PERGAMON

Social Science & Medicine 56 (2003) 1139–1153

SOCIAL SCIENCE & MEDICINE

www.elsevier.com/locate/socscimed

Mortality, inequality and race in American cities and states

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Abstract

A number of studies have found that mortality rates are positively correlated with income inequality across the cities and states of the US. We argue that this correlation is confounded by the effects of racial composition. Across states and Metropolitan Statistical Areas (MSAs), the fraction of the population that is black is *positively* correlated with average white incomes, and *negatively* correlated with average black incomes. Between-group income inequality is therefore higher where the fraction black is higher, as is income inequality in general. Conditional on the fraction black, neither city nor state mortality rates are correlated with income inequality. Mortality rates are higher where the fraction black is higher, not only because of the mechanical effect of higher black mortality rates and lower black incomes, but because *white* mortality rates are higher in places where the fraction black is higher. This result is present within census regions, and for all age groups and both sexes (except for boys aged 1–9). It is robust to conditioning on income, education, and (in the MSA results) on state fixed effects. Although it remains unclear why white mortality is related to racial composition, the mechanism working through trust that is often proposed to explain the effects of inequality on health is also consistent with the evidence on racial composition and mortality.

Keywords: Mortality; Inequality; Race; Cities; States; Income, USA

Introduction and background

In recent years there has been a great deal of interest in whether income inequality is a health hazard in the sense that individuals are less healthy in places where income is more unequally distributed. The strongest advocate of the income inequality hypothesis has been Richard Wilkinson (1992, 1996, 2000), who has put forward a variety of evidence, from individual, area, cross-country, and time-series data. A survey of the subsequent debate over this evidence is given in Deaton (2001b). In this paper, we are concerned with one of the most prominent of these relationships, the ecological association between income inequality and mortality across states and cities in the United States. One version of this correlation is shown in Fig. 1 below, which plots (directly) age-adjusted all-cause mortality against the gini coefficient of per adult-equivalent income; the District of Columbia is included and, although it has both higher mortality and higher inequality than any state, it lies on the regression line. (The definitions of these and other data are given in the next section.) The positive correlation between income inequality and mortality across the US states was first shown in studies by Kaplan, Pamuk, Lynch, Cohen, and Balfour (1996) and Kennedy, Kawachi, and Prothrow-Stith (1996a, b). Lynch et al. (1998) reproduced the correlation using data from 282 Metropolitan Statistical Areas (MSAs) in 1990, finding that the loss of life from income inequality "is comparable to the combined loss of life from lung cancer, diabetes, motor vehicle crashes, HIV infection, suicide, and homicide in 1995". These, and other related studies, are collected in Kawachi, Kennedy, and Wilkinson (1999).

In this paper, we investigate the robustness of the connection between income inequality and mortality across states and MSAs, with particular attention to the

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0277-9536/03/$ - see front matter
PII: S0277-9536(02)00115-6

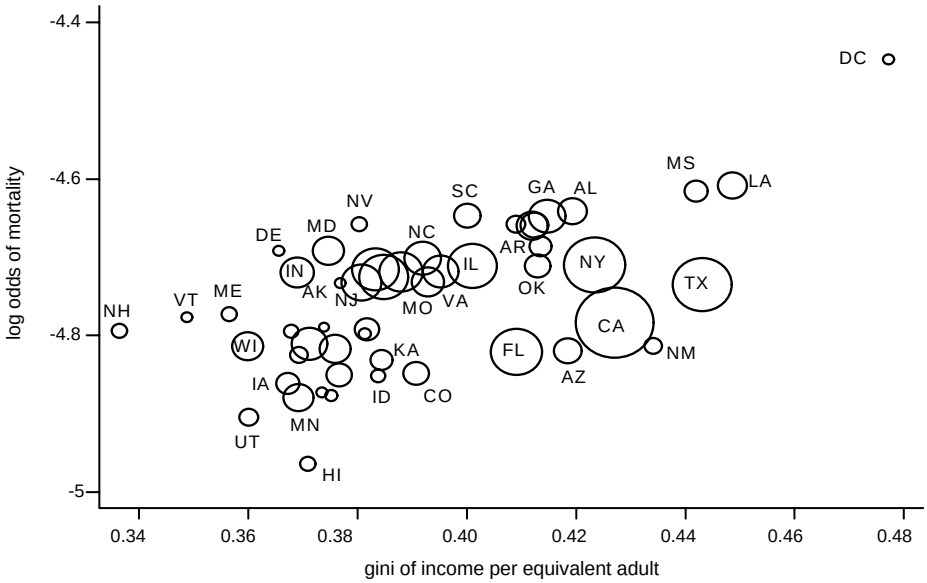

Fig. 1. Inequality and age-adjusted mortality across US states, 1990 (circles have diameter proportional to population).

