Relative income, race, and mortality

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Abstract

This paper examines the relationship between relative income and mortality. Our research is motivated by recent literature that posits that, holding individual income fixed, those whose income are low relative to the incomes of those in a reference group will have worse health. We develop an empirical model in which an individual’s health is a function of his or her own income and the incomes of those who live in the same geographical area. We show how this individual-level model can be estimated using semi-aggregated data on the mortality rates of people categorized by age, race, gender, and place of residence. The model is estimated using mortality data from the 1980 and 1990 Compressed Mortality Files, merged with income data from the 1980 and 1990 5% Public Use samples of the US Census. We find no evidence that having relatively wealthy neighbors, holding own income fixed, is associated with higher mortality. Instead, we find evidence that among some demographic and age groups—in particular working-aged black males—having relatively wealthy neighbors is associated with lower mortality. For example, among younger (aged 25–64) black men, an increase in the income of others is estimated to have a beneficial effect on mortality that is 40% as large as an equivalent increase in own income.

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1. Introduction

The topic of this paper is the relationship between relative income and health. We examine whether people whose incomes are high relative to others who live in the same geographic area...
have lower or higher mortality. This analysis holds own income fixed, so the question is not whether own income matters for mortality, but whether the incomes of others affects mortality given own income. It is well-established that wealthier individuals are healthier and live longer (see Sorlie et al., 1995; Elo and Preston, 1996, 1997, and the review in Adler et al., 1994).

The proposition that relative income affects health is more controversial. Although there are reasons why there could be health benefits from having wealthier neighbors, a growing body of literature argues that low relative income is a health hazard. This proposition runs counter to the Pareto principle and, if correct, could have very unorthodox implications for economic policy.

We examine this topic through the lens of racial differences in mortality in the United States. Blacks in the United States have much higher mortality rates than whites. Figures from the National Center for Health Statistics (1998) indicate that in 1990 the life expectancy at birth for black males was 64.5 years, compared to a life expectancy at birth of 72.7 years for white males. Life expectancy for white females was 79.4 years, in contrast to 73.6 years for black females. Fig. 1 uses data from the National Center for Health Statistic’s Mortality Detail Files, and shows the log odds of mortality by years of age for black and white men and women, in 1980 and 1990. Black mortality is higher at all ages except in the late teen-age years and at the very oldest ages.\(^1\) Black mortality is higher than white mortality for almost all causes of death, accidents and suicides being notable exceptions. There is a large body of research that seeks to explain the US black-white mortality differential, and specifically how much of the differential can be accounted for by racial differences in socioeconomic status. Although some questions remain, much of this

\(^{1}\) This “mortality cross-over”, where the oldest blacks have lower mortality rates than whites, has been extensively researched, and is very likely an artefact of great age misreporting for blacks in both Census data and death records (Elo and Preston, 1997).
evidence concludes that racial differences in socioeconomic status account for a large portion of racial mortality differences.\footnote{Research surveyed by Smith and Kington (1997) and House and Williams (2000) indicates that racial differences in socioeconomic status, typically measured by income, education and sometimes occupation, account for between 60\% to 100\% of the black-white mortality difference in the United States. Racial differences in socioeconomic status appear to explain more of the black-white mortality difference at older ages than at younger ages (Sorlie et al., 1992). House and Williams (2000) discuss often subtle racial differences in access to health care and the quality of medical treatment as possible explanations. (See also Multcherr and Burr, 1991; Escarce and Puffer, 1997, for discussions of racial differences in health care.)}

The research in this paper exploits differences in mortality and income levels of blacks and whites within geographical areas to examine how relative income affects health. Our starting point is a model in which an individual’s health is a function of his or her own income and the incomes of those who live in the same geographical area. We show how this individual-level model can be estimated using semi-aggregated data on the mortality rates of people categorized by age, race, gender and place of residence. The analysis uses mortality data from the 1980 and 1990 Compressed Mortality Files (CMF), which have information on all-cause mortality and mortality by cause of death broken out by county, gender, racial group, and age group, merged with income data are from the 1980 and 1990 5\% Public Use samples of the Census.

Our identification strategy is illustrated by the following example. Consider two different areas, County A and County B, and assume that the income levels of members of a specific race and gender group—for example, black men—are very similar in these two counties. Suppose also that the per capita income level in County A is higher than in County B. The question we ask is whether the mortality of black men is higher in County A or B. A finding that black men in County A have higher mortality suggests that low relative income is associated with worse health. A finding that black men in County A have lower mortality suggests that there is a positive association between health and having wealthy neighbors.

Section 2 of the paper discusses the mechanisms through which relative income might influence health, and reviews existing literature on this topic. Section 3 presents an empirical model of mortality and relative income and describes the data and methods used to estimate the model. One of our key concerns in this section is to show how models of the effects of relative income on the mortality of individuals can be estimated using semi-aggregated data. Section 4 shows our main results. We find no evidence that having relatively wealthy neighbors increases the risk of death. For some demographic groups—notably younger black men—those with wealthier neighbors have lower mortality. For example, for younger (aged 25–64) black men, an increase in the income of others is estimated to be associated with a reduction in mortality that is 40\% as large as an equivalent increase in own income. Section 5 contains results from a variety of extensions to the basic model, such as including controls for regional prices levels, examining cross-race effects, using education rather than income as the measure of economic status, and looking at the associations between relative income and specific causes of death. A conclusion is in Section 6.

2. Relative income, health and mortality

2.1. Mechanisms

Why might relative income matter for mortality? One theory which has been emphasized in the public health and epidemiological literatures posits that low relative income harms health...
by increasing psychosocial stress (Wilkinson, 1996, 1997, 1998). Specifically, a person whose income—or, more broadly, social status—is low relative to others in his “reference group”, holding his own income or level of social status fixed, experiences greater psychosocial stress, which has adverse biological and behavioral consequences. This hypothesis draws a clear distinction between absolute resources—which are thought to account for only a small part of the association between health and socioeconomic status—and relative resources, which involve social comparisons between one’s self and with one’s peers. The hypothesis as stated by biologist Robert Sapolsky is that “poor health is not so much the outcome of being poor, but of feeling poor, that is, feeling poorer than others” (Sapolsky, 2004, p. 410).

Empirical evidence on this hypothesis is incomplete. There is a great deal of solid evidence that psychosocial stress adversely affects health. Results from research on humans and other animals indicates that stress can harm the cardiovascular system, weaken the immune system, and even hasten cellular aging (see, for example, Sapolsky, 2004; Cohen et al., 1997; Cohen et al., 1991; Epel et al., 2004). However, it is much less clear whether low relative income or social standing increases psychosocial stress in a manner that harms health. In a recent review article, Sapolsky argues that among non-human mammals, those with low “status” within dominance hierarchies do not necessarily experience higher stress (Sapolsky, 2004). Patterns differ across species, and depend on whether hierarchies are stable or unstable. For example, in unstable hierarchies in which dominance is frequently challenged, the most dominant individuals may be subject to the most stress, whereas in stable hierarchies the pattern may be reversed.

