without individual education controls. These results are closely related to Figure 1 and show that a 10 percentage point increase in the area-level percent of the adult population with a college degree is associated with 103 fewer deaths per 100,000.

The third column presents the primary motivating fact for the paper. Even controlling for individual education, a 10 percentage point increase in the share of college graduates in an area is associated with 64 fewer deaths per 100,000 ($p < 0.01$), a 5.4% decrease relative to average mortality. The difference in the coefficients on the share of college graduates between the second and third columns of Table 2 implies that controlling for individual education explains just 38% of the relationship between area human capital and mortality shown in Figure 1.

Column 4 of Table 2 allows for the relationship between area human capital and mortality to vary by year. As with Figure 1, the relationship between area human capital and mortality increases over time. Previous studies have found widening mortality disparities across individuals with different levels of education over time (Meara, Richards, and Cutler, 2008; Cutler, Lange, Meara, Richards-Shubik, & Ruhm, 2011; Olshansky et al., 2012; Masters et al., 2012; Hayward et al., 2015; Sasson 2016; Bor et al., 2017). Our paper demonstrates that there exists a similarly increasing impact of area human capital on mortality over time.

Columns 5 and 6 match the specifications in columns 3 and 4 but include state-by-year fixed effects, which account for time-varying state-level characteristics that may be correlated with both area human capital and health (e.g., changing state-level health or education policies such as Medicaid coverage, tobacco taxes, smoking regulations, etc.). The impact of area human capital on mortality falls in these specifications but remains statistically significant and increasing over time.[16] Thus, differences in state-level policies cannot be the sole factor driving the correlation between area human capital and health nor the increase in this effect over time; rather, local relationships exist.[17]

[16] This is consistent with Karas Montez et al. (2022) findings on the relationship between state policies and the mortality of working-age adults.

[17] We obtain similar results when including a full set of individual-level age-sex-race-education interactions – a 10 percentage point increase in the percent college graduates in an area is associated with a decrease of 81.8 deaths per 100,000. Even if we control for the changing relationship between individual education and mortality over time by including fixed effects for year interacted with individual education, we find that a 10 percentage point increase in the percent college graduates in an area is associated with a decrease of 81.1 deaths per 100,000.

Finally, columns 7 and 8 include area fixed effects, which control for time-invariant area-level characteristics that may be correlated with area human capital and health. Within areas, there is a similar correlation between area human capital and health as in our baseline specifications in columns 5 and 6.[18]

Figure 2 presents estimates from our baseline regression from column 5 in Table 2 but with mortality rates separated by cause of death (Appendix Table C1 has full regression results). Area human capital is negatively correlated with mortality rates across all cause-of-death groupings we analyzed – medically amenable, smoking-related, obesity-related, and external; as well as deaths from heart disease, cancer, chronic lower respiratory disease, stroke, and drug poisoning. While the main correlation between area human capital and mortality persists across all causes of death, Appendix Table C2 shows that this correlation strengthens over time for deaths due to medically amenable causes, chronic respiratory disease, cancer, and external causes.

Heterogeneity in the Relationship Between Education and Mortality

Figure 3 shows estimates of area human capital using the same regression as in column 5 of Table 2, estimated separately by age, gender, individual education, and race.[19] In absolute terms, the relationship between area human capital and mortality is larger for older than younger individuals. However, in relative terms, the relationship between area human capital and health is stronger for younger individuals. A 10-percentage point increase in the area-level percent college graduates is associated with a 14% decrease in the mortality rate among individuals younger than 65 and a 5% decrease in the mortality rate among those over 65. The relationship between area human capital and mortality is slightly stronger for men and more educated individuals relative to the respective means. Coefficient estimates are similar for white, Black, and Hispanic individuals in absolute terms, but the relationship is strongest for Hispanic individuals relative to the average mortality rate. Appendix Table C4 shows that the relationship between area human capital and mortality increased over time for younger and older individuals, women and men, and the more and less educated. However, the relationship between area human capital and mortality strengthens over time for only white individuals.

Figure 4 examines how the impact of area education varies with area characteristics. Figure 4 shows the coefficient on the interaction of area human capital with being above or below the median on four area characteristics: area human capital, percent of the area population that is Black, percent of the population that is Hispanic, and population density. We use the specification

[18] Our within-area results are consistent with our findings that (a) if we control for the lag of area human capital, contemporaneous area human capital is strongly negatively correlated with all-cause mortality, and (b) changes in area human capital are strongly negatively correlated with all-cause mortality after controlling for initial area human capital levels. Altogether, these results suggest that it is the change in area human capital levels between years driving the correlation between area human capital and mortality rather than the initial levels of area human capital.

[19] Appendix Table C3 has the detailed regression results.

where the coefficients differ by year (column 6 of Table 2) to examine both levels and changes in the relationship.

There is a negative correlation between area human capital and mortality across all area characteristics. In general, the negative connection between area education and all-cause mortality is strong in areas that are less educated and rural (as of 1990). However, the coefficients are not statistically different across the groups (conditional on year). The strengthening correlation between area human capital and all-cause mortality over time was apparent across all strata of area characteristics. We observe similar patterns when considering mortality by cause of death instead of all-cause mortality.

Area Human Capital and Non-Fatal Health Outcomes

We also examine the relationship between area human capital and health for non-fatal health outcomes. To some extent, such relationships are presaged by the findings for mortality, but these are also of independent interest because they allow us to compare the magnitude of the impact on disease prevalence relative to disease outcomes. We focus on new diagnoses of cancer, lung disease, diabetes, heart disease, and stroke – the major conditions asked about in the HRS. With the BRFSS, we examine self-reported health. Table 3 shows results with our baseline set of covariates for these alternative health measures. Area human capital is negatively and statistically associated with new lung and heart disease onset cases but is not associated with the onset of other conditions. Area human capital is also strongly associated with self-reported health, as measured by the percent of BRFSS respondents self-reporting good, very good, or excellent health and the number of days in the last 30 days where the BRFSS respondent reported physical and mental health interfered with usual activities.

IV. Model of human capital spillovers and health

We posit three potential explanations for the observed relationship between area-level education and health. The first is selective migration – healthier individuals move to better-educated areas. The second theory is that higher human capital areas have more and/or better health-related amenities, such as less pollution, violent crime, or better medical care. The third theory is that there are differences in health-promoting behaviors across high and low human capital areas due to spillovers in information, time preferences, or other factors. Legislation that increases the costs of healthy behavior, such as rules about smoking in public, can be seen as either a health-related amenity or a local behavioral norm, albeit one with the force of law.

We do not focus specifically on income because in a spatial equilibrium, higher wages in an area (holding human capital constant) are offset either by worse amenities or higher costs. Thus, the logic of a spatial equilibrium suggests viewing human capital as a first cause of wages and not viewing the wages in an area as an independent causal variable.

We assume that individuals potentially live for two periods ($t = 1, 2$) and make health-related consumption decisions in the first period that impact their probability of survival in the second period. We do not allow savings but rather assume that at time $t = 2$, the individual achieves nonnegative utility equal to $V(E)$ if the person is living and zero otherwise, where E denotes the individual's human capital, perhaps translated into wages. There is no savings. At $t = 1$, expected utility for a representative individual living in area k equals:

$$U(T, N, B, B_k) + b(B)hQ_k\beta V(E), \qquad (1)$$

where T refers to traded goods bought at a numeraire price of 1, N refers to non-traded goods purchased at an endogenous price of p_k^N, B refers to health-related behaviors (e.g., smoking, overeating, and taking medication) which are bought at an exogenous price of p_k^B, and B_k refers to the average level of health-related behaviors in area k.

We assume that there are two levels of human capital (E_H and E_L), and we denote $\mathrm{V}(E_x) = V_x$ for $x=L, H$ where $V_H > V_L$. We will assume that the number of low human capital individuals in a location is fixed. For Proposition 2, we assume that a spatial equilibrium in which high human capital individuals choose where to live at the beginning of the first period. High human capital types all have the option of moving to a reservation locale that provides them with a lifetime expected utility of U_H. As $\mathrm{V}(E_x)$ is assumed to be independent of second period location, mobility decisions after the first period are irrelevant.

Second-period utility is discounted by a discount factor β and multiplied by the survival probability, $b(B) * h * Q_k$, which has three components: $b(B)$, a decreasing function of the individual's health-related behavior B; h which denotes the individual's innate well-being, which determines the probability of not dying from causes unrelated to the behavior; and Q_k, which represents area-specific health-related factors, based on first period location, such as healthcare quality and other health-related area attributes, including the social and physical environment (e.g., pollution, crime, health-related regulations). Assume that $b(B) = max[1 - d_0 B, 0]$, with d_0>0; thus, $b_B(B) \leq 0$ and B represents harmful health-related behaviors.

To derive explicit solutions, we assume that $U(T, N, B, B_k) = T + g(N) + b_0 B - \frac{b_1}{2} B^2 - \frac{b_2}{2}(B - B_k)^2$, where all parameters are positive. Utility is linear in traded goods consumption and concave in non-traded goods consumption ($g'(N) > 0$; $g''(N) < 0$). Without reference effects, utility is concave in the consumption of health-harming goods. In addition to that concavity, people get utility from having consumption similar to that of their peers. In this model, there are direct peer effects in health-related behaviors (i.e., $B - B_k$ enters utility directly); empirically, we will test whether more educated individuals shift the behavioral norm in the community via information spillovers or policies and legislation targeting health-related behaviors that make unhealthy behaviors costlier. Furthermore, previous studies suggest that smoking, obesity, healthy eating, depressive symptoms, sleep, substance abuse, and other related behaviors are complementary

across individuals with close social or geographical ties (Christakis and Fowler, 2007; Christakis and Fowler, 2008; Fowler and Christakis, 2008a and 2008b; Cacioppo et al., 2009; Cutler and Glaeser, 2010; Mednick et al., 2010; Rosenquist et al., 2011).

As there is no saving, consumption of the traded good (T) equals $Y_k^x - p_k^N N - p_k^B B$, where Y_k^x refers to the earnings in location k of an individual with education x=L, H. At an interior equilibrium, all individuals consume all three goods so that consumption of N satisfies $g'(N) = p_k^N$ for everyone. Low-human capital individuals are immobile and have an area-specific health level of $h_L = h_L^k$ reflecting environmental factors in their area. They work providing non-traded goods and are each able to produce n_k units of non-traded services. Highly educated individuals produce traded goods, which are produced using a constant returns-to-scale technology where productivity and wage per worker equals W_H^k. All high human capital individuals have health of $h_H > h_L^k$.

As mentioned above, in Proposition 1 below, we assume that there is an exogenous share of high-education individuals living in area k, denoted σ_k. In proposition 2, we allow the highly educated to move and impose a spatial equilibrium so that their lifetime expected utility must equal a reservation value of U_H.

Proposition 1: Unhealthy behavior is higher for the less educated group, and the levels of unhealthy behavior for both groups and the area overall are decreasing with the share of the highly educated population.[20]

Human capital spillovers stem from peer effects in unhealthy behavior. Better-educated people engage in less unhealthy behavior because they value longevity and thus derive more utility from an increased probability of survival. A greater share of the educated population then shifts the community's behavioral norm, which makes unhealthy behavior costlier for everyone. The desire to conform with the area-wide average means that factors that increase the share of the population that is educated will shape the health of the area, as we show in Proposition 2:

Proposition 2: Increases in either W_k^H or h_L^k or Q_k will cause (1) the share of the area that is educated to increase, (2) the level of unhealthy behavior for both high and low education groups in the area to decline and (3) the probability of survival for both groups to increase.

Proposition 2 highlights three forces that can induce an increase in an area's education level. First, a place with a more productive, skill-intensive export sector will attract more educated individuals. Second, a place where less educated individuals are innately healthier will attract more skilled individuals. This is because health is associated with less engagement in health-harming behavior, and individuals prefer moving to areas where others consume as they do. Thus, higher-skilled individuals will move to healthier areas (only highly educated people are mobile in this model).

[20] Proposition proofs are contained in Appendix D.

Third, a place with better health-related amenities will also attract the educated.[21] These three channels highlight the many reasons why health and schooling may move together across space. Differentiating between these reasons is the task of the rest of the paper.

Our model assumes that health-related amenities are exogenously given for each area, but we empirically examine how differential investment in health-related amenities across areas with different human capital relates to externalities to health. For instance, higher education levels in an area may induce more skilled doctors to move there since they might prefer living around other skilled individuals or because the demand for healthcare services is higher. Individuals may also be more willing to vote for public investments in medical care, public health, or external stressors in areas with higher human capital. Additionally, better-educated patients may provide doctors, hospitals, and insurers with more discipline in delivering high-quality care. The spatial aggregation of the highly educated (and better paid) may also generate greater demand for medical care and lead to quality improvements associated with scale and specialization. Higher area human capital may be associated with healthier physical and social environments, which we also consider health-related amenities.

V. Testing Explanations: Sorting, Behaviors, and Amenities

Spatial sorting

We first turn to the hypothesis that the relationship between area human capital and health is explained by spatial sorting: healthy individuals move to areas with higher human capital, or less healthy individuals move to areas with lower human capital. Under this hypothesis, area human capital need not have any direct effect on health; rather, healthier migrants are attracted to higher human capital areas because they can afford the higher housing prices, have preferences for amenities catering to healthier individuals (e.g., healthier food, gyms, parks, etc.), or because they prefer living among individuals with similar tastes, among other reasons. It is also possible that less healthy people may sort into higher human capital areas because they place greater value on amenities (e.g. high-quality hospital care) in those areas, which we explicitly examine below.

We use data from the HRS to test the sorting hypothesis. We create a measure of health status as the predicted probability of death in the next two years, given information on demographics and health conditions.[22] We then estimate a probit model for migration to another county in the next two years, using our baseline health measure as the main explanatory variable and individual demographic and area-level controls as in the previous analyses as controls.

[21] This result can easily persist in a model where both education groups are mobile, as long as the highly educated individuals value health more than less educated individuals.

[22] Specifically, we estimate a probit model for mortality that includes indicators for whether the respondent was working, baseline risk factors such as high blood pressure, ever and current smoker, BMI, and medical history (ever had heart disease, lung disease, cancer, stroke, arthritis, psychological conditions, hospitalizations).

Table 4 shows our results. The first column shows that people who are less healthy are more likely to move across counties than people who are healthier. This finding is consistent with Finkelstein, Gentzkow, and Williams (2016 and 2021), who report similar findings using Medicare data. The second column assesses whether healthier migrants sort into higher educated counties. Restricting the sample to movers, we estimate a regression that relates baseline health status to the difference in area education between the destination county and the origin county. There is no statistically significant association between baseline health status and the human capital differential between origin and destination counties. Among people over 50, healthier people are not more likely to move to better educated counties.