effects of race as a potential confounder. That the spatial link between income inequality and mortality might be spurious is suggested by several studies in the literature. For example, Mellor and Milyo (2001) pool census data on the 48 continental states from 1950, 1960, 1970, 1980, and 1990 and find that the relationship between the gini coefficient and mortality is not robust to the inclusion of various plausibly important controls, such as education, urbanization, and race, see also Muller (2002). There is also a lack of consistently positive evidence on the role of income inequality from follow-up data on individuals, in the National Longitudinal Mortality Study, where there is a relationship neither in the individual data nor in the state-level data (Deaton, 2001a), from the National Health and Nutritional Examination Survey (Fiscella & Franks, 1997), from the Panel Study of Income Dynamics (Daly, Duncan, Kaplan, & Lynch, 1998), and only relatively weak evidence from the National Health Interview Survey (Lochner, Pamuk, Makuc, & Kawachi, 2001). Furthermore, a study of cities and provinces in Canada failed to find any relationship between income inequality and mortality, Ross et al. (2000).

In the results presented below, we show that, once we control for the fraction of the population that is black, there is no relationship in 1980 nor in 1990 between income inequality and mortality across either states or cities. This result does *not* come from the pooling of black and white mortality; as emphasized in the earlier literature, the correlation between income inequality and mortality is present for each race separately. Instead, our results come from the fact that white mortality rates are higher in places where a higher fraction of the population is black. Although we do not know what causes this result, we note that the mechanisms that are emphasized in the literature on *inequality* and health, that work through the psychosocial environment, particularly stress and trust, are equally plausible as mechanisms through which *race* affects health. We also examine the robustness of the effect. In particular, the relationship between the fraction black and white mortality rates holds within broad geographical regions, and so is not driven by a comparison of the South with the rest of the country. The correlation is also robust to the inclusion of controls for state fixed effects and for education, holds for nearly all age groups and for males and females, and cannot readily be attributed to variations in local health provision.

The paper is organized as follows. The next section discusses data sources and methodology. We then present the main results from the states and MSAs, following with a section on robustness that investigates some alternatives. We focus on mortality among whites though, in line with earlier literature, we also show some results for all races pooled. The results for other races are of considerable interest in their own right, but we confine our attention here to one element of the story, leaving for future work the comparative results. That the fraction black increases mortality rates for blacks is shown in Miller and Paxson (2001) and Reidpath (2000).

Data and methodology

The data on mortality are taken from the *Compressed Mortality Files* (CMF), from the National Center for Health Statistics at the Center for Disease Control. The CMF contain a complete census of all deaths by year from 1968 to 1994, by cause of death, race, sex, age group, and county of residence, except for Alaska where only state-level data are available. The CMF files also provide population totals for each cell, which we use to calculate mortality rates as well as racial composition. We use data on deaths in 1980 and 1990. The county identifiers are used to aggregate deaths and populations to the Metropolitan Statistical Area (MSA) level; once again, Alaska is an exception and is excluded from our MSA analysis. We match 287 MSAs in 1980 and 1990. Not everyone lives in an MSA; the 287 MSAs used here contain 79.9 and 80.7 percent of the total population of the US in 1980 and 1990, respectively. The data aggregated by state cover the entire population of the US. The CMF data are disaggregated by 13 age groups; we preserve these age groups when aggregating to the state and MSA levels, and then calculate age-adjusted all-cause mortality rates by direct adjustment to the US population in 1990. Age adjustment is done separately by sex, and separately for all races combined and for whites alone. Hence, for example, the age-specific mortality rates for white females in New York City are weighted by the age-distribution of white females in the US population in 1990.

The creation of a consistent set of MSA mortality data requires a mapping of counties into MSAs, as well as a method of handling changes in the definitions of MSAs between the two years. MSAs are defined by the US Office of Management and Budget (OMB) and in some cases, their geographical boundaries changed from 1980 to 1990. MSAs are always collections of counties, except in New England where they are collections of cities and towns, so that counties may be split between multiple MSAs. Because the mortality data come at the county level, aggregating mortality to the MSA level is relatively straightforward outside of New England. Within New England we use New England County Metropolitan Areas, OMB's county-based alternatives to the city- and town-based MSAs.