Less is known about the relationship between relative income and stress among humans. Experiments done with animals, in which social status is experimentally manipulated while diets (the animal analog of absolute income) are held fixed, obviously cannot be conducted with humans. Instead, observational data must be used to construct measures of both absolute income and relative income, and examine how these are associated with stress. One line of literature has examined how measures of objective socioeconomic status (as measured by occupational grade, education, or income) and self-reported subjective social status is associated with different markers of stress and with health outcomes (Adler et al., 2000; Singh-Manoux et al., 2005). This research generally concludes that subjective social status is a “better” predictor of psychological distress and health measures than objective measures of socioeconomic status. However, the objective and subjective measures are typically not measured in terms of money, nor even measured in the same units as each other, making it difficult to interpret the results. Furthermore, it is possible that a person’s stress level or health status could influence his or her report of subjective social status. It is preferable to use an objective measure of relative income.

While the psychosocial stress model implies that low relative income is a health hazard, several alternative economic models imply that low relative income may be either harmful or beneficial to health. One such alternative is a model of pecuniary externalities, in which the availability and prices of locally produced goods and services that influence health are affected by the income level of a region. Suppose, for example, that certain medical services in a region have upward-sloping supply curves. Increases in regional incomes would cause the prices of these services to rise. In this case, holding own income fixed and assuming that medical care affects health, individuals will be harmed by living in wealthier areas. However, these effects could just as easily work in the opposite direction. If health services have decreasing costs, increases in aggregate income could work to reduce prices or improve availability of health-related goods. Consider an example in which mortality is a decreasing function of a specific type of medical facility—for example, a neonatal intensive care unit—which plausibly has high fixed costs of operation. Provided aggregate demand for the facility’s services increases with income, richer areas should be more likely to
have a facility, and (barring price regulation) to charge less for the facility’s services. In this case, relatively poor people may benefit from having wealthier neighbors, since their presence increases the availability and reduces the price of a service that improves health.

An alternative theory, which is closely related to the theory of pecuniary externalities, is that the link between relative income and health operates through the provision of public goods. Wealthier areas may choose to spend more on public goods that promote health. For example, higher incomes might be correlated with better policing, which reduces homicides; a greater demand for environmental regulations, which reduces exposures to toxins; and more spending on public health campaigns to control infectious diseases. In this case, holding one’s own income fixed, living in a wealthier area would confer a health benefit. It is still possible, however, that spending on public goods that promote health could decline as area income rises. For example, wealthier people might vote against public health programs that primarily benefit the poor. If so, it could be that poorer individuals who live in wealthier areas are disadvantaged. As in the case of pecuniary externalities, having wealthier neighbors could in theory confer a health benefit or a disadvantage.

Although the mechanisms that work through pecuniary externalities or the provision of public goods are plausible, it is difficult to identify the specific goods or services through which these effects might operate. Some evidence indicates that access to medical services does not have large effects on health, especially in adulthood (see, for example, Adler et al., 1994; House and Williams, 1995). If this is correct, then even if having wealthier neighbors affects the quantity or quality of health care available, there would still be no effect on health. Other research indicates that, although there are in fact great geographic differences in aspects of health care that do influence mortality, these differences are not related to income levels of regions. For example, Jonathan Skinner and Douglas Staiger find that, although there is substantial cross-state variation in the use of low-cost and effective medical technologies to treat heart attacks, the adoption of these technologies is not related to state per capita income (Skinner and Staiger, 2005). It is possible that these mechanisms work through other goods, such as environmental quality or safety. For example, Ash and Fetter (2004) find a negative association between median household income and local pollution within cities. Other evidence indicates that census tracts and blocks with higher poverty rates have higher rates of tuberculosis, sexually transmitted infections, and non-fatal weapons-related injuries (Krieger et al., 2003). This evidence supports the idea that wealthier areas may be healthier places to live, so that having low relative income (holding own income fixed) would confer a health benefit.

The preceding discussion focuses on the relationship between relative income and health. There is, however, a closely related literatures that examines the hypothesis that greater income inequality is related to worse health outcomes. Several of the theories of how relative income may affect health, discussed above, also have implications for the relationship between inequality and health. For example, researchers working in the “psychosocial stress” tradition have posited that greater inequality leads to less trust and greater hostility, which harms the health of people at all income levels; other have argued that there is less social cohesion in areas with greater inequality, which could adversely effect the provision of public goods that promote health (Wilkinson, 1999; Kawachi and Kennedy, 1999). Furthermore, some formal specifications of relative income

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3 The pollution measure used is based on the Risk-Screening Environmental Indicators Model developed by the Environmental Protection Agency. This measure aggregates levels of different pollutants, giving more weight to those that are more toxic.
models imply aggregate level associations between mean health and inequality (Deaton, 2003, pp. 122–123).

Although the theoretical underpinnings of the two hypotheses—that relative income affects health, and that inequality affects health—are similar, the two hypotheses are actually quite different. The first is concerned with whether, holding own income fixed, an individual’s health is related to the level of income in the surrounding area. The second is concerned with whether, holding own income fixed, an individual’s health is related to the spread of income in the surrounding area. These hypotheses can have quite different empirical implications. Consider, for example, an increase in inequality that is accomplished by reducing the incomes of those in the bottom half of the income distribution, leaving unchanged the incomes of those who are wealthier. If higher relative income improves health, this change in distribution should improve the health of those who are wealthy (whose relative incomes have increased even though their absolute incomes are the same); the health of the poor will be harmed because both their absolute and relative income levels have declined. However, if higher inequality harms health, then the health of everyone, rich and poor, will be adversely effected.

There is a large empirical literature on the relationship between inequality and health, and has yielded mixed findings. However, recent reviews of the evidence have concluded that inequality is not a health hazard (Lynch et al., 2004; Deaton, 2003). The hypothesis that relative income affects health, which is the topic of this paper, has received less empirical attention.

2.2. Definitions and data issues

Two key issues must be addressed prior to testing the relative income model. The first concerns the definition of relative income. Any measure of relative income requires a “reference” income to which own income is compared. The different theories discussed above offer vague guidelines as to how this reference group should be defined. The model of pecuniary externalities suggests that the reference group should consist of individuals in the same market for health-related goods: possibly those in the local community around a hospital. The political economy story indicates that the reference group consists of individuals living in the politically defined unit—a city, county, or state—in which decisions about relevant public goods are made. The psychosocial stress model provides even less direction, since the reference group is defined by the individual. Reference groups could in theory be defined by any level of geography (e.g. a neighborhood, a state, or even an entire country), or could instead by defined by membership in a common group (e.g. a religious or ethnic group) that cuts across regions.

Much of the existing work on relative income models, as well as similar work that examines the relationship between inequality and health, defines reference groups in terms of geography—either states, cities or counties. We follow this practice here. In most of the results we present, reference income is defined at the county-group (“puma”) level. This may be more likely to correspond to the models based on pecuniary externalities and public goods provision. As a result, it may be more difficult to interpret our results in light of the psychosocial models. We have also estimated all of our models using the state as the level of aggregation, and find similar results.

A second issue concerns data. As is discussed in Wagstaff and van Doorslaer (2000), the best kind of data for testing the relative income model are individual-level data on health or mortality that includes geographical identifiers, so that measures of individual health and mortality can be related to both individual income and income in the individual’s local area. However, such data are rare. Wagstaff and van Doorslaer (2000) present an excellent review of existing
evidence on the relative income model (along with evidence on a variety of other models) and cite only a handful of studies based on individual-level data. Mellor and Milyo (2002) use the Current Population Survey’s information on self-reported health status and conclude that, controlling for own-income, aggregate state income has no effect on health. Soobader and LeClere (1999) also use self-reported health information from the National Health Interview Study, matched to Census tract income data, and reach a similar conclusion. Although self-reported health status is a useful outcome to study in its own right and is correlated with subsequent mortality, this research does not directly address the relationship between relative income and mortality.