We also look at younger ages just preceding the HRS using data from the NLSY. We use a similar approach to the HRS. We start by predicting the probability of dying between the current and next interview using a probit model relating death to demographic and health characteristics in the current interview wave. Because the surveys asked different health-related questions for men vs. women, we use different predictors for the two groups and report results separately. For men, the controls include 5-year age categories by race/ethnicity interactions, individual education, year, whether the individual had any health limitations interfering with work, school, or other activities, and the type and duration of health limitations. Additional controls for women include BMI, whether they were a current smoker, whether they currently have angina, hypertension, congestive heart failure, whether they have ever had an acute myocardial infarction or cancer, and whether they have any health limitations affecting school, work, or other activities. We then relate baseline health to the probability of moving to a new SMSA or county before the next interview, using a probit model.

The results are shown in columns 3-5 of Table 4. Column 3 shows that young women in worse baseline health are more likely to move to a new SMSA or county. This is not true among young men, as shown in column 5, but this estimate is noisy. We do not observe geographic identifiers in the NLSY, but column 4 further shows that among young women who move to a new SMSA or county, those of worse baseline health are more likely to remain in an SMSA or move to an SMSA from a location that is not an SMSA. If we consider SMSAs to be urban, high human capital areas relative to non-SMSAs, this is consistent with the idea that those with worse baseline health are more likely to move to high human capital areas. We thus take the NLSY results as suggestive evidence that the sorting hypothesis might not hold for younger adults either.[23]

[23] As an alternative approach to address spatial sorting, we also examined an instrumental variables approach used in Moretti (2004b), Wheeler (2008), and Diamond (2016). These papers use two instruments for the level of human capital in an area: (1) the presence of a land-grant college in the area established by the federal Morrill Act of 1862 and (2) a shift-share instrument using the demographic structure of an area from 1980 in combination with secular national changes in educational attainment biased towards more college over time. Two issues arose when using these instruments. First, both instruments yielded F-statistics less than 10 in the first stage after including individual education, indicating weak instruments. Second, individual educational attainment can also be affected by these instruments, and thus controlling for individual education in addition to instrumenting for area human capital may violate instrumental variables assumptions.

Health Behaviors

We now turn to our second hypothesis, which suggests that area human capital affects health-related behaviors. We focus on the two behavioral health risk factors that contributed the most to mortality in the U.S. in 2000 – smoking and obesity (Mokdad et al., 2004; Cutler and Lleras-Muney, 2010) – and which already relate to area human capital as shown in Figure 2.

We use data on smoking status and obesity from the BRFSS and smoking status from the CPS, each matched to area characteristics measured in the decennial census or ACS wave immediately preceding the given year.[24] We use similar regression models as for our baseline mortality regressions in column 5 of Table 2 – including state-year but not area fixed effects – but instead of mortality as the dependent variable, we use whether the individual was a current, former, or never smoker, three categories of obesity based on BMI (very obese [BMI>35], obese [35>=BMI>30], and overweight [30>=BMI>25] vs. normal or underweight), and whether the individual reported mainly being physically inactive (vs. being physically active). We also use probit instead of OLS for estimation since all outcomes are binary variables.

Figure 5 shows the coefficients and standard errors for area human capital.[25] Area human capital is strongly negatively correlated with the probability of being a current smoker and being obese. The coefficient on area human capital for current smoking is similar in the two data sets. Focusing on the CPS data, which includes data from the 1990s, the coefficient implies that individuals living in areas with 10 percentage points more college graduates are 2.23 percentage points less likely to be current smokers, equivalent to a 13.5% decrease in the probability of smoking relative to the average smoking rate. People who live in high human capital areas are also more likely to have never smoked and to quit smoking conditional on ever starting smoking. Area human capital is also statistically significantly associated with a lower probability of being overweight or obese – a 10 percentage point increase in the percentage of college graduates in an area is associated with a 12.4% lower likelihood of being very obese and a 4.1% lower probability of being obese. Consistent with these findings, people are also less likely to engage in no physical activity in areas with higher human capital. These findings closely align with causal neighborhood effects on obesity from the Moving to Opportunity experiment (Ludwig et al., 2013). Appendix Tables C7 and C8 show that the relationship between area human capital and health-related behaviors has only slightly strengthened over time. We also find that area human capital spillovers on smoking and obesity are slightly stronger for areas that start off as least educated and rural in 1990.

We find significant heterogeneity across demographic groups in health behaviors. The correlation between area human capital and healthy behaviors (i.e., not smoking, normal BMI, physical activity) is stronger for women than for men, for younger than older individuals (especially never

[24] E.g., 1995 is merged to area data from the 1990 census, 2003 to area characteristics from the 2000 census, and 2014 to area data from the 2009-2011 ACS).
[25] See Appendix Table C5 and Appendix Table C6 for detailed regression results.

taking up smoking and obesity rates), for higher educated than lower educated individuals (although smoking quit rates are higher among the low educated), and non-white individuals relative to white for smoking but the reverse for obesity.

We highlight the correlation between area human capital and smoking by age in Figure 6. This figure plots the coefficients on area human capital interacted with age from regression models using our standard set of controls where the outcome is smoking non-initiation (never smoking) in Figure 6a and the smoking quit rate (formerly smoking conditional on ever smoking) in Figure 6b. Spillovers of area human capital on preventing smoking initiation begin in young adulthood, but the biggest spillovers on quitting smoking occur in the late 30s and early 40s. At this pivotal age, 10-15 years of prior smoking is sufficient to harm health, but quitting ameliorates the risk of these harms manifesting in 10-20 years when these individuals are in their 50s and 60s. In other words, in low human capital areas, young individuals are taking up smoking at higher rates relative to similarly educated peers in high human capital areas. Further, conditional on having taken up smoking, in high human capital areas, older individuals are quitting smoking at higher rates in their 30s and 40 relative to similarly educated individuals in low human capital areas. Higher quit rates and lower initiation of smoking across the older and younger cohorts of more educated areas will yield strengthening relationships between health and area human capital over the next 10-20 years as health benefits of healthy behaviors accrue to individuals as they age.[26]

To summarize how much these variables can explain the area's effect on mortality, we re-estimate our central model in column 5 of Table 2, further including measures of smoking and obesity in the area as controls. For smoking, we use the BRFSS data as a baseline and supplement it with CPS data if BRFSS smoking rates for a given area are missing (before 1999). Area-level data on obesity comes from the 1999-2001 and 2009-2011 from the BRFSS and is only available for those years.[27] Even with these noisy measures of smoking and obesity, Table 5 shows that controlling for differential smoking rates explains about 38%-46% of the effect of area human capital on all-cause mortality, depending on the year (as demonstrated by the difference in the coefficients between column 1 and column 2 for 1990-2010 and column 3 and 4 for 2000-2010). Controlling for both smoking and obesity explains 59% of the correlation between area human capital and mortality in 2000 and 2010, mostly driven by differences in smoking. Smoking and obesity are particularly good for explaining the correlation between area human capital and deaths due to cancer, chronic lower respiratory disease, stroke, and drug-related deaths, as shown in Appendix Table C9.

[26] Consistent with the hypothesis of accumulating effects, in a regression including lagged and contemporaneous area human capital, initial levels of human capital are strongly negatively correlated with mortality for young individuals (<65 years) but not their older counterparts.

[27] Since not all areas are represented in the CPS and BRFSS, we estimate the models including smoking, obesity, and physical activity among cells where we have available data on these behaviors. Thus, the number of observations and average mortality rates reported in Table 5 are lower than the ones reported in Table 2.

Other Health-Related Amenities

We next turn to whether some of the remaining differences in mortality across areas can be explained by correlations between area education and health amenities that may not operate primarily through smoking and obesity. We focus on two external stressors – air pollution and crime – as well as measures of healthcare quality and access, while acknowledging that other environmental factors beyond these may affect health.

Adverse health effects due to exposure to air pollution include increased lung disease incidence or aggravation of existing lung disease, cancer, and premature death (Environmental Protection Agency, 2023). High levels of air pollution may also discourage outdoor exercise and thus indirectly impact mortality through obesity. Higher area human capital could also be associated with less pollution because air quality may be priced into property values, leading to selection of the better educated (and wealthy) into such areas. While homicides are a crude measure that may not capture all aspects of crime, they are more reliably reported than other crimes (Bureau of Justice Statistics, 1994). Crime could also decrease health through indirect channels; for example, unsafe streets could increase stress, lead residents to stay inside and get less exercise, or make it difficult to obtain necessary healthcare or management of chronic conditions.

Additionally, pollution and crime might be lower in more educated areas for similar reasons that demand for high-quality medical care might be higher – people in higher human capital areas may vote more for public goods addressing environmental stressors and may possess the political clout to regulate crime and pollution. We also control for differences in healthcare quality captured by hospital quality since most deaths occur in hospitals/nursing homes and mortality may be particularly sensitive to this dimension of healthcare quality.

Figure 7 builds on the regressions with smoking and obesity controls from column 5 of Table 5 and examines whether controlling for differences in health-related amenities across areas can explain the correlation between area human capital and mortality above and beyond what is explained by health-related behaviors. The first set of models in Figure 7 start from the model in column 6 of Table 5, which adjusts for smoking and obesity patterns, and sequentially add in pollution, crime data, number of physicians, and number of hospital beds. The second set of models start from the model including all previously mentioned health amenities as controls and adds demand for preventative care as a control. The last set of two models uses data for 2010 only and controls for healthcare demand and quality.[28]

In total, external factors such as pollution and homicide rates explain a small share of the relationship between area human capital and mortality after controlling for smoking and obesity. Both pollution and homicides are positively correlated with mortality, but neither explains much

[28] Appendix Table C10 shows the corresponding regression results in table form. Appendix Figure C2 shows the same results but building on column 2 in Table 5, which does not include obesity as a control and thus allows us to include data from 1990.

of the relationship between area human capital and mortality. Similarly, measures of healthcare demand and quality, such as the number of physicians, hospital beds, and healthcare quality, which are also correlated with mortality, cannot solely explain the effect of area human capital on mortality beyond what is already explained by differences in smoking. Preventative care measures, particularly percent of women who timely go for mammogram screenings, explain 32% of the relationship beyond what is already explained by smoking and obesity (i.e., around 12% of the baseline relationship between area human capital and mortality reported in Table 2). Health-related amenities in total explain 17% of the relationship between area human capital and mortality conditional on individual education.

VI. Understanding Health-Related Behaviors

Regulation

Given the prominent role of health-related behaviors in explaining the correlation between area human capital and mortality, we consider why they are so related to area human capital. One possible theory is through prices. More educated areas may be more likely to support legislation and regulations aimed at improving health. For example, this may include tobacco control policies such as tobacco taxes, clean indoor air laws, and workplace smoking bans. Tobacco taxes are typically set at the federal or state level. States may also mandate clean indoor area laws in some places (e.g., in workplaces, restaurants, and bars). Thus, tobacco taxes and state clean indoor air laws and regulations will typically vary by state and year, and since we include state-by-year fixed effects in our specifications, we focus on private workplace smoking bans which can be implemented as company policy independent from law and thus may vary within states and years. The CPS data described above ask questions on workplace smoking policies for indoor workers. We focus on whether the workplace has an official smoking policy in place (which is likely a regulation) and whether the workplace bans smoking in all public and work areas.[29]

Table 6 shows the impact of area human capital on these policies. Controlling for the individual's own education, individuals living in more educated areas are more likely to work at places with a complete ban on smoking in all public and work areas. A worker with a 10 percentage point higher share of college graduates is 2.1% more likely to be employed at places with a complete smoking ban. This finding is completely attributed to local policies as we include state-year fixed effects in our specifications.

Peer Effects and Social Norms

[29] Several papers discuss the effectiveness of these bans in reducing smoking. For example, Evans, Farrelly, and Montgomery (1999) show that compared to a firm with few restrictions on smoking, adopting a smoke-free policy at a workplace reduces the probability of smoking by 5.7 percentage points and decreases the daily number of cigarettes smoked by 14% on average.

A second theory is that area human capital drives peer effects, leading to the development of different social norms in high and low human capital areas. For instance, the proximity of more educated individuals undertaking healthy behaviors may encourage individuals across the education distribution to undertake healthy behaviors themselves. Differences in information and beliefs about the harmful effects of smoking and obesity, which may correlate with area human capital, may also be driving these differences in smoking behavior across areas. Finally, people in areas with different levels of human capital may hold different attitudes towards smoking and obesity, e.g. how socially acceptable it is and what role regulation should play to limit adverse health impacts. These changes in social norms may result from public health messaging campaigns or may arise more organically as peer effects.

While we cannot directly assess direct peer effects because these inherently reflect preferences not captured in our data, we can examine informational spillovers and attitudes towards smoking. The 1987, 1992, and 2000 National Health Interview Surveys (NHIS) asked individuals about their agreement with a series of statements about the effects of smoking on health: smoking by pregnant women is harmful for the baby, someone else's smoke is harmful, and most lung cancer deaths are caused by smoking, among others. We consider how these are related to area education.[30]

Table 7 shows regression results. NHIS respondents living in counties with a 10 percentage point higher percentage of college graduates are 11% more likely to agree with the statement that smoking is harmful for pregnant women's babies and 15% more likely to agree that most lung cancer deaths stem from smoking, controlling for individual education. These results are statistically significant at the 10% level and 5% level, respectively. As the next columns show, individuals living in more educated areas are also more likely to support smoking bans in bars, restaurants, and work areas, based on data from the CPS, reflecting attitudes towards smoking and regulation of it.

We also examine whether smoking regulations and beliefs and attitudes about smoking mediate the correlation between area human capital and smoking behavior. These results are not causal since regulations and beliefs are not randomly assigned across areas, but are indicative of the relationship between beliefs about the harms of smoking or local smoking regulations and smoking initiation/cessation across areas. Table 8 shows the relationship between area human capital and the probability of smoking controlling for workplace smoking bans and beliefs about whether smoking should be banned in bars, restaurants, and workplaces. Both the presence of workplace smoking bans and the share of individuals who believe that smoking should be banned everywhere are strongly negatively correlated with being a current smoker and positively associated with the probability of being a never-smoker and former smoker. Differences in the coefficient on the

[30] Each year in the NHIS data was merged to area characteristics measured in the decennial census immediately prior to the given year (i.e., 1987 is merged to area characteristics from 1980, 1992 to area data from 1990, 2000 to area data from 2000). We use counties instead of CONSPUMAs in the NHIS.

percent college graduates across the columns of Table 8 suggest that believing that smoking should be banned everywhere can explain 12%, 15%, and 22% of the correlation between area human capital and current smoking, former smoking, and never smoking, respectively. Workplace smoking bans explain an additional 2-5% of the correlation. Shifting social norms around smoking are thus associated with a substantial share of the divergence in smoking and subsequent mortality between high- and low-human capital areas.

VII. Conclusion

Our paper documents a strong and robust relationship between area human capital and mortality, even after controlling for individual education. This relationship emerged in 1990 but has strengthened over time throughout the 2000s and 2010s. The correlation between area human capital and mortality is strongest, on a relative scale, for individuals younger than 65 and Hispanic individuals and further extends to non-fatal health outcomes such as lung disease, heart disease, and the number of days in poor physical or mental health. These spillovers are observed across many causes of death.