Data for income and education are taken from the 5 percent public-use samples of the 1980 (A sample) and 1990 censuses. Income data in the census refer to the previous year, i.e. 1979 or 1989, which is one year earlier than the mortality data from the CMF. Other choices of timing could be investigated, for example by averaging mortality over several years around the censuses, or by using mortality several years after each census, but given the arbitrariness of any choice, the one year lag seems as reasonable as any.

Census data do not come at the county level, but at the level of County Group in 1980 and PUMA ("Public Use Microdata Area") in 1990. The 1990 PUMAs do not necessarily match the 1980 County Groups, nor are they necessarily collections of counties. Instead, they can be parts of counties, single counties, collections of whole counties, or collections of parts of counties. Our procedure is to use the 1990 MSA definitions and create, as closely as possible, consistently defined metropolitan areas in 1980. We begin with the mapping of County Groups and PUMAs into MSA definitions given in Jaeger, Loeb, Turner, and Bound (1998) for cities with populations over 250,000 people. In 1990, 20 of the cities with populations over one million people are designated by OMB as CMSAs, essentially combinations of MSAs, and are treated as units by Jaeger et al. We split these CMSAs into their component cities, technically referred to as Primary Metropolitan Statistical Areas (PMSAs). For example, the Dallas-Fort Worth CMSA is composed of the Dallas PMSA and the Fort Worth-Arlington PMSA, and we treat each as one observation in our analysis of MSAs. We also include 110 smaller cities; these are defined by OMB, and are generally places with populations of at least 100,000 but less than 250,000. In the end, we have 287 MSAs consistently defined in 1980 and 1990. 110 of these are the MSAs in Jaeger et al., 54 come from our disaggregation of CMSAs, 110 are smaller cities that were not included by Jaeger et al., and there are 13 New England County Metropolitan Areas.

In some cases the 1980 County Groups and the 1990 PUMAs contain areas that are partly inside and partly outside of an MSA. For these, it is not possible to create an exact match between an area in 1980 and 1990, nor between Census and mortality data. In these cases, a judgment must be made as to whether to drop the unit, if it is impossible to make a reconciliation by aggregating up to a reasonably sized larger unit, or to include it, if the differences between the two years are small. Of our 287 MSAs, 237 contain identical counties in 1980 and in 1990. Of the 50 others, only a small fraction of the population lives in the areas that are included in only one year. For each MSA, we calculated the sum of the populations in the two years that lived in counties included in both years, and divided it by the sum of the total populations in the two years. The resulting ratio is unity for the 237 consistent MSAs. For the other 50, the mean of the ratio is 93.2 percent, the median is 94.7 percent, and the minimum is 71.9 percent. The definitions of our MSAs, and their relationship to counties, County Groups, and PUMAs is detailed in an Addendum to this paper that is available at http://www.wws.princeton.edu/~chw.

Each individual in the census is assigned an MSA according to the rules discussed above. Each is also assigned the adult equivalent household income for the

household in which he or she lives, where equivalent income is calculated by dividing total household income by the number of adults plus half the number of children, defined as household members aged 18 and younger. Logarithms of income and of income per equivalent are calculated at the individual level, and averaged over MSAs and states. Income from the 1980 census—which relates to 1979—is converted to 1989 prices using the CPI in order to make it comparable with data from the 1990 census. We make no attempt to deal with top-coding.

Our primary measure of income inequality is the gini coefficient, which is calculated on an individual basis, using income per equivalent adult imputed to each individual. We calculate gini coefficients and income levels separately by race and by sex, as well as over all races and both sexes. Note that if all households consisted of a male and female couple, and because the same per equivalent income is imputed to each, the male and female ginis would be identical. Although this is not the case, the cross-MSA correlation between the (white) male and female ginis is 0.97 in 1980 and 0.95 in 1990. For each individual we also record an indicator for the level of education achieved according to five categories; less than high school, high school, some college (education post high-school, but without a bachelor's degree), completed college, post-graduate education (in 1980, more than 16 years of education, in 1990 holding a master's, professional, or doctoral degree). The binary indicators are averaged within states and MSAs, for people aged 25 and above, again separately by sex and race. This gives us data, for example, on the fractions of adult men or women in Ohio or in Dallas whose highest education is in each of the five categories.