Because mortality is a rare event, studying mortality requires much larger sample sizes. Further, large-scale data sets with information on mortality, income, and geographical location are rare. We are aware of two studies that use micro data to address this question. Eibner and Evans (2005) use restricted access National Health Interview Survey data to examine the correlation between relative deprivation and health. They find that mortality is positively associated with relative deprivation for men for some (but not all) measures of relative deprivation examined. In contrast to their study, we present results for a wide set of demographic groups, focus on more finely defined geographic reference groups, and use somewhat different concepts of relative income. However, unlike Eibner and Evans, we are unable to define reference groups based on race and education. In addition, our study offers an approach that can be applied to many different years and geographic units. Gerdtham and Johannesson (2004) analyze data on Swedish individuals. They find that individual income is protective against mortality. Further, conditional on individual-level controls, local area mean income and income inequality are not strongly correlated with mortality. They note that Sweden is a relatively egalitarian society with universal health care. Our paper addresses similar questions (focusing in particular on relative income) in the US context, using methods suitable to the data available.

In the absence of publicly available large-scale individual-level mortality data, how can the relative income model be tested? As Wagstaff and Doorslaer point out, aggregated data on mortality and income are of limited use. It is difficult to test whether relative income within counties or states is related to mortality if the mortality data are aggregated at the county or state level. In this paper, we deal with this problem by using semi-aggregated mortality data, that breaks out mortality rates by age, race and gender within counties. Specifically, we examine whether the mortality of subgroups of the population within counties is related to only subgroup income, or whether the incomes of others not in the subgroup matter. Racial identity and age are useful because they serve to distinguish between members and non-members of subgroups.

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4 Evidence from the Whitehall studies of British civil servants indicates that, controlling for the individual’s employment grade (which is closely related to salary), those living in more disadvantaged neighborhoods report worse general health and mental health (Stafford and Marmot, 2003). This evidence supports the hypothesis that low relative income confers a health advantage. However, the analyses in this study did not control for income earned by other family members, such as a spouse, or other sources of income. It is possible that (for example) civil servants who earn low salaries but live in good neighborhoods are able to do so because they have other income sources—and their health advantage could be driven by this, rather than by neighborhood characteristics.

5 Several large data sets contain information on both income and mortality. These include the National Longitudinal Mortality Study and the National Health Interview Survey (mortality follow-back). However, geographic identifiers are suppressed in the public-use versions of these data sets. Further, they are limited in their temporal dimension, forcing researchers to focus on short time frames.
3. An empirical model of relative income and mortality

3.1. The model

To fix ideas, we start with a simple linear empirical relationship between the health of individual \( i \) living in region \( r \) in year \( t \) (\( h_{irt} \)), his or her own (logarithm of) income (\( y_{irt} \)), and the income of those in the individual’s reference group (\( z_{irt} \)):

\[
h_{irt} = \alpha_{at} + \beta_1 y_{irt} + \beta_2 z_{irt} + \epsilon_{irt} \tag{1}
\]

The subscripts on the intercept (\( \alpha_{at} \)) indicate that the intercept varies with age and time. On average, we expect that health will deteriorate as individuals grow older, and will improve over time holding age fixed. Assume, for now, that \( z_{irt} \) is the average logarithm of income of those within \( i \)’s reference group. This assumption will be loosened in one of the extensions discussed below.

An additional restriction that could be imposed on (1), and which will be tested in what follows, is that \( \beta_2 = -\beta_1 \), which implies that only relative income matters for health. This specification is the starting point in Deaton and Paxson (2001), which examines the implications for estimates of \( \beta_1 \) if \( z_{irt} \) is assumed to be unknown.

Eq. (1) could in principle be estimated with individual-level data on health, income, and reference group income. However, with data on mortality rates for groups of individuals, Eq. (1) must be aggregated to the appropriate unit of analysis. We work with region-level mortality data (where \( r \) denotes the region, which could be a state, county, or city) for \( G \) groups within each region and year, where groups are defined by gender, age, and race. Eq. (1) can be aggregated to the group level by averaging across all individuals within each region, year and group:

\[
\overline{h}_{grt} = \alpha_{at} + \beta_1 \overline{y}_{grt} + \beta_2 \overline{z}_{grt} + \epsilon_{grt} \tag{2}
\]

Estimation of all parameters in (2) requires that the reference groups differ from the groups over which (1) is aggregated. If individuals’ reference groups were defined along the lines of geography, age, race, and gender, then \( y_{grt} \) and \( z_{grt} \) would be identical and only the sum of \( \beta_1 \) and \( \beta_2 \) could be estimated. In this case, if own income and reference group income have equal but opposite effects, it will appear as if income has no effect on health. If instead the reference group consists of all people who live in the region, then \( \overline{z}_{grt} \) will simply equal average regional income, and will be identical for the \( G \) subgroups within each region. In what follows, we assume that reference group income is equal to average regional income, so that \( \overline{z}_{grt} \) is replaced by \( \overline{y}_{rt} \):

\[
\overline{h}_{grt} = \alpha_{at} + \beta_1 \overline{y}_{grt} + \beta_2 \overline{y}_{rt} + \epsilon_{grt}. \tag{3}
\]

A key assumption in going from the conceptual Eq. (1) to the estimating Eq. (3) is that the covariates enter linearly. Although it is plausible that there is some underlying index of “health” that is linear in income, the linear specification is not appropriate for a model of mortality. A better assumption is that the logarithm of the probability of death is linear in the observables.\(^6\)

Specifically, we assume that:

\[
p_{irt} = \exp(\alpha_{at} + \beta_1 y_{irt} + \beta_2 \overline{y}_{rt}), \tag{4}
\]

\(^6\) We also estimated models in which the log odds of mortality was assumed to be linear in the observables, with little effect on the results.
where \( p_{i\text{rt}} \) is the probability of death for individual \( i \), and we have incorporated the assumption that reference group income is equal to average regional income. Eq. (4) implies that, provided \( \beta_1 \) is negative, individual health (measured as \( 1 - p_{i\text{rt}} \), the survival probability) is concave in own income. The concavity of health in income is a common assumption and may produce a positive correlation between health and income inequality in aggregate data, even if no such relationship exists at the individual level. Miller (2000) uses an estimation methodology similar to the one here to examine the aggregate correlation between income inequality and health.

Without individual-level mortality data, (4) must be aggregated over individuals prior to estimation. We integrate over all individuals in the group to obtain:

\[
\bar{p}_{\text{grt}} = \int_{y \in g,r,t} \exp(\alpha_{at} + \beta_1 y + \beta_2 \bar{y}_{\text{rt}}) f_{\text{grt}}(y) dy + \epsilon_{\text{grt}} \tag{5}
\]

where \( f_{\text{grt}}(y) \) is the density function for income of people in group \( g \) in county \( r \) and year \( t \), and \( \epsilon_{\text{grt}} \) is a group/county/year error term.\(^7\) Although (5) is slightly more complicated than its linear analog (3), its parameters can be estimated given estimates of \( f_{\text{grt}}(y) \).