Motivated by a model of spatial sorting and investment in health with heterogeneity in human capital across individuals, we consider several pathways through which area human capital may impact health. More than half of the correlation between area human capital and mortality can be explained by differences in smoking rates and obesity rates across areas. We document strong correlations between area human capital and smoking and obesity conditional on individual education. We further examine two channels for these spillovers and find empirical evidence for both: regulatory policies that increase the price of unhealthy behaviors, such as workplace smoking bans, and peer effects about the harms of smoking. We further find that health-related amenities such as pollution, homicides, healthcare quality, and quantity can explain at most 17% of the correlation between area human capital and mortality. Lastly, we find little evidence for spatial sorting driven by health – healthier individuals are less likely to migrate than sicker individuals, and healthier individuals are not more likely to move to more educated areas.

Our paper points towards mechanisms that can help explain why locations have such a powerful impact on health, shedding light on local policies that do not target health directly but may affect it indirectly. Even without direct effects of local and place-based labor or educational policies on health, any welfare and general equilibrium analysis of such policies may need to incorporate spillovers on health. Health-related behaviors are particularly sensitive to human capital spillovers among younger individuals, implicating the role of changing social norms around smoking and obesity across generations in the widening geographic gaps in health between high and low human capital areas.

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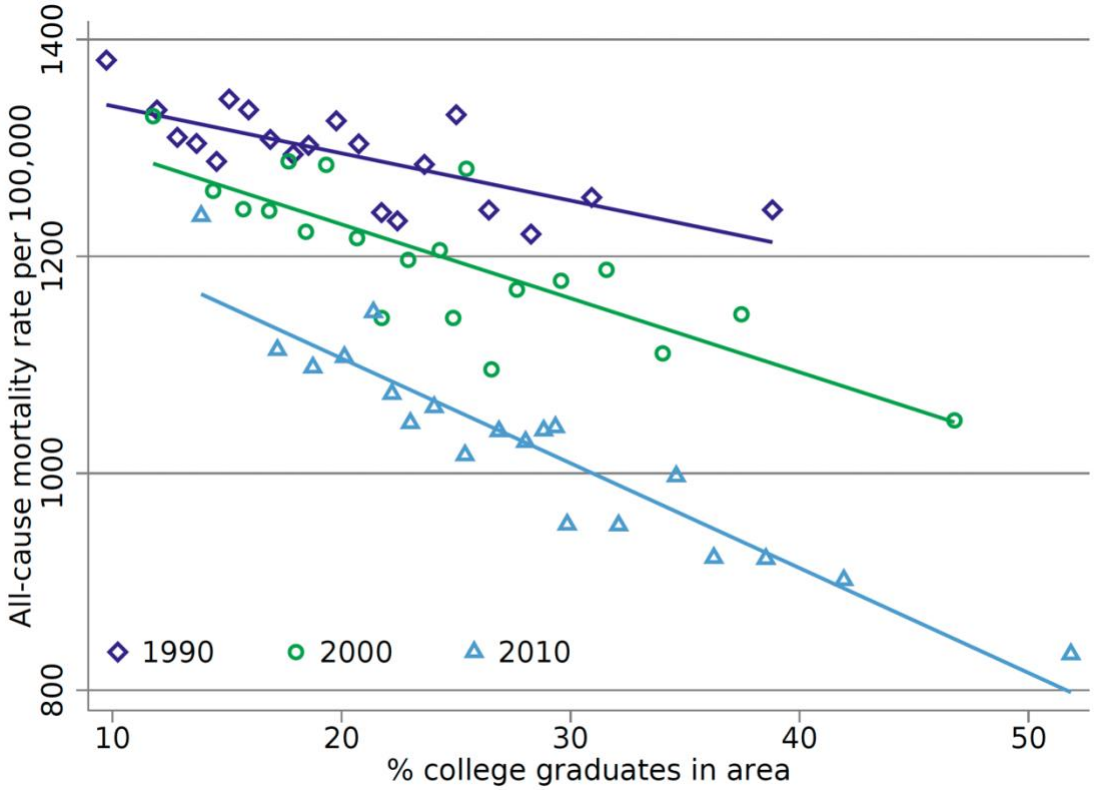
Tables and Figures

Table 1: Descriptive statistics on mortality and area characteristics

	Mean	SD
Cell characteristics		
Age 25-64	80.1%	---
Age 65+	19.9%	---
Female	51.8%	---
No high school	15.6%	---
High school graduate	36.9%	---
Some college	22.6%	---
College graduate	15.8%	---
Graduate education	9.0%	---
Missing education on death certificate	3.1%	4.3%
Mortality rates by cause (per 100,000)		
All cause	1,196	2,650
Heart disease	349	966
Cancer	289	518
Medically amenable causes	488	1,203
Smoking-related causes	672	1,653
Obesity-related causes	494	1,196
External causes	69	121
Area characteristics		
% college graduates	24.9%	8.8%
% Black	11.6%	11.4%
% Hispanic	13.2%	14.7%
Density (persons per square mile)	1,804	6,007
Population	1,878,063	2,051,136
Industry share: manufacturing	8.1%	3.6%
Number of observations		
Area-year-age-sex-race-education cells	369,707	---
Areas	486	---
Population	495,778,966	---
Deaths	5,934,489	---

Note. Death data by county-year-age-sex-education comes from the 1990, 2000, and 2010 Multiple Cause Mortality Files. Counties were aggregated to areas representing consistent public use microdata areas (CONSPUMAs). Mortality rates were calculated using population sizes from the 1990 and 2000 Census 5% samples, and the 2009-2011 ACS 5-year file for 2010. We exclude county-year-age-race-sex cells where 25% or more of reported deaths lacked education data. Statistics are weighted by cell size.

Figure 1: The relationship between area human capital and adjusted mortality per 100,000



Note. This graph is a binned scatter plot showing all-cause mortality across ventiles of area education in each year using mortality data that includes those with missing individual education. Each point includes approximately 5% of the population in that year, plotted at the mean percent college graduates and mean mortality rate (adjusted for age-sex using direct adjustment) across areas within each bin. The coefficients (and standard errors in parentheses) of the corresponding OLS regressions are -4.4*** (0.97) in 1990, -6.8*** (0.73) in 2000, and -9.7*** (0.97) in 2010. Fitted lines extend to the full range of the underlying data in each year. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

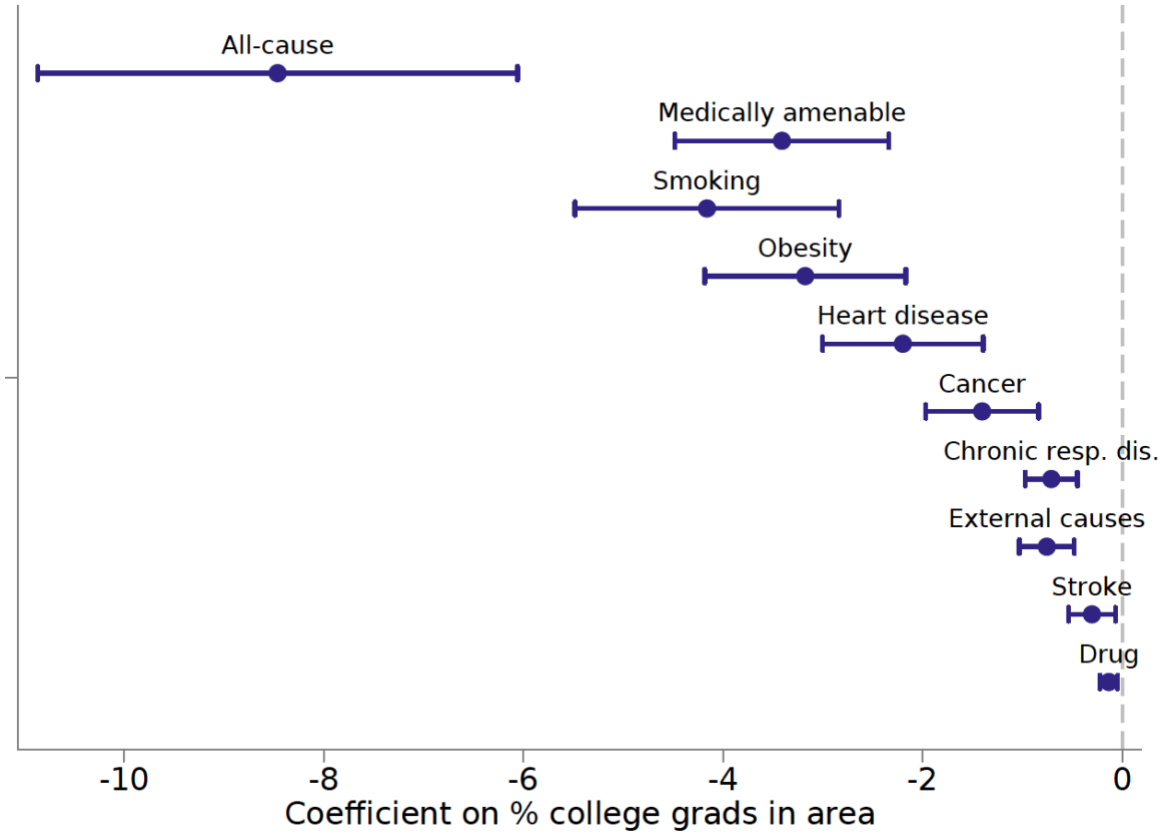
Table 2: Regression results of all-cause mortality rates per 100,000 on area human capital

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Area characteristics								
% college graduates		-10.30***	-6.41***	-0.72	-8.46***	-6.00***	-8.22***	-0.51
		(1.38)	(1.31)	(1.67)	(1.22)	(1.48)	(1.80)	(2.31)
% college graduates * year=2000				-4.25***		-1.47**		-2.09***
				(1.06)		(0.65)		(0.69)
% college graduates * year=2010				-7.32***		-3.35***		-4.22***
				(1.28)		(0.75)		(0.84)
Log density (pop/sq mi)	-7.42	-4.18	-5.89	-6.12	-3.35	-3.55	-1401.06**	-1029.62*
	(6.39)	(7.08)	(6.89)	(6.72)	(5.44)	(5.41)	(585.39)	(561.99)
Log population	-1.93	-1.04	-1.12	-0.91	-2.71	-2.29	1377.60**	1007.31*
	(4.77)	(4.87)	(4.76)	(4.78)	(4.18)	(4.17)	(584.55)	(560.48)
% Black	2.92***	2.45***	2.66***	2.71***	1.76***	1.83***	5.25**	6.35***
	(0.61)	(0.63)	(0.61)	(0.60)	(0.59)	(0.59)	(2.26)	(2.21)
% Hispanic	1.16*	0.21	1.37**	1.15*	-0.67	-0.75	-2.21	-2.98*
	(0.64)	(0.63)	(0.63)	(0.62)	(0.50)	(0.51)	(1.62)	(1.54)
Cell characteristics (ref. group: no high school)								
High school graduate	-265.23***		-265.65***	-266.58***	-263.47***	-263.81***	-261.23***	-261.43***
	(40.31)		(40.32)	(40.36)	(40.54)	(40.56)	(40.47)	(40.48)
Some college	-448.30***		-446.65***	-448.31***	-446.51***	-447.03***	-443.81***	-444.09***
	(29.07)		(29.06)	(29.12)	(29.26)	(29.30)	(29.17)	(29.18)
College graduate	-543.94***		-539.04***	-540.12***	-537.61***	-537.98***	-534.69***	-534.93***
	(35.32)		(35.23)	(35.26)	(35.42)	(35.44)	(35.33)	(35.34)
Post-graduate education	-729.68***		-724.10***	-724.89***	-722.09***	-722.38***	-720.41***	-720.49***
	(36.64)		(36.47)	(36.51)	(36.69)	(36.72)	(36.53)	(36.54)
% deaths with missing education	-7.10***	-6.22***	-7.32***	-7.02***	-4.41***	-4.27***	-3.94**	-3.84**
	(2.25)	(2.32)	(2.27)	(2.31)	(1.61)	(1.61)	(1.74)	(1.75)
State-year FE	No	No	No	No	Yes	Yes	Yes	Yes
Area FE	No	No	No	No	No	No	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966
Area-year-age-sex-race-educ cells	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707
Areas	486	486	486	486	486	486	486	486
R-squared	0.860	0.855	0.860	0.860	0.862	0.862	0.862	0.862
Dependent var. mean	1,196	1,196	1,196	1,196	1,196	1,196	1,196	1,196
% change from 10pp increase in % college grads		*-8.6*	*-5.4*		*-7.1*		*-6.9*	*-0.4*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

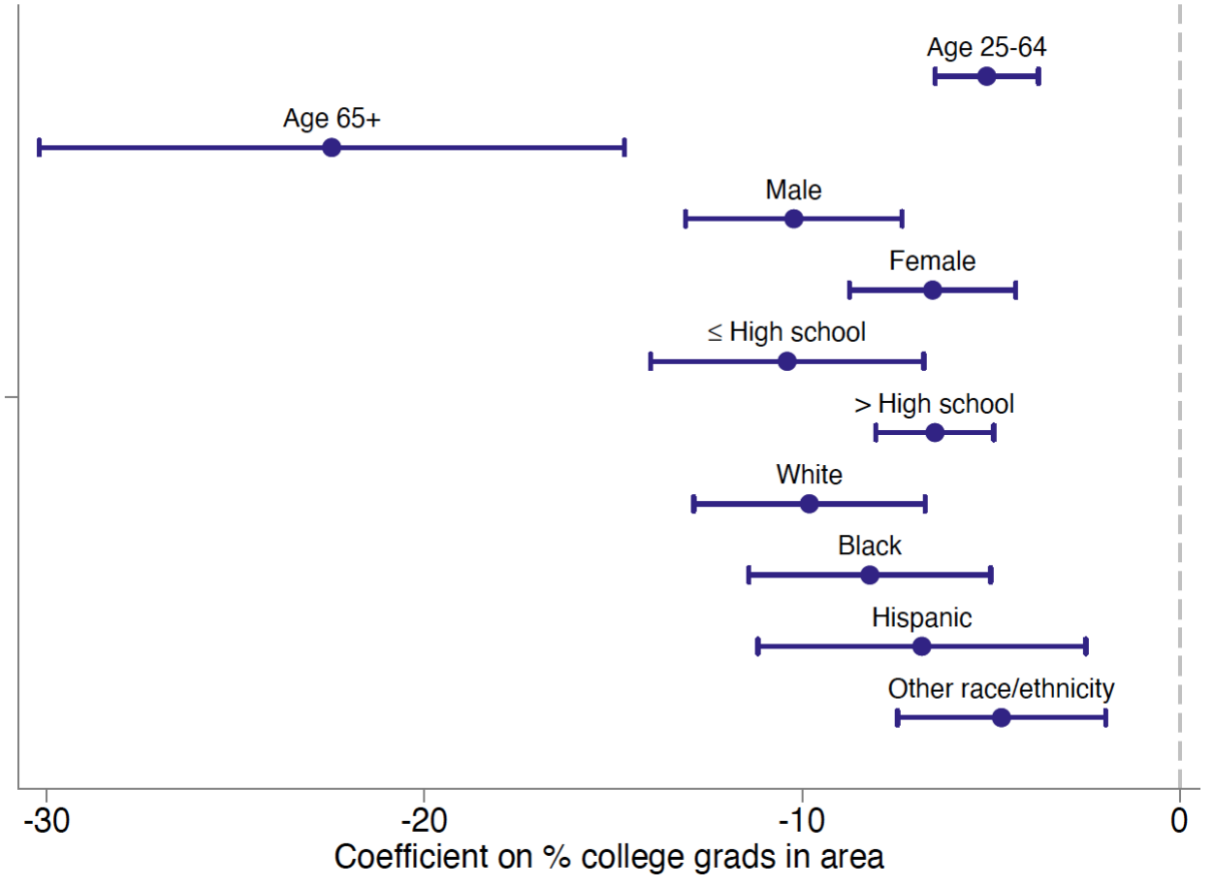
Note. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, year.