In the results that follow we use OLS regressions with either state or MSA-level data. The dependent variable is an age-adjusted mortality rate converted to a log odds. The independent variables are area averages of the explanatory variables, such as the logarithm of income per equivalent, or state or MSA-wide estimates of the gini coefficient, racial composition, or the fractions of the population whose highest level of education is in each of the education classes. Each regression is weighted by the square root of the population at risk in each state or MSA.

Basic results for states and MSAs

Table 1 shows results from the state data, including the District of Columbia, and pooling data from 1980 and 1990, so that there are 102 observations in each regression. All regressions include a dummy variable for 1990; if there is a decline in mortality rates that is unexplained by the included variables, the regression coefficient on the dummy should be negative, as is always in fact the case. The first two columns in the left-hand panels, for all males and all females irrespective of race, show the results that are typically reported in the literature. In the first regression, with no other variables included, the logarithm of per adult equivalent income has a protective effect that is about twice as large for males as for females, −0.22 versus −0.09. The 1990

Table 1
Log odds of mortality regressions: 50 US states plus DC, 1980 and 1990 pooled

	All males			White males only			
Equivalent income	−0.22 (4.1)	−0.11 (1.9)	−0.14 (3.3)	−0.11 (2.5)	−0.09 (2.2)	−0.09 (2.1)	−0.16 (4.2)
Gini coefficient		1.42 (3.9)	−0.24 (0.8)		0.92 (3.6)		0.01 (0.0)
Gini among whites						0.62 (1.8)	
Fraction black			0.71 (10.3)				0.42 (5.7)
1990 dummy	−0.11 (6.5)	−0.16 (7.9)	−0.11 (7.3)	−0.13 (9.4)	−0.16 (10.3)	−0.15 (8.3)	−0.12 (8.7)
	All females			White females only			
Equivalent income	−0.09 (2.0)	0.00 (0.1)	−0.02 (0.5)	0.02 (0.4)	0.03 (0.6)	0.03 (0.6)	−0.02 (0.5)
Gini		1.08 (3.4)	−0.36 (1.1)		0.38 (1.6)		−0.30 (1.0)
Gini among whites						0.24 (0.7)	
Fraction black			0.51 (7.4)				0.26 (3.2)
1990 dummy	−0.07 (5.0)	−0.11 (6.2)	−0.66 (4.1)	−0.08 (6.5)	−0.09 (6.2)	−0.09 (5.1)	−0.07 (4.2)

Notes: Equivalent income is the mean of the logarithm of income per adult equivalent, calculated on an individual basis with 1979 repriced to 1989 using the CPI. The gini coefficient relates to income per equivalent, again on an individual basis. The Gini coefficient among whites is calculated using white incomes only. Gini coefficients are calculated separately for males and for females, after imputing household income per equivalent adult to each individual. There are 102 observations in all regressions. The dependent variable is the log odds of age-adjusted mortality; mortality is adjusted to the 1990 US population; age adjustment is done separately by sex, and separately for all groups, and for whites. The figures in brackets are absolute *t*-values. All regressions are weighted by the square root of the relevant population.

dummy has a coefficient of −0.11 for men and −0.07 for women so that there is a background improvement in mortality that is not explained by changes in income, or at least cannot be explained by assigning the same effect to income over time as it is estimated to have over states. The second column shows the effects of including income inequality in the form of the gini coefficient of income per equivalent adult. The gini coefficient attracts large and significant positive coefficients for both males and females. Over the 51 states in 1990, the mean of the gini of per equivalent income was 0.37 with a standard deviation of 0.02, so if we move from one standard deviation below the mean to one above the mean, from Vermont to Mississippi, or from Michigan to Florida, the log odds increases by 0.057 for men and by 0.043 for women, corresponding to relative risks of (approximately) 1.06 for men and 1.04 for women. The coefficient on income is not significantly different from zero in these regressions. The coefficients on the 1990 dummy are larger than before. Mortality declined from 1980 to 1990 while income inequality increased, so that the hazardous effects of inequality that are estimated from the interstate differences must be offset by the time dummy.

That the estimated effects of income inequality are potentially confounded by the effects of race has been recognized since the first papers on the topic. Blacks have higher mortality rates than whites and, on average, have lower incomes, so that in places with a substantial black population, both income inequality and mortality tend to be higher. That there is some such problem is shown by the third column in the first panel. When the fraction of the state population that is black is added to the regressions, it attracts a significantly positive coefficient, and the coefficient on the gini coefficient is no longer significantly different from zero. But this regression does little more than illustrate that there is a problem with the first two columns. Indeed, as noted by Kaplan et al. (1996), separate regressions by race find that income inequality is estimated to be a hazard for each.