### 3.2. Data and implementation

We use two sources of data to estimate (5). The first is data from the 1980 and 1990 Compressed Mortality Files (CMF). The CMF is a census of all deaths in the US, and is compiled by the National Center for Health Statistics at the Center for Disease Control. The CMF contains information on the number of deaths by cause, within each county, gender group, racial group, and age group. The age of the deceased is recorded in 13 groups. We use information on deaths of people between the ages of 25 and 84, grouped into six 10-year age bands. We do not use data on deaths after age 85, because we are concerned about the well-documented racial differences in age misreporting at the oldest ages. We also restrict our analysis to blacks and whites, because it is difficult to obtain good estimates of \( f_{\text{grt}}(y) \) for smaller racial and ethnic groups. Information on mortality in 1980 and 1990 is matched to information from the 1980 and 1990 5% samples of the US Census. These are the largest samples available for public use.

Before the samples could be matched, it was necessary to define geographic regions that are common across the two data sources and the two years. The geographic identifiers provided in the CMF and Census data, although similar, are not identical. The CMF data are organized by county. The Census data contains identifiers for the “county group” (in 1980) and “puma” (in 1990). Although there are slight differences between county groups and pumas, they are similar in most ways. Both consist of counties or contiguous groups of counties within states, and are constructed to have populations of at least 100,000.\(^8\) In some cases, densely populated counties are broken into more than one puma. Because of these features of the data, it was necessary to aggregate the counties in the CMF up to the puma or country group level, and (in some cases) to aggregate pumas in the Census up to the county level. Because new counties were formed and some existing counties were redefined between 1980 and 1990, further aggregation was required to obtain geographical identifiers that were consistent between 1980 and 1990. After aggregation

\(^7\) We model the error term as a puma/age/year random effect. One alternate strategy is to model the aggregate error term as the sum of a collection of i.i.d. individual errors. This approach lends itself to a minimum chi-squared estimation, and is discussed in general in Powell and Stoker (1985).

\(^8\) Detailed information on the specific counties contained in county groups and pumas can be found at the IPUMS web site at the University of Minnesota.
and matching, there are 656 commonly defined geographic areas, which we continue to call “pumas” for ease of exposition.\footnote{9} The Census data are used for two purposes. The first is to construct puma-level income information that is included in the models. The variable $y_{irt}$ is measured as the average of $y_{irt}$, the logarithm of household income per adult equivalent, in each puma and year. Adult equivalents are defined to be the number of household members aged 18 or more, plus half the number of members aged 17 or less. We also constructed averages of $y_{irt}$ by racial group within pumas, and measures of the fraction of the population in each puma and year that is white.

Second, the Census data are used to construct estimates of the density of income, $f_{g r t}(y)$, for each of the $G$ groups within each puma and year. We did this simply by estimating a 200-bin histogram for the logarithm of income per adult equivalent for each group in each puma and year. Income is referred to by the midpoint of each of the 200 bands, $y^m$, where $m = 1, 2, \ldots, 200$. The probability that income for an individual in group $g$, puma $r$ and year $t$ falls into bin $m$ is denoted as $\lambda_{g r t}^m$. Eq. (5) is then replaced with:

$$ p_{g r t} - \sum_{m=1}^{200} \lambda_{g r t}^m \exp(\alpha_{at} + \beta_1 y^m + \beta_2 y_{irt}) = \epsilon_{g r t}, $$

where $p_{g r t}$ is equal to the mortality rate reported in the CMF for the relevant group. The parameters of (6) are chosen to minimize the sum of squared errors. In computing inference for the parameters in (6), we allow for unrestricted clustering at the puma/year level. Because the relationship between health and income may differ across whites and blacks and men and women, we estimate (6) separately for each of these groups. There is also evidence that the relationship between income and health becomes less pronounced at older ages (Backlund et al., 1996) and so we estimate separate models for younger (aged 25–64) and older (aged 65–84) age groups. This means that, for any one model we estimate, the data consist of mortality and income observations for either “older” or “younger” people of a given race, gender. Each observation is for a single puma/year/age group (where age group is defined by 10-year age bands) cell. There are four 10-year age groups represented in the younger samples (25–34, 35–44, 45–54, and 55–64) and two for the older samples (65–74 and 75–84), so the intercept term in (6) consists of eight age group/year dummies for the younger group, and four dummies for the older group.

For some groups, most notably black men, there are pumas in which very few people (or even no people) in the group died in a given year. These are typically sparsely populated pumas for which there are few Census observations. To cite an extreme example, the 1990 CMF indicates that there were 39 black men aged 55–64 living in all of Vermont, none of whom died. (Only one black male in this age group from Vermont is represented in the 5% Census sample.) Measures of mortality rates and estimates of the income densities in cases such as these are likely to be very noisy. We deal this “small cell” problem in two ways. First, we weight all errors by the square root of the population of the relevant racial group in the puma. This means that, for example, when we estimate models for blacks, those pumas with small numbers of blacks relative to other pumas receive less weight. We also exclude observations if the population in the cell is less than 500.\footnote{10} This results in the elimination of a large number of observations for blacks. For example,

\footnotetext{9}{These cover the entire population of the US. The median puma has an overall population of 186,000. The smallest puma has a population of approximately 87,000, and the largest has 8.5 million residents.}
\footnotetext{10}{We experimented with excluding groups with populations less than 1500, and obtained similar point estimates to those shown here. For most black groups, the restrictions reduce the number of observations significantly, resulting in larger standard errors and occasional loss of statistical significance, especially in the puma fixed effects models.}
the sample sizes for each group shown in Table 1 indicate that there are 2636 observations for younger black men, as compared to 5264 for younger white men, and 717 observations for older black men compared to 2625 observations for older white men.\footnote{This selection results in pumas that cover 95\% of younger black men, 82\% of older black men, 97\% of younger black women, and 89\% of older black women.}

To make sure that differences in the results between blacks and whites are not driven by differences in the type of pumas being selected, we have also estimated a set of models for whites that use the same samples of pumas used for blacks.\footnote{For the younger group, the results do not depend on which sample is used. For the 65–84 year old group, mean income is more strongly protective with respect to mortality for white men in the levels model, and less precisely estimated (but with a similar point estimate) for white women in the fixed effects model.} In most of our results, we also include a control for the fraction of the population in the puma that is white. Research by Deaton and Lubotsky (2003) indicates that racial composition is correlated with health outcomes for both blacks and whites, and we do not want to confound the effects of racial composition with the effects of relative income. For similar reasons, we include controls for the fraction of the population aged 18–64 and 65 and older.

In some specifications, we include puma-level fixed effects. In these models, the estimated effects of own and puma-level income on mortality are identified only by changes within pumas over time. The use of puma-level fixed effects has both advantages and disadvantages. The advantage is that their inclusion ensures that results are not driven by non-time-varying puma specific characteristics that may be correlated with the income terms. The disadvantage is that changes in incomes over the space of a decade may produce little effect on health and mortality. Instead, it may be that health is affected by own and relative income received over a longer time horizon. If so, including puma-level fixed effects may sweep out the effects we hope to estimate. Of course, if economic status over a longer time period is what matters for health, then current income is not an appropriate measure of economic status to use, regardless of whether fixed effects are included. To examine whether the results are sensitive to the choice of measure of economic status, we estimate models that use years of schooling rather than income. Years of schooling may be a better measure of long-run economic status than current income.