Figure 2: Regression results of cause-specific mortality rates per 100,000 on area human capital



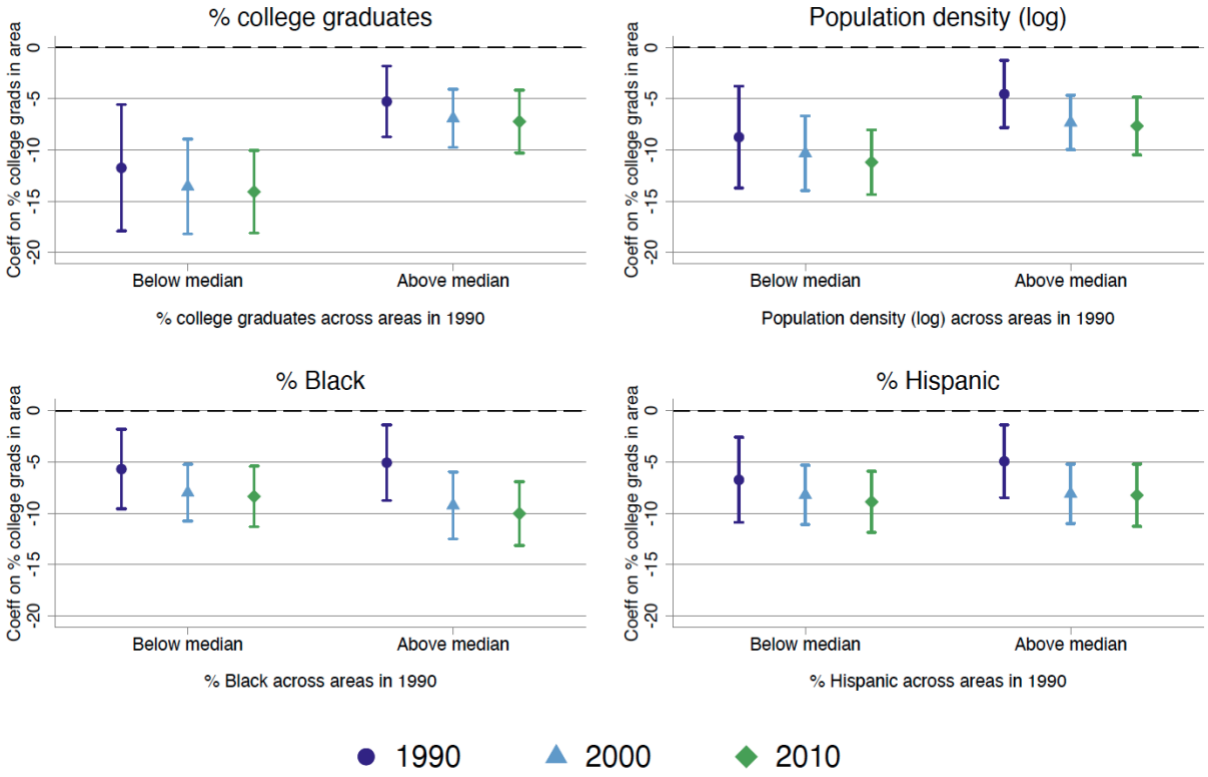
Note. This figure plots the coefficient on area human capital estimated separately for each cause of death. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Smoking-related, medically amenable, and obesity-related causes of death include all deaths to causes associated with that risk factor and are not mutually exclusive categories (see Appendix B for details). Confidence intervals are clustered at the area level.

Figure 3: Regression results of all-cause mortality rates per 100,000 on area human capital by demographic subgroups



Note. This figure plots the coefficient on area human capital estimated separately for each subgroup. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.

Figure 4: Regression results of all-cause mortality rates per 100,000 on area human capital by area characteristics in 1990



Note. This figure plots the coefficient on area human capital interacted by whether the area-year-age-sex-race-education cell is in an area above/below median percent college graduates, population density (log), percent Black, or percent Hispanic across areas in 1990, weighted by population. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.

Table 3: Regression results of non-fatal health outcomes on area human capital

	(1) **Cancer per 100,000**	(2) **Lung disease per 100,000**	(3) **Diabetes per 100,000**	(4) **Heart disease per 100,000**	(5) **Stroke per 100,000**	(6) **Good, very good, or excellent self-reported health**	(7) **Poor health, number of days in last 30 days**
	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	HRS 1992-2008	BRFSS 1999-2001 2009-2011	BRFSS 1999-2001 2009-2011
Area characteristics							
% college grads	6.5	-18.7**	-7.4	-37.8**	9.1	0.00215***	-0.03897***
	(11.5)	(8.2)	(10.5)	(16.0)	(9.1)	(0.00025)	(0.00517)
Cell characteristics (ref. group: no high school)							
High school graduate	-135.9	-883.2***	-1,051.5***	-1,080.2***	-461***	0.10657***	-2.16753***
	(132.6)	(140.6)	(181.1)	(220.1)	(102.2)	(0.00221)	(0.11493)
Some college	56.9	-980.1***	-1,185.6***	-959.4***	-625.9***	0.14587***	-2.54005***
	(135.5)	(158.6)	(213.0)	(282.1)	(130.4)	(0.00261)	(0.12252)
College graduate	-101.7	-1,676.3***	-1,843.4***	-2,372.7***	-879.8***	0.23313***	-3.91896***
	(162.7)	(152.3)	(195.7)	(256.7)	(118.0)	(0.00357)	(0.12271)
Observations	113,890	115,694	108,075	100,174	119,554	1,553,211	1,459,505
R-squared	0.0307	0.0228	0.0321	0.0579	0.0346	0.116	0.048
Dependent var. mean	2,800	2,200	3,300	6,200	1,700	0.834	3.774
% increase from 10pp increase in % college graduates	*2.3%*	*-8.5%*	*-2.2%*	*-6.1%*	*5.3%*	*2.6%*	*-10.3%*

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the area level.

Note. Cancer, lung disease, diabetes, heart disease, stroke, and hospitalizations in the Health and Retirement Survey (HRS) were defined as conditions or hospitalizations reported since the prior wave. Area characteristics in the HRS are measured at the time of HRS entry. OLS regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, Behavioral Risk Factor Surveillance System (BRFSS) state-year fixed effects or HRS wave fixed effects. We exclude individuals with missing education. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

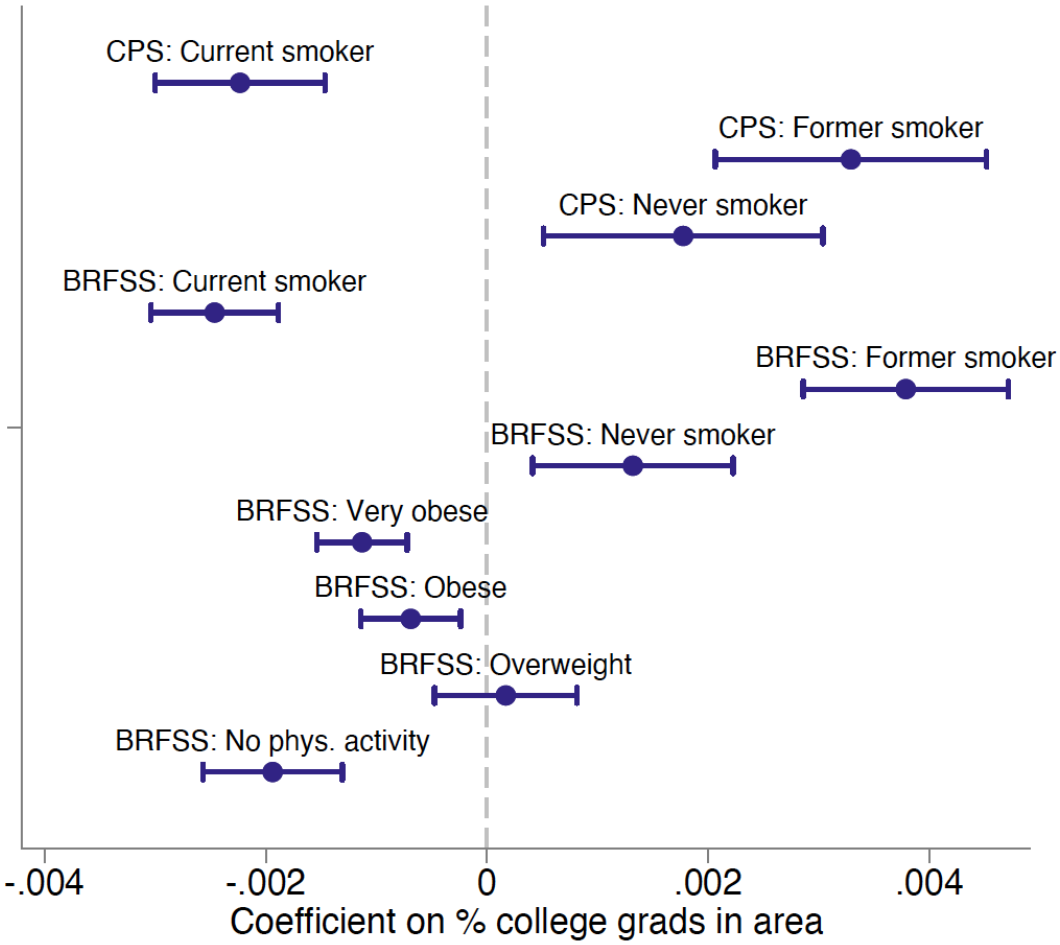
Table 4: Spatial sorting, baseline health status, and selective migration

Dependent var.	(1) **Migrated to different county in next 2 years?**	(2) **% college grads in destination county - minus - % college grads in county of origin**	(3) **Migrated to different SMSA/county between interviews?**	(4) **Stayed in SMSA or moved to SMSA from non-SMSA?**	(5) **Migrated to different SMSA/county between interviews?**
	HRS 1992-2008	HRS 1992-2008	NLSY Young Women 1968-2001	NLSY Young Women 1968-2001	NLSY Young Men 1969-1971, 1976
Model	Probit, dy/dx (SE)	OLS, coef. (SE)	Probit, dy/dx (SE)	Probit, dy/dx (SE)	Probit, dy/dx (SE)
Baseline health status: predicted mortality until next interview	0.051** (0.022)	0.092 (5.23)	0.162** (0.064)	0.568 (0.385)	-0.114 (0.172)
Observations	71,717	3,101	50,722	3,010	4,527
R-squared	0.0031	0.017	0.0341	0.08	0.0318
Dependent var. mean	0.043	-0.42	0.094	0.573	0.092

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the individual-level.

Note. All regressions use sampling weights and control for individual education, 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, National Longitudinal Survey of Youth (NLSY) wave or Health and Retirement Survey (HRS) survey wave, Black, and Hispanic. In the NLSY Young Men regressions, we use 1-year age categories instead of 5-year due to similar ages in the sample. Columns 1-2 additionally control for area characteristics: log population, log density, and industry shares. Baseline health in the HRS regressions was measured as the probability of death between the current and next interview and as probability of dying by 2011 in the NLSY regressions; it is estimated in a separate probit regression of mortality on measures of health status in the current interview and demographics.

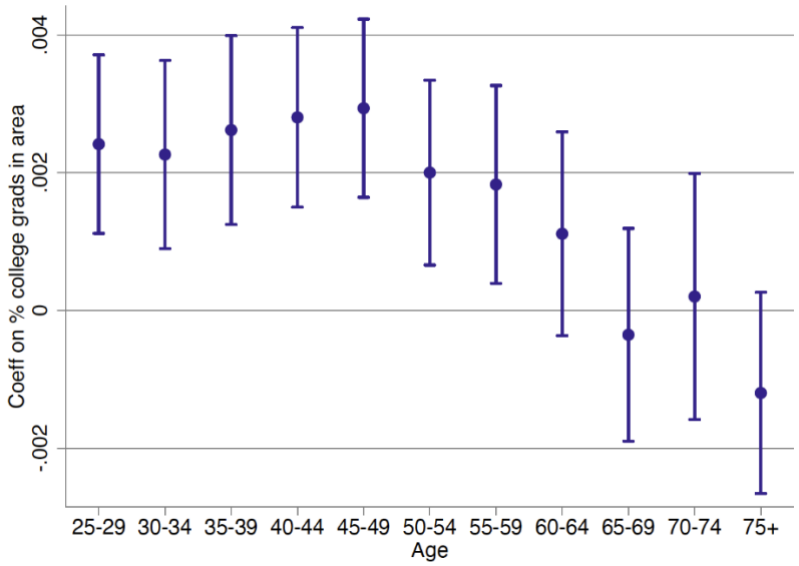
Figure 5: Regression results of health-related behaviors on area human capital



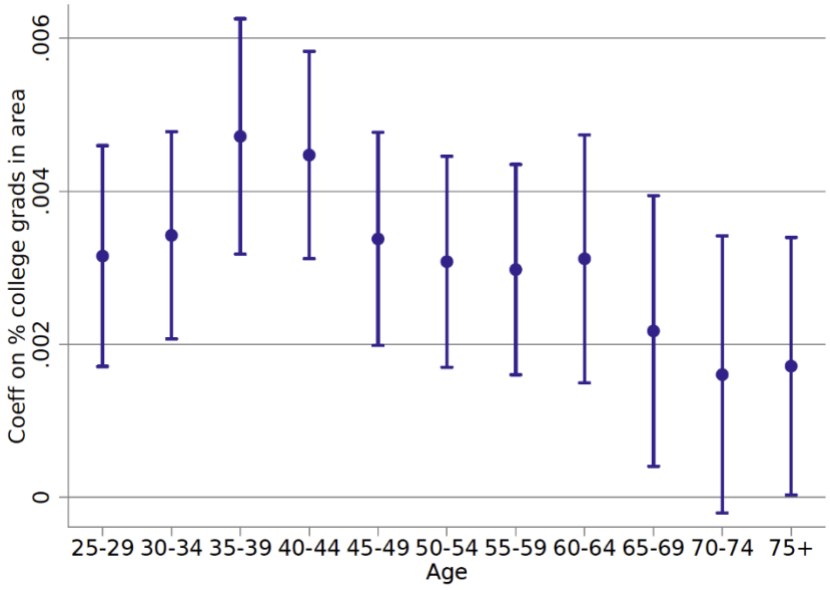
Note. This figure plots the coefficient on area human capital estimated separately for each smoking-related and obesity-related behavior, all of which are defined as binary variables. Former smoker is defined conditional on ever smoking. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS) or the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