The results for whites alone are shown in the right-hand panel of Table 1. The coefficient on the gini coefficient in the second column of the right hand panel is a good deal smaller for whites than it was for all races taken together, and for women the effect is no longer significantly different from zero. Once we look only at whites, it is unclear which concept of income inequality is the appropriate one, inequality among whites in the state, or inequality among everyone in the state. The third column shows the effect of replacing the gini coefficient for all incomes with the gini coefficient for white incomes alone. Both coefficients are further reduced, and neither is significantly different from zero. From this, we can deduce that the component of income inequality that matters for mortality is income inequality between races, not income inequality within them. Because blacks are in the minority and have lower incomes, the all-race gini coefficient will be larger where the fraction black is larger, which suggests including it in the regressions. The final columns show the regression containing the fraction black together with the original all-race gini coefficient. The fraction black is estimated to increase white mortality for both males and females. Taking the same example as before, the difference between Vermont and Mississippi, with fractions black of zero and 0.34, gives relative risks of 1.14 for white men, and 1.09 for white women.

The results in Table 1 are important because they show that the effects of income inequality on mortality at the state level are not robust to the inclusion of the fraction of the population that is black. They thus demonstrate that the income inequality hypothesis is incorrect, but they tell us nothing about what actually drives mortality rates. In these state-level data, the fraction black is higher in the southern states, and it is not difficult to think of reasons why mortality, including white mortality, might be higher in the South. But alternative hypotheses are difficult to test with data from only 51 states, so it is useful to move on to the larger number of observations offered by the MSA-level data where it is possible, for example, to look at different regions separately. Quite apart from the fact that there are more of them, cities are more plausibly salient than states for the health of their residents.

Our 1990 MSA data yield the same correlation patterns as those used by Lynch et al. (1998) (LKP). Although the underlying census data are the same, LKP calculate inequality measures from grouped income data, as opposed to the individual records used here. Neither method is necessarily better; the individual data avoid discarding information, but the grouped data may better deal with topcoding. Moreover, by using the aggregate data, LKP are effectively using the responses from all individuals who filled out the "long form" (about 15 percent of the population), compared with the 5 percent public use sample used here. For the key variables, the correlation coefficients between age-adjusted mortality and the gini coefficient of per equivalent income is 0.28 (compared with 0.25 with the gini of per capita income in LKP) between age adjusted mortality and the logarithm of per equivalent income is −0.32 (compared with −0.28 with per capita income in LKP), and between the logarithm of per equivalent income and its gini coefficient is −0.58 (compared with −0.28 between mean income per capita and the gini of income per capita in LKP). Between the top and bottom quartile of the gini of income per equivalent we find a difference in overall age-adjusted mortality of 75 per 100,000, compared with 65 per 100,000 in LKP. Fig. 2, which corresponds to Fig. 1 for the states, shows the correlation between the gini and the log odds of age

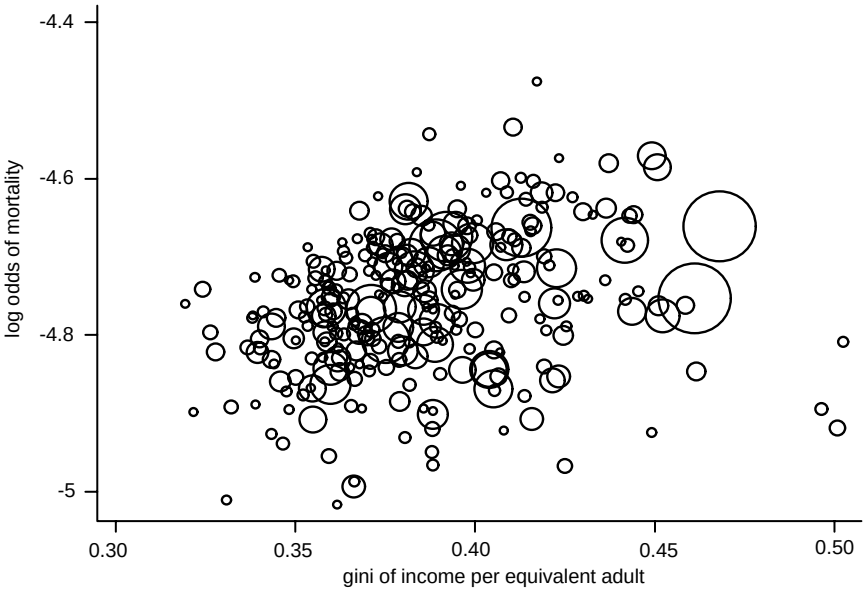

Fig. 2. Income inequality and mortality across US MSAs, 1990 (circles have diameter proportional to population).