Fig. 2 illustrates how our data are organized, and highlights the source of identification for our models. The Figure shows income data for one sub-group of people—black men aged 55–64—who live in pumas in which their population is at least 500. Each point represents a pair of averages of the logarithm of household income per adult equivalent for a single puma. The y-axis shows averages over all people in the puma, and the x-axis shows averages for black men aged 55–64, and the 45° line is marked. The figure indicates that although the incomes of black men in this age group are positively correlated with average incomes in their pumas, men in this demographic group have, on average, lower incomes than others. There is also a great deal of variation in the average income of black men in this group relative to the puma means, and it is this variation that allows us to identify the correlations between relative income and mortality.

4. Basic results

We start by examining whether there is a relationship between mortality and own-group income. Table 1 presents variants of (6) that do not include $\overline{y}_{rt}$, puma-level average income, for each of the eight subgroups used for the analysis. Column (1) includes only own-group income, column (2) adds puma level control variables, and column (3) includes a set of puma fixed effects. The
Table 1
Effects of own income on the probability of mortality

<table>
<thead>
<tr>
<th>Puma fixed effects?</th>
<th>No</th>
<th>Yes</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(1)</td>
</tr>
<tr>
<td>ln((y))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Own age/race/sex group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black men, aged 25–64 (obs = 2636)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ln((y))</td>
<td>−0.222 (0.038)</td>
<td>−0.339 (0.024)</td>
<td>−0.260 (0.028)</td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.413 (0.049)</td>
<td>−0.311 (0.081)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White men, aged 25–64 (obs = 5264)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ln((y))</td>
<td>−0.246 (0.024)</td>
<td>−0.384 (0.014)</td>
<td>−0.223 (0.054)</td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.562 (0.036)</td>
<td>−0.317 (0.072)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black women, aged 25–64 (obs = 2759)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ln((y))</td>
<td>−0.166 (0.034)</td>
<td>−0.261 (0.029)</td>
<td>−0.201 (0.034)</td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.164 (0.039)</td>
<td>−0.067 (0.115)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White women, aged 25–64 (obs = 5264)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>ln((y))</td>
<td>−0.122 (0.024)</td>
<td>−0.260 (0.022)</td>
<td>−0.177 (0.040)</td>
<td>0.032 (0.032)</td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.412 (0.031)</td>
<td>−0.276 (0.049)</td>
<td></td>
<td>−0.074 (0.043)</td>
</tr>
</tbody>
</table>

Puma-level data. Maximum likelihood estimates. Standard errors in parenthesis. Note: All regressions include age group dummies that differ across years, and region dummies that differ by years. Regressions are weighted by the group’s population in the puma. Standard errors are computed allowing for unrestricted clustering at the Puma/year level. Columns 2 and 3 include controls for the fraction of the population aged 18–64 and 65 and over.
puma level control variables included in column (2) include the fraction of the population in the puma that is white, as well as the fraction of the population aged 18–64 and the fraction aged 65 and over. Each of these models also includes a set of age-group/year dummies.

The results in column (1) indicate that, for younger groups, high own-income is related to lower mortality. For older groups, the association between income and mortality is less strong. The association between income and mortality is larger for males than females. These patterns are consistent with other evidence that income gradients in mortality decline with age and are larger for males (Deaton and Paxson, 2001). The coefficients on own income for the younger group range from −0.122 for white females to −0.246 for white males. These coefficients are large. For example, the estimate for black males of −0.222 implies that a doubling of income is associated with a reduction in the probability of death of 22%. These gradients shrink for older groups; own income is protective only for older white men.

The second column in each panel includes a control for the fraction of the puma population that is white as well as the age-based demographic controls. Consistent with results in Deaton and Lubotsky (2003), the fraction white has large protective associations for both racial groups, especially for males and younger adults. The coefficient of −0.562 for younger white men

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13 We do not report coefficients on the age-based demographic control variables. They are generally highly significant. For most models, they indicate that for prime-aged individuals, living in a puma with fewer prime-aged adults and fewer elderly adults is associated with lower mortality. For elderly age groups, living in a puma with fewer prime-aged adults is associated with lower mortality. We have also estimated models with controls for the fraction aged 6–17, 18–29, 30–64, and 65 and over, with results very similar to those we report.

14 Although these coefficients are not shown, they indicate that (holding income fixed) mortality declined between 1980 and 1990 for nearly all demographic groups. Exceptions include black and white men aged 25–34 and 35–44, as well as black women 25–34. Mortality increased between 1980 and 1990 for these groups. HIV/AIDS is the likely explanation.

15 Deaton and Lubotsky (2003) examine how local racial composition is related to mortality, and obtain similar results to those shown in Table 1 using city-level data. In addition, they find that the inclusion of the racial composition measure accounts for the positive relationship between income inequality and mortality that is observed at both the state and city level. The source of the association between mortality and racial composition is a puzzle that they (and we) do not resolve.
implies that an increase in the fraction white from 82% to 97% (which is a move from the 25th to the 75th percentile in fraction white across pumas) is associated with a reduction in the probability of death by 8.4%. The inclusion of puma fixed effects in column 3 does not generally affect the protective effects of both own-income and fraction white, so these results are not the product of unobserved non-time-varying state characteristics. Tests for the joint significance of the puma fixed effects rejects the null of the effects being jointly zero. This is true for all results shown in this paper.

The central results of our analysis are in Table 2. The results shown in the first and third columns in each panel are for models that include own income, average puma income, the age/year interactions, as well as the age-demographic controls and the fraction white. As before, both “fraction white” and own income are typically associated with lower mortality. The coefficient on mean puma income provides evidence that having relatively wealthy neighbors is not harmful to health, and in some cases is beneficial. For younger white and black men and older black men the coefficient on mean puma income is negative and significant at the 5% level, indicating that higher levels of mean puma income are associated with lower mortality. For younger black women the coefficient is negative and significant at the 10% level. For the other groups the coefficient is not significantly different from zero. The magnitude of the coefficient for younger white men indicates that doubling the mean income in a puma (holding own income fixed) is associated with a 13.2% decrease in the mortality rate. Given that own income and puma income typically have the same sign, these results provides no evidence for the hypothesis that only relative income matters for health.16

The results in columns 2 and 4 of each panel include puma fixed effects. The introduction of fixed effects generally does not have a large or consistent effect on the parameter estimates. In some cases—especially for younger black men and women—including puma fixed effects increases the absolute value of the coefficient on puma income and reduces the absolute value of the coefficient on own income. However, for these groups, the results are qualitatively the same with and without fixed effects. For other groups, such as older men and women of both races, including fixed effects has little effects on the results. We take these results as evidence that non-time-varying puma-level unobservables were not driving the results in columns 1 and 3 for most groups.