Figure 6: Regression results of smoking on area human capital interacted with age

(a) Never smoker



(b) Former smoker



Note. This figure plots the coefficient on area human capital interacted by age and estimated separately for each smoking-related behavior, all of which are defined as binary variables. Former smoker is defined conditional on ever smoking. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

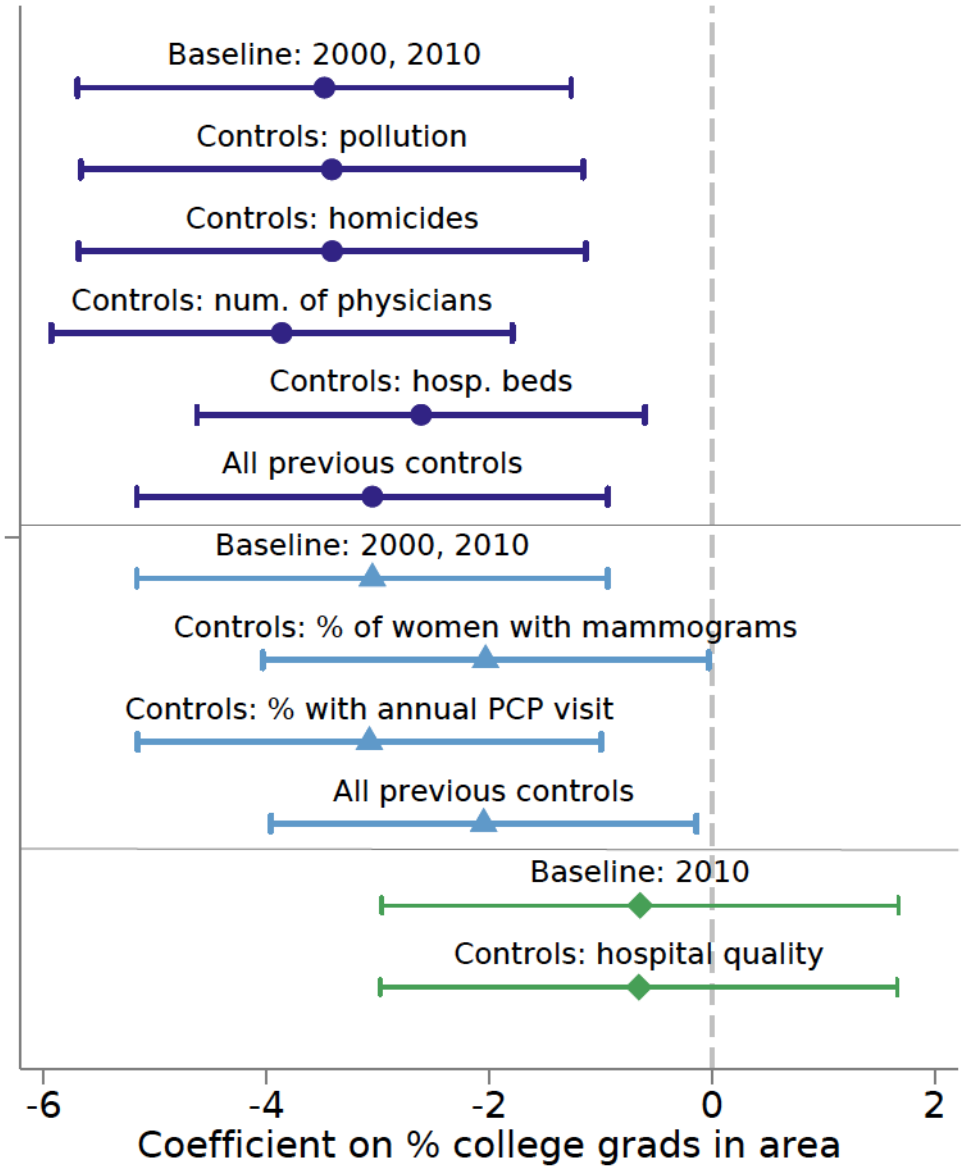
Table 5: Regression results of all-cause mortality per 100,000 on area human capital and health-related behaviors

	(1)	(2)	(3)	(4)	(5)	(6)
	Cells with non-missing data on smoking behavior		**Cells with non-missing data on smoking behavior and obesity-related behaviors**			
	1990-2010		2000-2010			
Area characteristics						
% college graduates	-8.97***	-5.58***	-8.53***	-4.63***	-7.51***	-3.48***
	(1.20)	(1.09)	(1.22)	(1.14)	(1.22)	(1.13)
% current smoker		5.24**		7.23***		6.76**
		(2.19)		(2.76)		(2.70)
% former smoker		-229.65		-165.63		-205.95
		(142.57)		(184.21)		(176.17)
% overweight, obese, very obese					3.88***	4.18***
					(1.08)	(1.09)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	433,629,834	433,629,834	357,070,962	357,070,962	357,070,962	357,070,962
Cells	308,969	308,969	257,906	257,906	257,906	257,906
Areas	485	485	485	485	485	485
R-squared	0.871	0.871	0.879	0.879	0.879	0.879
Dependent var. mean	1,192	1,192	1,206	1,206	1,206	1,206
% change from 10pp increase in % college grads	*-7.5*	*-4.7*	*-7.1*	*-3.8*	-6.2	*-2.9*

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. The percentage of current or former smokers was calculated using the 1995-1996, 1998-1999 CPS, 1999-2001 BRFSS, and 2009-2011 BRFSS. The percent of individuals that were overweight, obese, or very obese, and those with no physical activity were calculated using the 1999-2001 and 2009-2011 BRFSS.

Figure 7: Regression results of all-cause mortality per 100,000 on area human capital and health-related amenities



Note. All regressions are estimated separately at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. The main control in the baseline regressions is the percentage of individuals currently smoking, the percentage of individuals formerly smoking (conditional on ever smoking), and the percentage of individuals who are overweight, obese, or very obese. All regressions further control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.

Table 6: Regression results of workplace smoking policies on area human capital

	(1) Any official smoking policy	(2) Smoking ban in all areas of the office
Area characteristics		
% college graduates	0.00033	0.00181**
	(0.00048)	(0.00071)
Individual characteristics (ref. group: no high school)		
High school graduate	0.03908***	0.02555***
	(0.00382)	(0.00378)
Some college	0.06120***	0.04699***
	(0.00399)	(0.00449)
College graduate	0.08425***	0.08420***
	(0.00383)	(0.00533)
Post-graduate education	0.10847***	0.11909***
	(0.00450)	(0.00629)
Individual chars	Yes	Yes
State-year FE	Yes	Yes
Area chars	Yes	Yes
Area-level industry shares	Yes	Yes
Weighted obs.	200,075,093	180,130,632
Cells	192,286	173,071
Areas	297	297
R-squared	0.055	0.058
Dependent var. mean	0.908	0.852
% change from 10pp increase in % college grads	*0.4*	*2.1*

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the area level.

Note. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015, use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

Table 7: Regression results of beliefs about smoking on area human capital

	(1)	(2)	(3)	(4)	(5)	(6)
	Strongly agree or agree with the following statement					
	"Smoking by pregnant women is harmful for baby"	**"Someone else's smoke is harmful"**	**"Most lung cancer deaths are caused by smoking"**	**"Smoking should be banned in bars"**	**"Smoking should be banned in restaurants"**	**"Smoking should be banned in work areas"**
Area characteristics						
% college grads	0.0103*	0.00328	0.0112***	0.00381***	0.00313*	0.00285***
	(0.00537)	(0.00336)	(0.00412)	(0.00124)	(0.00162)	(0.00107)
Individual characteristics (ref. group: no high school)						
High school graduate	0.218***	0.182***	0.122***	-0.03241***	-0.00892	0.01466***
	(0.0455)	(0.0294)	(0.0413)	(0.00518)	(0.00581)	(0.00439)
Some college	0.500***	0.335***	0.234***	-0.01654**	0.03696***	0.06028***
	(0.0595)	(0.0345)	(0.0471)	(0.00664)	(0.00791)	(0.00654)
College graduate	0.597***	0.541***	0.387***	0.04482***	0.09166***	0.11960***
	(0.0721)	(0.0377)	(0.0591)	(0.00699)	(0.00991)	(0.00800)
Post-graduate	0.771***	0.695***	0.552***	0.08092***	0.13279***	0.15032***
	(0.0782)	(0.0422)	(0.0595)	(0.00728)	(0.01146)	(0.00965)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	No	No	No	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes
Dependent var. mean	0.922	0.922	0.740	0.451	0.607	0.775
% change from 10pp increase in % college graduates	*11.2%*	*3.6%*	*15.1%*	*8.5%*	*5.2%*	*3.7%*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. Outcomes were defined relative to no opinion, disagreeing, or strongly disagreeing. All probit regressions in columns 1-3 pool data from National Health Interview Survey (NHIS) from 1987, 1992, and 2000. All probit regressions in columns 4-6 pool data from the Tobacco Use Supplement in the Current Population Survey (CPS). All regressions use sampling weights and include controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, and year. We exclude individuals with missing education. % college graduates was measured at the county level using data from the immediately preceding census for the given year for NHIS regressions and from the immediately preceding census or 3-year ACS for the CPS regressions. We also include controls for area log density and log population, % Black, % Hispanic, and industry shares, defined similarly as percent college graduates.

Table 8: Regression results of smoking behavior on area human capital, workplace smoking policies, and smoking beliefs

	(1) Current smoker	(2) Current smoker	(3) Current smoker	(4) Former smoker	(5) Former smoker	(6) Former smoker	(7) Never smoker	(8) Never smoker	(9) Never smoker
Area characteristics									
% college graduates	-0.00218***	-0.00191***	-0.00186***	0.00407***	0.00344***	0.00339***	0.00132*	0.00103	0.00098
	(0.00050)	(0.00049)	(0.00049)	(0.00092)	(0.00089)	(0.00088)	(0.00068)	(0.00066)	(0.00066)
Smoking should be banned in bars, restaurants, and work		-0.19644***	-0.19467***		0.26890***	0.26686***		0.20864***	0.20697***
		(0.00695)	(0.00689)		(0.00855)	(0.00850)		(0.00497)	(0.00495)
Smoking ban in all areas of the office			-0.02910***			0.04168***			0.03257***
			(0.00327)			(0.00600)			(0.00438)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	123,182,251	123,182,251	123,182,251	123,182,251	123,182,251	123,182,251	123,182,251	123,182,251	123,182,251
Cells	120,508	120,508	120,508	120,627	120,627	120,627	120,627	120,627	120,627
Areas	297	297	297	297	297	297	297	297	297
R-squared	0.082	0.143	0.144	0.091	0.129	0.129	0.080	0.113	0.114
Dependent var. mean	0.168	0.168	0.168	0.549	0.549	0.549	0.629	0.629	0.629
% change from 10pp increase in % college grads	*-13.0*	*-11.4*	*-11.1*	*7.4*	*6.3*	*6.2*	*2.1*	*1.6*	*1.6*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. All probit regressions pool data from the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015, use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and year. We exclude individuals with missing education. Area-level % college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, % Black, % Hispanic, and industry shares, defined similarly as % college graduates.

Appendix A: Data sources

Mortality data

Data type	Source	Years	Key variables
Mortality	Multiple Cause Mortality Files (MCMF)	1990, 2000, 2010	All-cause and cause-specific mortality, age, sex, race, education, county of residence
Population counts	U.S. Decennial Census (5% sample), American Community Survey (ACS)	1990 (Census), 2000 (Census), 2009-2011 (ACS)	

Data on non-fatal health outcome

Data type	Source	Years	Key variables
Disease prevalence	Health and Retirement Survey	1992-2008	Prevalence of cancer, lung disease, diabetes, heart disease, stroke
Self-reported health	Behavioral Risk Factor Surveillance System	1999-2001, 2009-2011	Good/very good/excellent self-reported health, number of days in poor mental or physical health

Data on health-related behaviors

Data type	Source	Years	Key variables
Smoking behavior	Behavioral Risk Factor Surveillance System	1999-2001, 2009-2011	Current former, never smoker
Smoking behavior	Current Population Survey	1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015	Current former, never smoker

Obesity and physical activity	Behavioral Risk Factor Surveillance System	1999-2001, 2009-2011	BMI, any physical activity in last 30 days

Area characteristics data

Data type	**Source**	**Years**	**Key variables**
Human capital	U.S. Decennial Census (5% sample) and ACS	1990 (Census), 2000 (Census), 2009-2011 (ACS)	% with at least a college degree
Demographics	U.S. Decennial Census (5% sample) and ACS	1990 (Census), 2000 (Census), 2009-2011 (ACS)	% Black, % Hispanic, industry shares
Demographics	Area Resource Files	1990, 2000, 2010	Population, land area
Healthcare	Area Resource Files	1990, 2000, 2010	Number of MDs per 1,000, number of hospital beds per 1,000
Healthcare	Dartmouth Health Atlas	2003-2015	% Medicare enrollees with annual ambulatory visit, % Medicare enrollees with mammogram
Homicides	Uniform Crime Reports	1989-1991, 1999-2001, 2009-2011	Homicide rate
Pollution	van Donkelaar (2019)	1999-2001, 2009-2011	PM-2.5
Pollution	Environmental Protection Agency (EPA)	1989-1991	PM-10
Hospital quality	Centers for Medicare and Medicaid Services (CMS) Hospital Compare Database	2003-2008	Process-of-care indicators for pneumonia, congestive heart failure, and acute myocardial infarction

Migration data

Cross-county migration	Health and Retirement Survey	1992-2008	Probability of changing county of residence in the next two years; difference in area human capital across counties of residence
Cross-county or cross-MSA migration	National Longitudinal Survey of Young Women and Men	1969-1971, 1976 (Young Men); 1968-2001 (Young Women)	Probability of changing county or SMSA of residence since last interview; moving to non-SMSA

Smoking regulations

Workplace smoking policies	Current Population Survey	1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015	Any official smoking policy in place for indoor workers, smoking ban in public or work areas at the workplace for individuals working in indoor workplace

Beliefs about smoking

Beliefs about harms of smoking	National Health Interview Survey	1987, 1992, 2000	Agreement with whether smoking is harmful for pregnant women, whether most lung cancer deaths are caused by smoking, whether someone else's smoke is harmful
Beliefs about smoking regulation	Current Population Survey	1995-1996, 1998-1999, 2001-2002, 2006-2007, 2014-2015	Whether smoking should be banned in bars, restaurants, workplaces

Appendix B: Definitions of mortality due to smoking-related, obesity-related, medically amenable, and external causes

Smoking-related

Malignant Neoplasms: of the Lip, Oral Cavity, Pharynx, Esophagus, Stomach, Pancreas, Larynx, Trachea, Lung, Bronchus, Cervix Uteri, Kidney and Renal Pelvis, Urinary Bladder, and Acute Myeloid Leukemia; Cardiovascular Diseases: Ischemic Heart Disease, Other Heart Disease, Cerebrovascular Disease, Atherosclerosis, Aortic Aneurysm, Other Arterial Disease; Respiratory Diseases: Pneumonia, Influenza, Bronchitis, Emphysema, Chronic Airway Obstruction.