Table 2
Log odds of mortality regressions: 287 Metropolitan Statistical Areas, 1990

	All males			White males only			
Equivalent income	−0.16 (5.6)	−0.12 (3.7)	−0.17 (7.2)	−0.10 (4.1)	−0.08 (3.4)	−0.09 (3.5)	−0.15 (6.8)
Gini coefficient		0.55 (2.7)	−0.38 (2.5)		0.46 (3.0)		−0.09 (0.6)
Gini among whites						0.16 (0.9)	
Fraction black			0.83 (16.7)				0.50 (9.0)
	All females			White females only			
Equivalent income	−0.09 (3.8)	−0.05 (2.0)	−0.09 (4.1)	−0.01 (0.7)	−0.01 (0.3)	−0.02 (0.7)	−0.06 (2.8)
Gini		0.44 (2.6)	−0.42 (2.8)		0.26 (1.9)		
Gini among whites						−0.03 (0.2)	−0.22 (1.5)
Fraction black			0.54 (13.0)				0.31 (6.4)

Notes: Equivalent income is the mean of the logarithm of income per adult equivalent, calculated on an individual basis with 1979 repriced to 1989 using the CPI. The gini coefficient relates to income per equivalent, again on an individual basis. The Gini coefficient among whites is calculated using white incomes only. Gini coefficients are calculated separately for males and for females, after imputing household income per equivalent adult to each individual. There are 287 observations in all regressions. The dependent variable is the log odds of age-adjusted mortality; mortality is adjusted to the 1990 US population; age adjustment is done separately by sex, and separately for all groups, and for whites. The figures in brackets are absolute *t*-values. All regressions are weighted by the square root of the relevant population.

adjusted mortality for all persons. Each circle represents an MSA, and the diameters of the circles are in proportion to the population of each, a procedure that makes it clear that the correlation is not driven by a few large MSAs.

Table 2 reproduces Table 1 using MSA data for 1990. As before, the gini coefficient is a significant risk factor when the data are pooled across races, and once again, the effect is removed (indeed reversed) once we control for the fraction of each MSA's population that is black. When we restrict the regression to whites, the fraction black is a significant health hazard for both men and women and the coefficients are similar to those estimated from the state data. Once the fraction black is controlled for, income inequality has no effect, whether we use income inequality over everyone, as shown in the final column, or income inequality among whites, not shown. Table 3 repeats the MSA results for

Table 3
Log odds of mortality regressions: 287 Metropolitan Statistical Areas, 1980

	All males			White males only			
Equivalent income	−0.16 (4.8)	−0.15 (3.7)	−0.19 (6.3)	−0.05 (1.8)	−0.05 (1.6)	−0.10 (3.1)	−0.17 (5.9)
Gini coefficient		0.08 (0.4)	−1.15 (6.3)		0.05 (0.3)		−0.90 (5.3)
Gini among whites						−0.64 (3.2)	
Fraction black			0.74 (15.7)				0.54 (10.2)
	All females			White females only			
Equivalent income	−0.07 (2.1)	−0.04 (0.9)	−0.08 (2.2)	0.05 (1.6)	0.05 (1.5)	0.00 (0.1)	−0.04 (1.3)
Gini		0.25 (1.1)	−0.91 (4.3)		−0.02 (0.1)		−0.75 (3.7)
Gini among whites						−0.54 (2.5)	
Fraction black			0.56 (11.2)				0.34 (5.7)

Notes: Equivalent income is the mean of the logarithm of income per adult equivalent, calculated on an individual basis with 1979 repriced to 1989 using the CPI. The gini coefficient relates to income per equivalent, again on an individual basis. The Gini coefficient among whites is calculated using white incomes only. Gini coefficients are calculated separately for males and for females, after imputing household income per equivalent adult to each individual. There are 287 observations in all regressions. The dependent variable is the log odds of age-adjusted mortality; mortality is adjusted to the 1990 US population; age adjustment is done separately by sex, and separately for all groups, and for whites. The figures in brackets are absolute *t*-values. All regressions are weighted by the square root of the relevant population.

1980. These are shown separately from the 1990 results because, unlike 1990, the gini has no effect on mortality even in the all-race regressions. Even so, the final regressions for white males and females are similar to those for 1990. Whites die at younger ages in places where a larger fraction of the population is black and, conditional on fraction black, there is no mortality risk associated with income inequality. Indeed, in both Tables 2 and 3, and in the latter significantly so, income inequality is associated with *lower* mortality once we control for the racial composition of the MSAs.