The inclusion of fixed effects produces a large change in the results for younger white men. Specifically, the coefficient on mean puma income, which was negative and significant in the absence of fixed effects, becomes positive and marginally significant when fixed effects are included. The parameter value of 0.106 indicates that increases in puma income, holding own-group income fixed, increases mortality of those in this group. One possible reason for this anomalous result is that HIV related deaths increased from nearly 0 to a rate of about 14 per
Table 2
Effects of own education and aggregate income on the probability of mortality

<table>
<thead>
<tr>
<th>Puma fixed effects?</th>
<th>Men aged 25–64</th>
<th>Men aged 65–84</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No (1)</td>
<td>Yes (2)</td>
</tr>
<tr>
<td></td>
<td>Black (obs = 2636)</td>
<td>White (obs = 5264)</td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.346 (0.064)</td>
<td>−0.114 (0.122)</td>
</tr>
<tr>
<td>ln(y), Own age/race/sex group</td>
<td>−0.316 (0.026)</td>
<td>−0.235 (0.036)</td>
</tr>
<tr>
<td>Mean ln(y), Puma</td>
<td>−0.125 (0.064)</td>
<td>−0.230 (0.104)</td>
</tr>
<tr>
<td></td>
<td>No (1)</td>
<td>Yes (2)</td>
</tr>
<tr>
<td></td>
<td>Black (obs = 2759)</td>
<td>White (obs = 5264)</td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.104 (0.054)</td>
<td>0.371 (0.142)</td>
</tr>
<tr>
<td>ln(y), Own age/race/sex group</td>
<td>−0.234 (0.032)</td>
<td>−0.134 (0.041)</td>
</tr>
<tr>
<td>Mean ln(y), Puma</td>
<td>−0.106 (0.063)</td>
<td>−0.516 (0.105)</td>
</tr>
</tbody>
</table>

Puma-level data. Maximum likelihood estimates. Note: Standard errors in parenthesis. See note to Table 1.
100,000 for white men between 1980 and the time frame around 1990. In the early years of the AIDS epidemic, mortality was concentrated among younger white men. HIV related mortality was also geographically concentrated in New York and California (Pickle et al., 1996, pp. 144–151). We calculate that these areas also happened to experience large increases in income over this time period. We re-estimated the models shown in Table 2, excluding all pumas in New York and California. Doing so had little effects on the results for all demographic groups except for younger white men. With New York and California excluded, the coefficient on mean puma income in the fixed effects model for younger white men was \(-0.077\), with a standard error of 0.055.

To summarize the findings from Table 2, there is evidence for some demographic groups that having a lower relative income is associated with lower mortality. We find no consistent evidence for the idea that living in wealthy areas, controlling for own income, increases the risk of death.

5. Extensions

5.1. Spatial price deflators

The results discussed above could be biased by unobserved heterogeneity in price levels across pumas. Suppose, for example, that mortality is negatively related to own real income but uncorrelated with puma-level income, so that \(\beta_1\) is negative and \(\beta_2\) is zero. Suppose also that there is unobserved heterogeneity in the price levels across pumas that is not accounted for by our deflation using the national CPI. In this case, mean puma income may serve as a proxy for a puma-price deflator. The coefficient on puma-level income would then be biased in a positive direction, making it appear as if having wealthier neighbors is harmful to health. Although we find little evidence of this in the results discussed above, it is worth checking whether the use of regional price deflators alters our results.

To do this, we use state and city price level data provided to us by Victor Fuchs. These price index data are generated using chamber of commerce cost of living indices for 100 MSAs, and then regressed on local wage levels and property values. Predicted values from this regression are then given for each MSA with population over 100,000, all smaller MSAs within a state, and all rural areas within a state. As our data are not grouped at the MSA level, we are unable to get a one-to-one match between the price level data and our data. We take the (unweighted) average deflator for all urban areas in each state, as well as the rural deflator, and apply this to our data as appropriate.

When we re-estimate our main results using the regional price level deflators, we find similar results in sign and magnitude for nearly all models. The results with respect to own and relative income are relatively insensitive to the use of regional price deflators. We interpret these results as indicating that our previous results were not driven by the use of a national price index.17

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17 We have also explored the sensitivity of our results to including urban/rural controls. For each puma, we have computed the fraction of its residents living in urban or rural settings. When we include these variables as additional puma level controls in the “levels” specification (corresponding to columns 1 and 3 of Table 2), the results do not change greatly. The one exception is that the coefficient on puma income for the sample of older black women, which was negative but insignificantly different from zero in the previous specification, becomes significant. When we include these controls in the fixed effects specification, the coefficient on puma income for younger black men, which was previously negative and significant, becomes insignificant.
5.2. Within- and cross-race-group identification

So far we have focused on income relative to mean income for everyone in the puma. It is plausible that the incomes of those who are members and non-members of the individual’s racial group have different correlations with health. For example, if the “political economy” mechanism discussed above is valid and if people are more altruistic towards members of their own racial group, then having relatively wealthy neighbors of the same race could be beneficial, whereas having relatively wealthy neighbors of a different race could be harmful. It is also possible that, in a psychosocial stress model, reference groups are defined along racial lines. We have estimated models (results not shown) that control for both the mean income of puma residents in the same racial group and the mean income of puma residents from the other racial group.

Although the results are somewhat mixed across the different demographic groups, several patterns emerge. First, for all the black population groups, the coefficient on own income falls in magnitude and significance, and the coefficient on own-group income is strongly associated with lower mortality. This may indicate the difficulty of separately identifying these two effects for African Americans in this sample. Average own age/race/sex income and overall racial group income are likely to be highly correlated. Further, if the former is measured with some error (despite our cell size restrictions), then the own-race income coefficient may be actually picking up own age/race/sex group effects. As such, for this group focusing on the effects of “other race” puma income may be a cleaner test of the relative income hypothesis. Here, we find no significant evidence in support of the hypothesis that relative income affects mortality. For nearly all groups, the coefficient on “mean income, other racial groups” is small in magnitude and not statistically significant. The exception to this is for older white women, for whom other’s income is positively associated with mortality.

5.3. Within-age-group identification

An individual may have low relative income compared to those who live nearby, but at the same time may have life-cycle opportunities for upward mobility. If the psychosocial model of relative income is the applicable one, then the comparison against overall mean income may not be appropriate. Instead, comparing against peer-aged mean income may make more sense. We have re-estimated our models in Table 2, replacing “puma income” with “same age group puma income”. In this way we can test to see if (for example) 25–34 year old white mortality is related to average income of all 25–34 year olds. Results (not shown) generally indicate no effects of peer-aged mean income. There is no consistent pattern that higher peer-aged incomes are associated with greater mortality. These results do not provide support for the psychosocial model of relative income.

5.4. Education versus income

The theory that low social status has adverse effects on health does not necessarily imply that relative income determines social status. Other alternatives are possible: social status could be driven by education, occupation, “standing” within a community, or a number of other factors that may be correlated with income. In addition, even if income is the correct measure of social status, it is not clear whether permanent or current income is most relevant. Permanent income may be a more important determinant of social status than current income—so that, for example,
young people with steeper age earnings profiles may have higher “status” than those with identical current income who will experience low future earnings growth. An alternative is to use relative education rather than relative income as our measure of social status. Education serves as a proxy for lifetime resources, and also has the added advantage that it may be more precisely measured than income. Further, education does not suffer from the spatial price differences discussed in the previous subsection.