Source: CDC's National Center for Chronic Disease Prevention and Health Promotion (2014).

Obesity-related

Coronary Heart Disease, Other Cardiovascular Diseases; Cancers of the Colon, Breast, Esophagus, Uterus, Ovaries, Kidney, and Pancreas; Diabetes, and Kidney Disease.

Source: Flegal et al. (2007).

Medically amenable

Intestinal Infections, Tuberculosis, Other Infections (Diphtheria, Tetanus, Septicaemia, Poliomyelitis), Whooping Cough, Measles; Malignant Neoplasms of: Colon and Rectum, Skin, Breast, Cervix Uteri, Uterus, Testis; Hodgkin's Disease, Leukaemia, Diseases of the Thyroid, Diabetes, Epilepsy, Chronic Rheumatic Heart Disease, Hypertensive Disease, Ischaemic Heart Disease (50% of all such deaths), Cerebrovascular Disease, All Respiratory Diseases, Peptic Ulcer, Appendicitis, Abdominal Hernia, Cholelithiasis and Cholecystitis, Nephritis and Nephrosis, Benign Prostatic Hyperplasia, Misadventures to Patients during Surgical and Medical Care, Maternal Death, Congenital Cardiovascular Anomalies, Perinatal Deaths (excl. stillbirths).

Source: Nolte & McKee (2008).

External causes

Accidents, Intentional Self-Harm, Assault, Events of Undetermined Intent, Legal Intervention, Operations of War and Their Sequelae, Complications of Medical and Surgical Care.

Source: ICD-10-CM Codes V01-Y9.

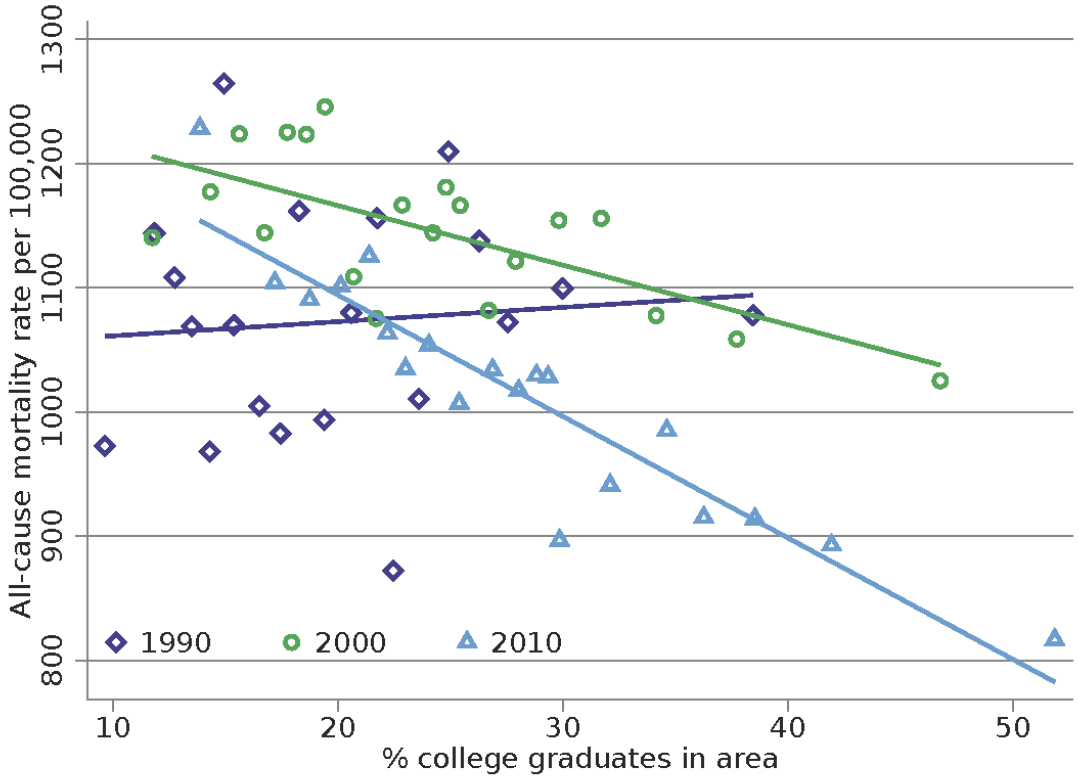
Drug poisoning (overdose)

Deaths from unintentional overdose of a drug, suicide, or drug poisoning of undetermined intent

Source: ICD-10-CM Codes X40–X44, X60–X64, or Y10–Y14.

Appendix C: Additional Tables and Figures

Appendix Figure C1: The relationship between area human capital and adjusted mortality per 100,000



Note. This graph is a binned scatter plot showing all-cause mortality across ventiles of area education in each year for all data in our sample (excluding deaths with missing individual education). Each point includes approximately 5% of the population in that year, plotted at the mean percent college graduates and mean mortality rate (adjusted for age-sex using direct adjustment) across areas within each bin. The coefficients (and standard errors in parentheses) of the corresponding OLS regressions are -1.2 (2.2) in 1990, -4.8*** (0.98) in 2000, and -9.8*** (0.96) in 2010. Fitted lines extend to the full range of the underlying data in each year. *** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Appendix Table C1: Regression results of cause-specific mortality rates per 100,000 on area human capital

	(1) **All-cause**	(2) **Medically amenable**	(3) **Smoking**	(4) **Obesity**	(5) **Heart disease**	(6) **Cancer**	(7) **Chronic resp. dis.**	(8) **External causes**	(9) **Stroke**	(10) **Drug poisoning**
Area characteristics										
% college graduates	-8.46***	-3.41***	-4.16***	-3.18***	-2.20***	-1.40***	-0.71***	-0.75***	-0.30**	-0.13***
	(1.22)	(0.55)	(0.67)	(0.51)	(0.41)	(0.29)	(0.13)	(0.14)	(0.12)	(0.04)
Cell characteristics (ref. group: no high school)										
High school graduate	-263.47***	-122.73***	-191.17***	-109.50***	-98.86***	-19.99**	-29.11***	-25.87***	-17.81***	-5.56***
	(40.54)	(18.03)	(26.28)	(17.82)	(13.75)	(8.95)	(3.07)	(1.90)	(2.87)	(0.43)
Some college	-446.51***	-188.44***	-298.10***	-179.06***	-152.48***	-64.15***	-42.07***	-50.50***	-22.18***	-9.75***
	(29.26)	(13.21)	(19.45)	(12.72)	(10.01)	(6.30)	(2.66)	(1.75)	(2.14)	(0.48)
College graduate	-537.61***	-220.75***	-343.36***	-204.21***	-168.76***	-82.50***	-52.75***	-65.79***	-23.09***	-15.53***
	(35.42)	(15.72)	(22.88)	(15.01)	(11.55)	(7.84)	(3.16)	(2.16)	(2.52)	(0.60)
Post-graduate	-722.09***	-300.69***	-468.79***	-283.60***	-229.10***	-131.24***	-68.78***	-68.04***	-32.36***	-16.11***
education	(36.69)	(16.37)	(23.72)	(15.34)	(11.74)	(8.24)	(3.44)	(2.21)	(2.58)	(0.60)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966
Cells	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707
Areas	486	486	486	486	486	486	486	486	486	486
R-squared	0.862	0.824	0.828	0.811	0.795	0.710	0.563	0.252	0.648	0.111
Dependent var. mean	1,196	488	672	494	349	289	62	69	77	9
% change from 10pp increase in % college grads	*-7.1*	*-7.0*	*-6.2*	*-6.4*	*-6.3*	*-4.9*	*-11.4*	*-10.9*	*-3.9*	*-14.8*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

Appendix Table C2: Regression results of cause-specific mortality rates per 100,000 on area human capital by year

	(1) **All-cause**	(2) **Medically amenable**	(3) **Smoking**	(4) **Obesity**	(5) **Heart disease**	(6) **Cancer**	(7) **Chronic resp. dis.**	(8) **External causes**	(9) **Stroke**	(10) **Drug poisoning**
Area characteristics										
% college graduates	-6.00***	-2.27***	-4.03***	-3.15***	-2.76***	-0.86**	-0.21	-0.56***	-0.25	-0.05
	(1.48)	(0.66)	(0.85)	(0.63)	(0.51)	(0.36)	(0.16)	(0.17)	(0.15)	(0.05)
% college graduates * year=2000	-1.47**	-0.81***	0.04	-0.25	0.30	-0.47***	-0.30***	-0.10	0.02	0.00
	(0.65)	(0.31)	(0.42)	(0.33)	(0.28)	(0.16)	(0.07)	(0.10)	(0.09)	(0.03)
% college graduates * year=2010	-3.35***	-1.47***	-0.24	0.10	0.78**	-0.65***	-0.67***	-0.28**	-0.10	-0.14***
	(0.75)	(0.34)	(0.47)	(0.36)	(0.31)	(0.18)	(0.09)	(0.11)	(0.10)	(0.04)
Cell characteristics (ref. group: no high school)										
High school graduate	-263.81***	-122.88***	-191.19***	-109.49***	-98.78***	-20.06**	-29.17***	-25.90***	-17.82***	-5.57***
	(40.56)	(18.04)	(26.29)	(17.83)	(13.76)	(8.95)	(3.07)	(1.90)	(2.87)	(0.43)
Some college	-447.03***	-188.67***	-298.14***	-179.04***	-152.35***	-64.25***	-42.17***	-50.54***	-22.20***	-9.77***
	(29.30)	(13.23)	(19.47)	(12.74)	(10.02)	(6.31)	(2.66)	(1.76)	(2.14)	(0.48)
College graduate	-537.98***	-220.91***	-343.39***	-204.20***	-168.67***	-82.57***	-52.83***	-65.82***	-23.10***	-15.54***
	(35.44)	(15.73)	(22.89)	(15.02)	(11.56)	(7.85)	(3.17)	(2.16)	(2.52)	(0.60)
Post-graduate education	-722.38***	-300.82***	-468.80***	-283.60***	-229.03***	-131.30***	-68.84***	-68.06***	-32.37***	-16.13***
	(36.72)	(16.38)	(23.73)	(15.35)	(11.75)	(8.25)	(3.44)	(2.21)	(2.58)	(0.60)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966	495,778,966
Cells	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707	369,707
Areas	486	486	486	486	486	486	486	486	486	486
R-squared	0.862	0.824	0.828	0.811	0.795	0.710	0.563	0.252	0.648	0.111
Dependent var. mean	1,196	488	672	494	349	289	62	69	77	9

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

Appendix Table C3: Regression results of mortality rates per 100,000 on area human capital by demographic subgroup

	(1) **Age 25-64**	(2) **Age 65+**	(3) **Male**	(4) **Female**	(5) **<= High school**	(6) **>= Some college**	(7) **White**	(8) **Black**	(9) **Hispanic**	(10) **Other race/ethnicity**
Area characteristics										
% college graduates	-5.11***	-22.46***	-10.22***	-6.54***	-10.40***	-6.48***	-9.81***	-8.21***	-6.83***	-4.72***
	(0.70)	(3.94)	(1.46)	(1.12)	(1.84)	(0.79)	(1.56)	(1.63)	(2.21)	(1.40)
Cell characteristics (ref. group: no high school)										
High school graduate	-210.80***	-228.68**	-332.57***	-192.10***	-221.45***	--	-416.12***	-108.01**	10.65	-76.25
	(16.20)	(105.39)	(41.54)	(39.93)	(40.88)	--	(49.36)	(46.61)	(28.15)	(46.38)
Some college	-347.65***	-613.23***	-615.91***	-286.73***	--	242.29***	-598.28***	-393.96***	-86.43***	-173.40***
	(14.62)	(57.13)	(30.96)	(28.65)	--	(9.19)	(36.02)	(31.54)	(15.25)	(44.77)
College graduate	-413.81***	-898.80***	-725.04***	-355.09***	--	148.01***	-699.62***	-470.64***	-109.05***	-159.81***
	(15.91)	(89.43)	(37.66)	(33.90)	--	(7.28)	(41.76)	(41.39)	(31.97)	(50.23)
Post-graduate	-483.75***	-1613.11***	-942.33***	-495.44***	--	--	-891.86***	-726.68***	-230.46***	-203.69***
education	(17.01)	(83.40)	(39.29)	(34.61)	--	--	(40.73)	(35.70)	(35.88)	(46.05)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	396,880,881	98,898,085	238,768,366	257,010,600	260,481,118	235,297,848	370,677,266	51,326,357	50,075,991	23,699,352
Cells	240,034	129,673	188,263	181,444	161,373	208,334	172,361	86,386	57,403	53,557
Areas	486	485	485	486	486	486	485	477	485	480
R-squared	0.626	0.793	0.842	0.882	0.882	0.811	0.887	0.716	0.675	0.582
Dependent var. mean	364	4,536	1,232	1,164	1,661	682	1,333	1,197	528	470
% change from 10pp increase in % college grads	*-14.0*	*-5.0*	*-8.3*	*-5.6*	*-6.3*	*-9.5*	*-7.4*	*-6.9*	*-12.9*	*-10.0*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

Appendix Table C4: Regression results of mortality per 100,000 on area human capital by demographic subgroup, by year