The key to understanding the mechanics behind these results is the relationship between income and race across American states and cities. Average incomes for the population as a whole, as well as average incomes among blacks, are negatively correlated with the percent of the population that is black, but the reverse is true for average white incomes. Average incomes of whites are *higher* in cities with a larger fraction of blacks. This divergent behavior of black and white incomes means that the income difference between blacks and whites is larger in cities with larger black populations, which is what induces the relationship between overall income inequality and racial composition. Of course, this *does not* mean that racial composition and income inequality are the same thing, nor that either one is an equally valid marker for the same underlying health risk. In regressions containing both the fraction black and income inequality, the former drives out the latter so that, even if we cannot tell what it is about a high fraction black that drives the mortality results, it is not the associated income inequality.

It is important to check whether the basic results in Tables 1–3, that income inequality plays no role in city or state mortality conditional on racial composition, are robust to various alternative estimation procedures and data definitions. We report only a summary of the various experiments; more detailed tables are available on request from the authors.

The income variable used in our regressions is the mean in each state or city of the logarithm of per equivalent income. This choice was motivated by the fact that, in the individual data of the National Longitudinal Mortality Study, the probability of death is loglinear in equivalent income, see Deaton (2001a). If the within-unit distribution of income is approximately lognormal, it then follows that the logarithm of the mortality rate (which is closely approximated by the log odds) is linear in the mean and variance of log income. We report results using the variance of log income below but, in order to conform to most of the literature, we began with the gini coefficient as our measure of income inequality. An alternative specification of income is to work with the mean of income per equivalent, or its logarithm, a possibility that needs to be handled with care because, as pointed out to us by a referee, the difference between the logarithm of mean income and the mean of the logarithm of income is itself an inequality measure, the mean log deviation, or Theil-Bernoulli measure. We have replicated Table 2, the results for MSAs in 1990, replacing the mean of the logarithm of income per equivalent with the logarithm of the mean of income per equivalent. The estimates are very similar to those reported in Table 2, especially on the race and income variables. The coefficients on the

gini coefficient are somewhat larger in the equations excluding the race variable, 0.77 instead of 0.55 in the second column and 0.57 instead of 0.47 in the fifth column, but once the race variable is introduced we get the same patterns of sign reversal and/or insignificance. Nothing about the confounding story is changed if we work with the log of mean income instead of the mean of the logs.

We have also repeated Table 2 using a range of different measures of income inequality. In particular, we repeated the regressions with the gini replaced, in turn, by the variance of logs, the mean log deviation, the coefficient of variation, the Theil measure (the mean of $z \ln z$ where z is the ratio of income per equivalent to mean income per equivalent), the relative mean deviation (or Robin Hood Index), and three Atkinson inequality measures with the inequality aversion parameter, ε, set at 0.5, 1.0, and 2.0. With the exception of the Atkinson measure with $\varepsilon = 2$ where the correlation coefficient averages 0.45, these measures are each correlated with the other with correlation coefficients greater than 0.85. (Note that we are using the unit record data, and the sensitivity of the Atkinson index to low incomes increases with the value of ε, which is why the $\varepsilon = 2$ Atkinson measure is somewhat different from the others. When the data are grouped into income classes, this measure is also similar to the others.) The patterns in Table 2 replicate with all measures. Without race, inequality appears to be a mortality risk. With conditioning on race, inequality is insignificant or attracts a negative sign as shown in Table 2. Nothing in our results depends on the particular measure of income inequality.

Another test of specification is to control for the fraction black in a more flexible way. At the suggestion of a referee, we have divided the 1990 MSAs into five quintiles according to the fraction of the population black and then re-estimated the regressions of the log odds of mortality on the mean log income per equivalent and the gini coefficient. For men and for all quintiles together, the coefficient on the gini coefficient is 0.55 with a t-value of 2.7—the second column of Table 2—while for each of the quintiles (with t-values in brackets) we get −0.41 (0.9), −1.51 (4.4), 0.23 (0.7), −0.40 (1.2), and 0.20 (0.7). For white men, where the original coefficient on the gini is 0.46 (3.0), the by quintile estimates are −0.13 (0.3), −0.81 (2.8), 0.61 (1.9), 0.07 (0.2) and 0.39 (1.5). For women, the pattern is similar, and the only significant estimate (for all women, quintile 2) is negative, as are four out of the five estimates. The more flexible treatment of fraction black removes the apparent hazardous effects of income inequality just as did our original specification.