We re-estimate our basic models (corresponding to columns 1 to 4 of Table 2) using years of schooling in place of income. Results are presented in Table 3. For all of the younger groups, and for older white men, higher own-group education is associated with lower mortality: an additional year of schooling reduces mortality by 20–33% for younger men and women and by 14% for older white men. For younger men from both racial groups, younger white women, older white men and women, we find evidence that those who live in more highly educated pumas, holding own-group education fixed, have lower mortality. For the other groups the coefficient is not significantly different than zero. When puma fixed effects are included, the standard errors for the coefficients on puma-level education increase sharply, and puma-level education typically no longer has a significantly protective effect on mortality. Given that average years of schooling within a puma change only slowly over time, it is not surprising that these coefficients are imprecisely estimated when fixed effects are included. Overall, the results are consistent with those found earlier: own resources (education/income) are protective, and there is some weak evidence that living near more well-off neighbors is associated with lower mortality.

5.5. Alternate measures of reference income

Our primary specification uses puma level mean log income as a proxy reference group income \((z_{irf})\) in Eq. (1)). This choice of average income is arbitrary. It is possible that the effects of relative income on health work not through mean income but through some other summary measure of the income level on the puma. We have explored using two alternate measures of puma level income: the 25th and 75th percentiles. Results from this exercise are presented in Table 4. Several patterns emerge. First, in general the coefficients on fraction white and own income are insensitive to the choice of reference group income. Second, our main conclusions about relative income are also invariant to the choice of proxy. For most groups, living in a puma with wealthier neighbors is not generally associated with worse health—and in some cases is associated with better health. This is especially true for men (excluding younger black men), and when considering the 75th percentile of puma income. The sole exception to this pattern is that the 25th percentile of income is positively associated with mortality for younger white women. For most groups, the imprecision of the estimates implies that the coefficients on mean income (from Table 2), 25th percentile, and 75th percentile could very well be equal to one another. To summarize, exploring alternate measures of reference group income does not substantively affect our conclusions about relative income.

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18 Lleras-Muney (2005) finds that a year of schooling reduces 10-year mortality by 3.6 percentage points from a base of 10.6 percentage points, for a population with a mean age of 50. As such, we view our own-education results as broadly consistent with these estimates.

19 The one exception is for younger white women, for whom puma education is estimated to be positively and significantly associated with mortality when fixed effects are included. We do not have an explanation for this finding.
Table 3
Effects of own education and aggregate education on the probability of mortality

<table>
<thead>
<tr>
<th>Puma fixed effects?</th>
<th>Men aged 25–64</th>
<th>Men aged 65–84</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black (obs = 2636)</td>
<td>White (obs = 5264)</td>
</tr>
<tr>
<td></td>
<td>−0.382 (0.048)</td>
<td>−0.236 (0.104)</td>
</tr>
<tr>
<td></td>
<td>−0.330 (0.025)</td>
<td>−0.257 (0.028)</td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−0.043 (0.014)</td>
<td>−0.065 (0.043)</td>
</tr>
<tr>
<td>Schooling, own age/race/sex group</td>
<td>−0.146 (0.041)</td>
<td>−0.055 (0.122)</td>
</tr>
<tr>
<td>Mean schooling, puma</td>
<td>−0.253 (0.030)</td>
<td>−0.201 (0.034)</td>
</tr>
<tr>
<td></td>
<td>−0.022 (0.015)</td>
<td>−0.010 (0.033)</td>
</tr>
</tbody>
</table>

Puma-level data. Maximum likelihood estimates. Note: Standard errors in parenthesis. See note to Table 1.
Table 4
Alternate measures of puma-level income

<table>
<thead>
<tr>
<th></th>
<th>(1)</th>
<th>(2)</th>
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<tr>
<td></td>
<td>Men aged 25–64</td>
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<td>Men aged 65–84</td>
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<tr>
<td></td>
<td>Black (obs = 2636)</td>
<td>White (obs = 5264)</td>
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<td>Black (obs = 717)</td>
<td>White (obs = 2625)</td>
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<tr>
<td>Fraction white in puma</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ln(y), Own age/race/sex group</td>
<td>−0.341 (0.072)</td>
<td>−0.411 (0.051)</td>
<td>−0.515 (0.043)</td>
<td>−0.552 (0.037)</td>
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<td>−0.073 (0.063)</td>
<td>−0.066 (0.044)</td>
<td>−0.234 (0.034)</td>
<td>−0.192 (0.028)</td>
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<tr>
<td>25th percentile ln(y), puma</td>
<td></td>
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<td>−0.315 (0.025)</td>
<td>−0.338 (0.025)</td>
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<td></td>
<td>−0.073 (0.046)</td>
<td>−0.068 (0.052)</td>
<td>−0.191 (0.022)</td>
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<tr>
<td>75th percentile ln(y), puma</td>
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<td>−0.089 (0.050)</td>
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<td>−0.012 (0.071)</td>
<td></td>
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<td>0.019 (0.023)</td>
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<tr>
<td>Women aged 25–64</td>
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<tr>
<td></td>
<td>Black (obs = 2759)</td>
<td>White (obs = 5264)</td>
<td></td>
<td></td>
<td></td>
<td>Black (obs = 929)</td>
<td>White (obs = 2631)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>ln(y), Own age/race/sex group</td>
<td>−0.139 (0.061)</td>
<td>−0.153 (0.041)</td>
<td>−0.458 (0.042)</td>
<td>−0.415 (0.031)</td>
<td></td>
<td>−0.063 (0.057)</td>
<td>−0.055 (0.046)</td>
<td>−0.202 (0.030)</td>
<td>−0.176 (0.026)</td>
</tr>
<tr>
<td>25th percentile ln(y), puma</td>
<td></td>
<td>−0.250 (0.030)</td>
<td>−0.254 (0.030)</td>
<td>−0.314 (0.025)</td>
<td></td>
<td>0.000 (0.050)</td>
<td>0.011 (0.042)</td>
<td>−0.101 (0.026)</td>
<td>−0.058 (0.028)</td>
</tr>
<tr>
<td>75th percentile ln(y), puma</td>
<td></td>
<td>−0.030 (0.049)</td>
<td>0.073 (0.029)</td>
<td>−0.012 (0.040)</td>
<td></td>
<td></td>
<td>0.018 (0.021)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Puma level data. Maximum likelihood estimates. Note: Standard errors in parenthesis. See note to Table 1.
<table>
<thead>
<tr>
<th></th>
<th>Infectious disease</th>
<th>Cancers</th>
<th>Diabetes</th>
<th>Cardio–vascular</th>
<th>Accidents</th>
<th>Homicides</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Black men in age group 25–64 (obs = 2636)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−1.726 (0.538)</td>
<td>−0.290 (0.097)</td>
<td>−0.286 (0.256)</td>
<td>−0.219 (0.089)</td>
<td>0.081 (0.129)</td>
<td>−0.932 (0.226)</td>
</tr>
<tr>
<td>ln(y), own age/race/sex group</td>
<td>−0.513 (0.053)</td>
<td>−0.294 (0.052)</td>
<td>−0.175 (0.124)</td>
<td>−0.274 (0.033)</td>
<td>−0.353 (0.044)</td>
<td>−0.409 (0.064)</td>
</tr>
<tr>
<td>Mean ln(y), puma</td>
<td>0.246 (0.427)</td>
<td>0.112 (0.105)</td>
<td>−0.135 (0.246)</td>
<td>−0.155 (0.091)</td>
<td>−0.127 (0.146)</td>
<td>−0.257 (0.200)</td>
</tr>
<tr>
<td><strong>White men in age group 25–64 (obs = 5264)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−2.018 (0.828)</td>
<td>−0.327 (0.051)</td>
<td>−0.235 (0.181)</td>
<td>−0.424 (0.057)</td>
<td>0.123 (0.103)</td>
<td>−1.807 (0.215)</td>
</tr>
<tr>
<td>ln(y), own age/race/sex group</td>
<td>1.122 (0.567)</td>
<td>−0.237 (0.028)</td>
<td>−0.191 (0.116)</td>
<td>−0.265 (0.029)</td>
<td>−0.269 (0.092)</td>
<td>−0.661 (0.049)</td>
</tr>
<tr>
<td>Mean ln(y), puma</td>
<td>−2.872 (0.874)</td>
<td>0.050 (0.045)</td>
<td>−0.409 (0.184)</td>
<td>−0.177 (0.047)</td>
<td>−0.424 (0.106)</td>
<td>−1.759 (0.760)</td>
</tr>
<tr>
<td><strong>Black women in age group 25–64 (obs = 2759)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−1.572 (0.935)</td>
<td>−0.070 (0.088)</td>
<td>0.437 (0.196)</td>
<td>0.039 (0.077)</td>
<td>0.432 (0.206)</td>
<td>−0.520 (0.260)</td>
</tr>
<tr>
<td>ln(y), own age/race/sex group</td>
<td>−0.395 (0.104)</td>
<td>−0.097 (0.071)</td>
<td>−0.101 (0.095)</td>
<td>−0.267 (0.044)</td>
<td>−0.395 (0.085)</td>
<td>−0.458 (0.066)</td>
</tr>
<tr>
<td>Mean ln(y), puma</td>
<td>0.623 (0.770)</td>
<td>−0.074 (0.097)</td>
<td>−0.180 (0.240)</td>
<td>−0.224 (0.092)</td>
<td>−0.564 (0.245)</td>
<td>0.362 (0.215)</td>
</tr>
<tr>
<td><strong>White women in age group 25–64 (obs = 5264)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction white in puma</td>
<td>−1.808 (0.299)</td>
<td>−0.349 (0.053)</td>
<td>0.318 (0.146)</td>
<td>−0.512 (0.056)</td>
<td>0.081 (0.110)</td>
<td>−1.298 (0.179)</td>
</tr>
<tr>
<td>ln(y), own age/race/sex group</td>
<td>−0.628 (0.048)</td>
<td>−0.142 (0.051)</td>
<td>−0.351 (0.116)</td>
<td>−0.344 (0.027)</td>
<td>−0.068 (0.085)</td>
<td>−0.566 (0.049)</td>
</tr>
<tr>
<td>Mean ln(y), puma</td>
<td>−0.878 (0.550)</td>
<td>0.178 (0.058)</td>
<td>−0.562 (0.175)</td>
<td>−0.152 (0.053)</td>
<td>−0.351 (0.115)</td>
<td>−0.081 (0.234)</td>
</tr>
</tbody>
</table>