	(1) Age 25-64	(2) Age 65+	(3) Male	(4) Female	(5) <= High school	(6) >= Some college	(7) White	(8) Black	(9) Hispanic	(10) Other race/ethnicity
Area characteristics										
% college graduates	-3.09***	-17.05***	-8.16***	-3.54**	-5.93**	-3.51***	-6.75***	-6.44***	-11.19**	-5.08***
	(0.77)	(5.07)	(1.65)	(1.42)	(2.31)	(0.87)	(1.83)	(2.36)	(5.14)	(1.86)
% college graduates * year=2000	-1.42***	-0.10	-1.10	-1.94***	-1.75*	-3.10***	-1.49**	-1.39	4.07	1.88*
	(0.42)	(2.29)	(0.87)	(0.65)	(0.99)	(0.52)	(0.72)	(1.46)	(3.53)	(1.13)
% college graduates * year=2010	-2.65***	-8.63***	-2.87***	-3.99***	-6.66***	-3.29***	-4.50***	-2.00	4.46	-0.20
	(0.48)	(2.60)	(0.91)	(0.79)	(1.24)	(0.56)	(0.83)	(1.65)	(3.83)	(1.38)
Cell characteristics (ref. group: no high school)										
High school graduate	-211.00***	-229.91**	-332.84***	-192.53***	-221.62***	0.00	-416.87***	-108.11**	10.72	-76.08
	(16.22)	(105.47)	(41.56)	(39.96)	(40.89)	(.)	(49.40)	(46.62)	(28.14)	(46.43)
Some college	-348.01***	-614.63***	-616.28***	-287.44***	0.00	242.33***	-599.30***	-394.07***	-86.28***	-173.38***
	(14.64)	(57.21)	(30.98)	(28.69)	(.)	(9.19)	(36.07)	(31.56)	(15.23)	(44.81)
College graduate	-414.07***	-899.31***	-725.37***	-355.51***	0.00	147.99***	-700.31***	-470.66***	-108.95***	-159.74***
	(15.93)	(89.46)	(37.69)	(33.92)	(.)	(7.28)	(41.80)	(41.39)	(31.96)	(50.25)
Post-graduate education	-483.98***	-1612.54***	-942.75***	-495.52***	0.00	0.00	-892.41***	-726.78***	-230.26***	-203.44***
	(17.03)	(83.37)	(39.33)	(34.61)	(.)	(.)	(40.76)	(35.71)	(35.85)	(46.07)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	396,880,881	98,898,085	238,768,366	257,010,600	260,481,118	235,297,848	370,677,266	51,326,357	50,075,991	23,699,352
Cells	240,034	129,673	188,263	181,444	161,373	208,334	172,361	86,386	57,403	53,557
Areas	486	485	485	486	486	486	485	477	485	480
R-squared	0.626	0.793	0.842	0.882	0.882	0.811	0.887	0.716	0.675	0.582
Dependent var. mean	364	4,536	1,232	1,164	1,661	682	1,333	1,197	528	470

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and state-year fixed effects. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

Appendix Table C5: Regression results of smoking on area human capital

	(1) **CPS: Current smoker**	(2) **CPS: Former smoker**	(3) **CPS: Never smoker**	(4) **BRFSS: Current smoker**	(5) **BRFSS: Former smoker**	(6) **BRFSS: Never smoker**
Area characteristics						
% college graduates	-0.00223***	0.00329***	0.00177***	-0.00246***	0.00379***	0.00132***
	(0.00039)	(0.00063)	(0.00064)	(0.00029)	(0.00047)	(0.00046)
Individual characteristics (ref. group: no high school)						
High school graduate	-0.02866***	0.06475***	0.00256	-0.05600***	0.06444***	0.04979***
	(0.00461)	(0.00510)	(0.00518)	(0.00508)	(0.00520)	(0.00668)
Some college	-0.06789***	0.13527***	0.02177***	-0.10064***	0.12258***	0.08155***
	(0.00627)	(0.00543)	(0.00719)	(0.00573)	(0.00554)	(0.00757)
College graduate	-0.16886***	0.23568***	0.15031***	-0.23354***	0.24969***	0.24235***
	(0.01004)	(0.00678)	(0.01135)	(0.00802)	(0.00651)	(0.00952)
Post-graduate education	-0.22027***	0.30450***	0.19999***	--	--	--
	(0.01196)	(0.00943)	(0.01264)	--	--	--
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	613,858	238,830	613,858	1,551,524	738,024	1,551,524
Cells	613,858	238,830	613,858	1,551,524	738,024	1,551,524
Areas	297	297	297	484	484	484
R-squared	0.086	0.123	0.081	0.088	0.121	0.063
Dependent var. mean	0.165	0.561	0.624	0.192	0.585	0.538
% change from 10pp increase in % college grads	*-13.5*	*5.9*	*2.8*	*-12.8*	*6.5*	*2.5*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS) or the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

Appendix Table C6: Regression results of obesity on area human capital

	(1) **BRFSS: Very obese**	(2) **BRFSS: Obese**	(3) **BRFSS: Overweight**	(4) **BRFSS: No physical activity**
Area characteristics				
% college graduates	-0.00113***	-0.00069***	0.00017	-0.00194***
	(0.00021)	(0.00023)	(0.00033)	(0.00032)
Individual characteristics (ref. group: no high school)				
High school graduate	-0.01824***	-0.01485***	0.01653***	-0.07400***
	(0.00172)	(0.00266)	(0.00357)	(0.00292)
Some college	-0.02380***	-0.01687***	0.01938***	-0.14805***
	(0.00170)	(0.00274)	(0.00347)	(0.00261)
College graduate	-0.06571***	-0.05732***	0.02175***	-0.24288***
	(0.00186)	(0.00320)	(0.00382)	(0.00378)
Individual chars	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes
Weighted obs.	1,490,722	1,490,722	1,490,722	1,452,041
Cells	1,490,722	1,490,722	1,490,722	1,452,041
Areas	484	484	484	484
R-squared	0.049	0.023	0.024	0.063
Dependent var. mean	0.091	0.168	0.378	0.258
% change from 10pp increase in % college grads	*-12.4*	*-4.1*	*0.5*	*-7.5*

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS). All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

Appendix Table C7: Regression results of smoking on area human capital

	(1) CPS: Current smoker	(2) CPS: Former smoker	(3) CPS: Never smoker	(4) BRFSS: Current smoker	(5) BRFSS: Former smoker	(6) BRFSS: Never smoker
Area characteristics						
% college graduates	-0.00204***	0.00282***	0.00157**	-0.00189***	0.00327***	0.00067
	(0.00046)	(0.00078)	(0.00072)	(0.00030)	(0.00052)	(0.00045)
% college graduates *	-0.00007	0.00054	-0.00013	--	--	--
year=2000	(0.00025)	(0.00048)	(0.00033)	--	--	--
% college graduates *	-0.00043*	0.00052	0.00072	-0.00085***	0.00077***	0.00094***
year=2010	(0.00026)	(0.00051)	(0.00047)	(0.00018)	(0.00029)	(0.00024)
Individual characteristics (ref. group: no high school)						
High school graduate	-0.02868***	0.06477***	0.00261	-0.05599***	0.06441***	0.04979***
	(0.00461)	(0.00510)	(0.00518)	(0.00508)	(0.00520)	(0.00668)
Some college	-0.06794***	0.13531***	0.02188***	-0.10064***	0.12257***	0.08156***
	(0.00628)	(0.00543)	(0.00720)	(0.00573)	(0.00554)	(0.00757)
College graduate	-0.16889***	0.23574***	0.15037***	-0.23351***	0.24964***	0.24232***
	(0.01004)	(0.00678)	(0.01136)	(0.00801)	(0.00651)	(0.00951)
Post-graduate education	-0.22031***	0.30456***	0.20004***	--	--	--
	(0.01197)	(0.00944)	(0.01265)	--	--	--
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	613,858	238,830	613,858	1,551,524	738,024	1,551,524
Cells	613,858	238,830	613,858	1,551,524	738,024	1,551,524
Areas	297	297	297	484	484	484
R-squared	0.086	0.123	0.081	0.088	0.121	0.063
Dependent var. mean	0.165	0.561	0.624	0.192	0.585	0.538

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS) or the Tobacco Use Supplement in the Current Population Survey (CPS) from waves 1995-1996, 1998-1999, 2001-2002, 2003, 2006-2007, 2010-2011, and 2014-2015. All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

Appendix Table C8: Regression results of obesity on area human capital

	(1) BRFSS: Very obese	(2) BRFSS: Obese	(3) BRFSS: Overweight	(4) BRFSS: No physical activity
Area characteristics				
% college graduates	-0.00087***	-0.00060**	-0.00058**	-0.00165***
	(0.00026)	(0.00030)	(0.00029)	(0.00033)
% college graduates *	--	--	--	--
year=2000	--	--	--	--
% college graduates *	-0.00034*	-0.00012	0.00108***	-0.00038
year=2010	(0.00020)	(0.00020)	(0.00025)	(0.00024)
Individual characteristics (ref. group: no high school)				
High school graduate	-0.01825***	-0.01485***	0.01654***	-0.07400***
	(0.00172)	(0.00266)	(0.00357)	(0.00292)
Some college	-0.02380***	-0.01687***	0.01940***	-0.14805***
	(0.00170)	(0.00274)	(0.00347)	(0.00261)
College graduate	-0.06569***	-0.05732***	0.02171***	-0.24287***
	(0.00185)	(0.00320)	(0.00382)	(0.00378)
Individual chars	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes
Weighted obs.	1,490,722	1,490,722	1,490,722	1,452,041
Cells	1,490,722	1,490,722	1,490,722	1,452,041
Areas	484	484	484	484
R-squared	0.049	0.023	0.024	0.063
Dependent var. mean	0.091	0.168	0.378	0.258

*** p<0.01, ** p<0.05, * p<0.1. Standard errors are clustered at the area level.

Note. All probit regressions pool data from the 1999-2001 and 2009-2011 Behavioral Risk Factor Surveillance System (BRFSS). All regressions use sampling weights and include individual-level controls for 5-year age (25-29, 30-34, …, 85+, missing) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic, missing race/ethnicity) interactions, individual education, and state-year fixed effects. We exclude individuals with missing education. Area-level percent college graduates in each year was measured using data from the immediately preceding census or 3-year ACS. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares, defined similarly as percent college graduates.

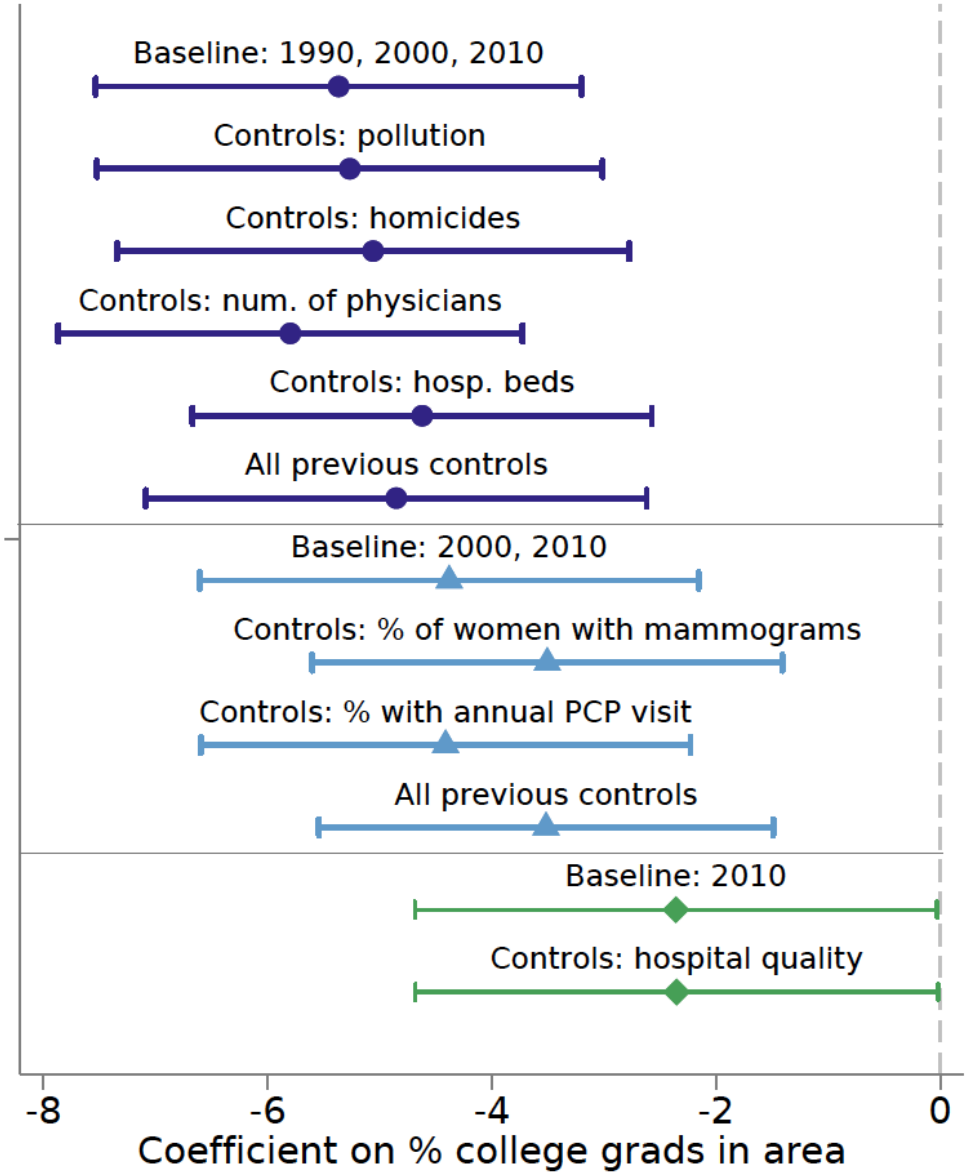
Appendix Table C9: Regression results of mortality per 100,000 by cause of death on area human capital and health-related behaviors

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Cancer		**Chronic lower respiratory disease**		**Stroke**		**Drug poisoning**	
Area characteristics								
% college graduates	-1.46***	-0.44	-0.69***	-0.10	-0.32**	-0.04	-0.15**	-0.04
	(0.28)	(0.29)	(0.15)	(0.14)	(0.13)	(0.16)	(0.06)	(0.07)
% current smoker		2.50***		0.88***		0.26		0.18
		(0.62)		(0.31)		(0.37)		(0.11)
% former smoker		32.23		-26.51		-17.30		-3.40
		(38.39)		(20.79)		(22.38)		(6.52)
% overweight,		0.73***		0.30*		0.25*		0.08
obese, very obese		(0.22)		(0.15)		(0.15)		(0.06)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Weighted obs.	357,070,962	357,070,962	357,070,962	357,070,962	357,070,962	357,070,962	357,070,962	357,070,962
Cells	257,906	257,906	257,906	257,906	257,906	257,906	257,906	257,906
Areas	485	485	485	485	485	485	485	485
R-squared	0.749	0.749	0.614	0.614	0.672	0.672	0.110	0.111
Dependent var. mean	288	288	66	66	75	75	12	12
% change from 10pp increase in % college grads	-5.1	*-1.5*	-10.5	-1.5	-4.3	-0.6	-12.3	-3.0

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. OLS regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 2000 and 2010. All regressions control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. The percentage of current or former smokers was calculated using the 1995-1996, 1998-1999 CPS, 1999-2001 BRFSS, and 2009-2011 BRFSS. The percent of individuals that were overweight, obese, or very obese, and those with no physical activity were calculated using the 1999-2001 and 2009-2011 BRFSS.