Puma-level data. Maximum likelihood estimates. Note: Standard errors in parenthesis. See note to Table 1.
5.6. Results by cause of death

In order to shed more light on the relative income effects, we separate mortality into by-cause categories, and estimate our models separately for each category. In this section we focus our analysis on the younger (25–64) age groups. We examine six categories of mortality: infectious diseases, cancers, diabetes, cardiovascular, accidents, and homicide. We estimate our relative income model at the puma level with terms for own income, mean puma income, age-demographic-region controls, and fraction white in the puma.

Results are presented in Table 5. Overall, two patterns emerge. First, the estimated standard errors on mean puma income are large, indicating that there is limited statistical power when disaggregating mortality by cause. Second, there is no systematic pattern indicating that low relative income is harmful to health: in only one case out of 24 is puma income statistically positively correlated with mortality.

Mortality from diabetes and cardiovascular disease is of special interest, since these illnesses may be related to stress. If the psychosocial stress model is correct, we would expect to see that low relative income is related to greater mortality for these conditions. We do not find evidence that this is so. Diabetes mortality in our data is not correlated with relative income for black men or women, and is negatively related to puma income for white men and women. A similar pattern holds for cardiovascular mortality. It is also interesting to consider the cancer results together with the cardiovascular results. If the correlation between cancer mortality and relative income was due to greater levels of behavioral risks such as smoking, we would also expect cardiovascular and cancer mortality to be correlated with relative income in a similar fashion. This is not the case.

We also include two external causes of death in our analysis, accidents (which for the younger age groups is largely comprised of motor vehicle accidents) and homicide. Mean puma income is not significantly related to mortality from accidents for black males. However, puma income is strongly protective with respect to accident mortality for all other groups. Finally, for homicide mortality we find that the impacts of puma income are not precisely estimated, but it appears to be protective for white men. For these causes of death, we do not find evidence that living in a wealthier area (holding own income fixed) is a health hazard.

When we re-estimate the models in Table 4 with puma-level fixed effects, the coefficients on mean puma income are generally imprecisely estimated. The exceptions to this include cardiovascular mortality (for which puma income is protective for all four groups), cancer (for which puma income is protective for black men, and black and white women), and homicides (for which puma income is protective for white men, and positively associated with mortality for white women). Again, considering both the cross section and the fixed effects estimates, there is no strong evidence to suggest that having wealthier neighbors is harmful to one’s health. If anything, the evidence is that it is protective.

6. Conclusion

The results in this paper provide no evidence that being relatively poor is harmful to health. If anything, the evidence indicates that conditional on one’s own income, having wealthier neighbors is either uncorrelated, or negatively correlated, with own mortality. For example, for younger black men a 10 percentage point increase in average puma income (holding own income fixed) is estimated to have the same effect on mortality as a 4 percentage point increase in own income. In no case do we find robust support for the idea that there are general health threats from having wealthier neighbors.
Our findings stand in contrast to much of the public health and social epidemiological literature on the topic. In several papers and a book, Wilkinson (1996, 1997, 1998) has argued that low relative income is primary reason why those who are poor are in worse health than those are wealthier. An implication is that policies that reduce the incomes of the rich will improve the health of the poor. Our evidence, however, suggests that low relative income is not a health hazard. Indeed, our findings suggest that younger black men and women may benefit (in terms of reduced mortality) from having wealthier neighbors. If this association reflects a causal effect, then policies that induce very low income families to move into wealthier areas may have health benefits on those who move. However, we note that Kling et al. (2004) find mixed evidence for this conclusion in the context of the Moving to Opportunity experiment, which randomly provided poor families with vouchers to live in wealthier neighborhoods. Although adults in the treatment group experienced improvements in mental health and declines in obesity, they did not experience improvements in several other physical health measures. In any case, however, we do not find evidence that runs counter to the Pareto principal: increases in own incomes are shown to be associated with reduced mortality, and increases in the incomes of others who live in the same area are not associated with poorer health outcomes.

The evidence in this paper does not speak strongly to the mechanisms that underlie the association between relative income and mortality, although it takes some first steps in that direction. Some of the mortality causes presumed to be associated with psychosocial stress (such as diabetes and cardiovascular mortality) show relative income effects opposite to those expected. The fact that the association appears for some but not all causes of death makes it unlikely that a single simple story can be identified. A possible direction for future research is to move beyond the correlational strategy employed in this paper, identifying exogenous shifts in other’s income and then tracing their external health impacts. An alternate direction for future research is to study how the income levels of regions influence the production of health-related public goods and the prices of health-related goods and services.

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References