Appendix Figure C2: Regression results of all-cause mortality per 100,000 on area human capital and health-related amenities



Note. All regressions are estimated separately at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. The main control in the baseline regression is the percent of individuals currently smoking and the percent of individuals formerly smoking (conditional on ever smoking). All regressions further control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares. Confidence intervals are clustered at the area level.

Appendix Table C10: Regression results of all-cause mortality per 100,000 on area human capital and health-related amenities

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Area characteristics												
% college graduates	-3.48***	-3.41***	-3.41***	-3.86***	-2.61**	-3.05***	-2.03**	-3.08***	-2.05**	-0.65	-0.66	-0.66
	(1.13)	(1.15)	(1.16)	(1.05)	(1.02)	(1.08)	(1.02)	(1.06)	(0.97)	(1.18)	(1.18)	(1.18)
Air pollution (PM-2.5)		3.66				4.05	2.93	3.93	1.34	-1.92	-2.03	-2.03
		(4.27)				(3.96)	(4.08)	(4.01)	(4.05)	(5.27)	(5.33)	(5.33)
Homicide rate per			0.48			-0.55	-1.73	-0.52	-1.70	-1.61	-1.58	-1.58
100,000			(1.67)			(1.64)	(1.62)	(1.63)	(1.46)	(3.31)	(3.29)	(3.29)
Physicians per 1,000				13.12***		8.77**	9.18**	8.89**	10.71***	10.52**	10.55**	10.55**
				(4.12)		(4.10)	(4.06)	(4.03)	(3.94)	(4.13)	(4.14)	(4.14)
Hospital beds per 1,000					12.10***	9.66***	10.40***	9.68***	10.84***	10.49**	10.34**	10.34**
					(3.14)	(3.30)	(3.40)	(3.29)	(3.31)	(4.10)	(4.17)	(4.17)
% with mammogram							-5.81***		-7.39***	-8.27***	-8.36***	-8.36***
							(1.25)		(1.27)	(1.16)	(1.21)	(1.21)
% with annual PCP visit								0.32	3.52***	2.22**	2.21**	2.21**
								(1.01)	(0.99)	(1.06)	(1.06)	(1.06)
Hospital quality (z-score)											4.21	4.21
											(11.82)	(11.82)
% currently smoking	6.76**	7.06***	6.71**	5.63**	5.34**	5.26**	5.59**	5.27**	5.77**	6.78***	6.84***	6.84***
	(2.70)	(2.69)	(2.75)	(2.61)	(2.47)	(2.44)	(2.41)	(2.44)	(2.34)	(2.44)	(2.44)	(2.44)
% formerly smoking	-205.93	-187.47	-205.84	-247.03	-244.69	-244.05	-189.96	-241.08	-142.74	-273.32*	-272.27	-272.27
	(176.18)	(172.34)	(177.15)	(168.95)	(166.43)	(159.08)	(153.37)	(161.27)	(148.80)	(165.76)	(166.00)	(166.00)
% overweight, obese,	4.18***	4.01***	4.19***	4.67***	4.44***	4.52***	4.85***	4.51***	4.88***	7.13***	7.17***	7.17***
very obese	(1.09)	(1.13)	(1.09)	(1.08)	(1.09)	(1.10)	(1.08)	(1.11)	(1.07)	(1.47)	(1.47)	(1.47)
Individual chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
State-year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area chars	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area-level industry shares	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Cells	257,598	257,598	257,598	257,598	257,598	257,598	257,598	257,598	257,598	145,531	145,531	145,531
Areas	485	485	485	485	485	485	485	485	485	485	485	485
R-squared	0.879	0.879	0.879	0.879	0.879	0.879	0.879	0.879	0.879	0.874	0.874	0.874
Dependent var. mean	1,206	1,206	1,206	1,206	1,206	1,206	1,206	1,206	1,206	1,175	1,175	1,175
% change from 10pp increase in % college grads	-2.9	-2.8	-2.8	-3.2	-2.2	-2.5	-1.7	-2.6	-1.7	-0.5	-0.6	-0.6

* p<0.1, ** p<0.05, *** p<0.01. Standard errors are clustered at the area level.

Note. Former smoker is defined conditional on ever smoking. All regressions are estimated at the area-year-age-sex-race-education cell level, weighted by cell population, and pooled across 1990, 2000, and 2010. All regressions further control for cell-level 5-year age (25-29, 30-34, …, 85+) by sex by race (white non-Hispanic, Black non-Hispanic, other non-Hispanic, Hispanic) interactions, individual education, percent of death certificates without education information, and year. We also include controls for area log density and log population, percent Black, percent Hispanic, and industry shares.

Appendix D: Proofs of Propositions

Proof of Proposition 1:

The first order condition for unhealthy behavior is $B_x = \frac{b_0 + b_2 B_k - d_0 Q_k \mathrm{h}_x \beta V_x - p_k^B}{b_1 + b_2}$, where B_x for x=H, L refers to the optimal level of B for the two groups (high human capital and low human capital individuals). Given the exogenous share of high human capital individuals σ_k, the first order condition implies that:

$$B_k = \frac{1}{b_1}(b_0 - p_k^B - d_0 Q_k \beta(\sigma_k h_H V_H + (1-\sigma_k) h_L V_L)$$

$$B_H = \frac{b_0 - p_k^B}{b_1} - \frac{d_0 Q_k \beta}{b_1(b_1 + b_2)}\big((b_1 + b_2\sigma_k) h_H V_H + b_2(1-\sigma_k) h_L V_L\big)$$

$$B_L = \frac{b_0 - p_k^B}{b_1} - \frac{d_0 Q_k \beta}{b_1(b_1 + b_2)}\big(b_2\sigma_k h_H V_H + (b_1 + b_2(1-\sigma_k)) h_{\,L} V_L)\big)$$

These terms are decreasing with σ_k, p_k^B, h_L, h_H, Q_k, β, d_0, V_L and V_H, and increasing with b_0. The difference $B_L - B_H = \frac{d_0 Q_k \beta}{b_1 + b_2}(h_H V_H - h_L V_L) > 0$ and $B_k - B_H = \frac{(1-\sigma_k)}{b_1 + b_2} d_0 Q_k \beta (h_H V_H - h_L V_L) > 0$ as $V_H h_H > V_L h_L$, and $\frac{dB_k}{d\sigma_k} = -\frac{Q_k \beta d_0}{b_1}(\mathrm{h}_H V_H - \mathrm{h}_L V_L)$.

Proof of Proposition 2:

The only endogenous price is the price of non-traded services, which must clear the market, and this requires $g'(p_k^N)^{-1} = (1-\sigma_k) n_k$ so that the per capita production of non-traded services equals per capita consumption. This implies that $\frac{dp_k^N}{d\sigma_k} = -n_k g''\big((1-\sigma_k) n_k\big)$, which is positive.

The spatial equilibrium for the highly educated workers implies that:

$$\begin{aligned} W_k^H - p_k^N(1-\sigma_k) n_k + \; & g\big((1-\sigma_k) n_k\big) - \frac{(b_0 - p_k^B)^2}{2b_1} \\ & - \frac{(d_0 Q_k \beta)^2}{2b_1(b_1 + b_2)^2}\Big(\big((b_1 + b_2\sigma_k) h_H V_H + b_2(1-\sigma_k) h_L V_L\big)^2 \\ & - b_1 b_2 (1-\sigma_k)^2 (h_H V_H - h_L V_L)^2\Big) = U_H \end{aligned}$$

We have $\frac{d\sigma_k}{dW_k^H}$ equals 1 divided by $-n_k^2(1-\sigma_k)g''\big((1-\sigma_k)n_k\big)+\frac{b_2}{b_1(b_1+b_2)}(d_0Q_k\beta)^2(h_HV_H-h_LV_L)(\sigma_k h_HV_H+(1-\sigma_k)h_LV_L)$, which is positive. As W_k^H does not directly impact health, we know that $\frac{dB_x}{dW_k^H}=\frac{\partial B_x}{\partial\sigma_k}\frac{d\sigma_k}{dW_k^H}<0$, and $\frac{dB_k}{dW_k^H}=\frac{\partial B_k}{\partial\sigma_k}\frac{d\sigma_k}{dW_k^H}<0$.

The probability of survival is $Q_k h_x(1-d_0B_x)$ and denoted S_x for each type, and for the area overall equals $Q_k(\sigma_k h_H(1-d_0B_H)+(1-\sigma_k)h_L(1-d_0B_L))$. Consequently, the effect of W_k^H for the survival rate of each group is $\frac{dS_x}{dW_k^H}=-Q_k h_x d_0\frac{\partial B_x}{\partial\sigma_k}\frac{d\sigma_k}{dW_k^H}>0$ and overall is $\frac{dS_k}{dW_k^H}=Q_k\big(h_H(1-d_0B_H)+h_L(1-d_0B_L)\big)\frac{d\sigma_k}{dW_k^H}-Q_k d_0\left(h_H\sigma_k\frac{dB_H}{dW_k^H}+h_L(1-\sigma_k)\frac{dB_L}{dW_k^H}\right)$ with both terms positive.

We also have that $\frac{d\sigma_k}{dh_L}$ equals $\frac{b_2(1-\sigma_k)}{b_1(b_1+b_2)}(d_0Q_k\beta)^2V_L(\sigma_k h_HV_H+(1-\sigma_k)h_LV_L)$ divided by $\Big(-n_k^2(1-\sigma_k)g''\big((1-\sigma_k)n_k\big)+\frac{b_2}{b_1(b_1+b_2)}(d_0Q_k\beta)^2(h_HV_H-h_LV_L)(\sigma_k h_HV_H+(1-\sigma_k)h_LV_L)\Big)$, which is also positive.

We know that $\frac{dB_H}{dh_L}=\frac{\partial B_H}{\partial\sigma_k}\frac{d\sigma_k}{dh_L}-\frac{V_Lb_2}{(b_1+b_2)b_1}(1-\sigma_k)Q_k\beta d_0$, $\frac{dB_L}{dh_L}=\frac{\partial B_L}{\partial\sigma_k}\frac{d\sigma_k}{dh_L}-\frac{(b_1+(1-\sigma_k)b_2)}{(b_1+b_2)b_1}Q_k\beta d_0V_L$ and $\frac{dB_k}{dh_L}=\frac{\partial B_k}{\partial\sigma_k}\frac{d\sigma_k}{dh_L}-\frac{1}{b_1}Q_k\beta d_0V_L(1-\sigma_k)$. As $\frac{d\sigma_k}{dh_k}>0$, $\frac{\partial B_L}{\partial\sigma_k}<0$, $\frac{\partial B_H}{\partial\sigma_k}<0$, and $\frac{\partial B_k}{\partial\sigma_k}<0$, these terms are all negative.

For the survival rates we have $\frac{dS_H}{dh_L}=-d_0Q_kh_H\frac{\partial B_H}{\partial h_L}>0$, $\frac{dS_L}{dh_L}=Q_k(1-d_0B_L)-d_0Q_kh_L\frac{\partial B_L}{\partial h_L}>0$, and $\frac{dS_k}{dh_L}=Q_k\big(h_H(1-d_0B_H)-h_L(1-d_0B_L)\big)\frac{d\sigma_k}{dh_L}-\sigma_kQ_kh_Hd_0\frac{\partial B_H}{\partial h_L}-(1-\sigma_k)Q_kh_kd_0\frac{\partial B_L}{\partial h_k}+(1-\sigma_k)Q_k(1-d_0B_L)$ and all terms are positive.

Let $X_1=\frac{-(d_0Q_k\beta)^2}{2b_1(b_1+b_2)^2}$ (which is negative), $X_2=(\,(b_1+\sigma_k)h_HV_H+b_2(1-\sigma_k)h_LV_L)^2$ (which is positive), and $X_3=b_1b_2(1-\sigma_k)^2(\,h_HV_H-h_LV_L)^2$, which is positive.

We then have $\frac{d\sigma_k}{dQ_k}$ equal to $\frac{-(d_0\beta)^2Q_k}{b_1(b_1+b_2)^2}(X_2^2+X_3)$ divided by $n_k^2(1-\sigma_k)g''\big((1-\sigma_k)n_k\big)-2X_1(b_1+b_2)\big(\,b_2(1-\sigma_k)(h_LV_L)^2-b_2\sigma_k(h_HV_H)^2-b_2(1-\sigma_k)(h_LV_L)(h_HV_H)\big)$. $\frac{d\sigma_k}{dQ_k}>0$ if $\big(\,b_2(1-\sigma_k)(h_LV_L)^2-b_2\sigma_k(h_HV_H)^2-b_2(1-\sigma_k)(h_LV_L)(h_HV_H)\big)<0$, assuming $(1-\sigma_k)(h_LV_L)^2<(h_HV_H)^2(\sigma_k(h_HV_H)+(1-\sigma_k)(h_LV_L)$, which holds if differences between h_LV_L and h_HV_H are large enough.

We know that $\frac{dB_H}{dQ_k} = \frac{\partial B_H}{\partial \sigma_k}\frac{d\sigma_k}{dQ_k} - \frac{d_0\beta}{b_1(b_1+b_2)}\big((b_1 + b_2\sigma_k)h_H V_H + b_2(1-\sigma_k)h_L V_L\big)$, $\frac{dB_L}{dQ_k} = \frac{\partial B_L}{\partial \sigma_k}\frac{d\sigma_k}{dQ_k} - \frac{d_0\beta}{b_1(b_1+b_2)}\big(b_2\sigma_k h_H V_H + (b_1 + b_2(1-\sigma_k))h_{\ L}V_L\big)$ and $\frac{dB_k}{dQ_k} = \frac{\partial B_k}{\partial \sigma_k}\frac{d\sigma_k}{dQ_k} - \frac{d_0\beta}{b_1}(\sigma_k \mathrm{h}_H V_H + (1-\sigma_k)\mathrm{h}_L V_L)$. As $\frac{d\sigma_k}{dQ_k} > 0$, $\frac{\partial B_L}{\partial \sigma_k} < 0$, $\frac{\partial B_H}{\partial \sigma_k} < 0$, and $\frac{\partial B_k}{\partial \sigma_k} < 0$, these terms are all negative.

For the survival rates we have $\frac{dS_H}{dQ_k} = h_H(1 - d_0 B_H) - d_0 Q_k h_H \frac{\partial B_H}{\partial Q_k}$, $\frac{dS_L}{dQ_k} = h_L(1 - d_0 B_L) - d_0 Q_k h_L \frac{\partial B_L}{\partial Q_k}$, and $\frac{dS_k}{dQ_k} = Q_k\big(h_H(1 - d_0 B_H) - h_k(1 - d_0 B_L)\big)\frac{d\sigma_k}{dQ_k} - \sigma_k Q_k h_H d_0 \frac{\partial B_H}{\partial Q_k} - (1 - \sigma_k)Q_k h_k d_0 \frac{\partial B_L}{\partial Q_k} + (1-\sigma_k)h_k(1 - d_0 B_L) + \sigma_k h_H(1 - d_0 B_H)$ and all terms are positive.