We have also repeated Table 2 without using population weights. This is not our preferred strategy—the MSAs are of very different size, and the regressions should be thought of as on an individual basis not an MSA basis—but is nevertheless a useful specification text. The effect of the gini in the first specification is now somewhat smaller, 0.36 instead of 0.55 for men, but the patterns of sign reversal and subsequent insignificance for whites is the same in the unweighted regressions as that shown in Table 2. We have also experimented with adding the mean age of the inhabitants of an MSA as an explanatory variable. This allows for the fact that the mortality rates on the left-hand side of the regression are age-adjusted, while the explanatory variables are not, Rosenbaum and Rubin (1984). Although mean age is significant in its own right, adding it has no effect on the pattern of the other results. We also recognize that our income figures are not adjusted for differences in the cost of living across different cities and that the cost of living might be correlated with other variables in the analysis. The US official statistical system does not produce spatial price indexes; instead, we have used the price index in Fuchs, McClellan, and Skinner (2001), which is derived by regressing the US Chamber of Commerce city price index on indexes of local wage rates and property values and using the results to extrapolate to the full set of MSAs. Replacing nominal by real income per equivalent changes none of our results, although the effect of income on mortality is estimated to be larger.

Finally, we note that our standard errors are calculated on the assumption that the error terms in the regressions are independent across observations. This is particularly problematic in Table 1, where we have two observations for each state, and may be questionable in Tables 2 and 3, where some MSAs may be geographically linked, for example by state. We have rerun the regressions in Table 1 with robust estimates of standard errors allowing for heteroskedasticity and for arbitrary correlations over time within each state. This does not change the parameter estimates (by construction), but reduces the t-values somewhat, so that the gini coefficient is now sometimes insignificant. The race variable always attracts large t-values, whether the standard errors are robustly calculated or not. The same is true for Table 2 when we calculate standard errors allowing for heteroskedasticity and for arbitrary correlations across MSAs within the same state. For example, in the third column of the top left block of Table 2, the (absolute) t-value on income drops from 7.2 to 4.2, remains 2.5 for the gini coefficient, and falls from 16.7 to 14.8 for the fraction black.

Discussion and further exploration

What is it about the racial composition of places that affects their mortality rates? Or are the effects of racial composition as spurious as those of income inequality?

One interpretation is that our results demonstrate, once again, the ecological fallacies and aggregation biases that are always a potential risk in using city or state level data and for which there is already a good deal of evidence in the health and education literatures; examples are Geronimus, Bound, and Neidert (1996) (health) and Hanushek, Rivkin, and Taylor (1996) and Loeb and Bound (1996) (education). On this view, area racial composition is a variable that is useful for demonstrating the invalidity of the inequality hypothesis, but should be treated no more seriously as a determinant of mortality.

However, there is evidence from data at the *individual* level that the effects of racial composition on mortality need to be treated seriously in their own right and are not simply an artefact of aggregation. LeClere, Rogers, and Peters (1997), using data from the National Health Interview Survey merged to mortality and census data, show that the mortality of men and women of all races is higher when the census tract in which they live has a larger fraction of African Americans. Indeed, conditioning on individual socioeconomic characteristics together with the racial composition of the census tract is sufficient to eliminate the mortality differential between blacks and non-Hispanic whites. Although individual ethnicity is highly correlated with ethnicity at the census tract level, the same is true for the individual socioeconomic characteristics (income, education, and marital status). But when LeClere et al. condition on the census tract average of these variables, there is no change in the estimated mortality effects of the individual characteristics. At a more aggregated level, Deaton (2001a), uses a two-stage estimation method with data from the National Longitudinal Mortality Study to show that the fraction of the *state* population that is black is an individual-level risk factor for whites. There is no similar effect of state-level income inequality. These two studies show that there is an association between the ethnic composition of the area of residence and *individual* mortality.

Which leads us back to the question of what it is about racial composition that matters. As with income inequality, there are a reasons why the effect might be spurious. One that seems unlikely is that race is a proxy for income inequality itself. The argument would be that income is poorly measured, so that income inequality is poorly measured, and that the correlation between racial composition and income inequality is sufficiently strong that racial composition is a *better* measure of "true" income inequality than is measured income inequality itself, so that it drives measured income inequality out of the regression. This argument seems far-fetched, particularly given the robustness of our findings to alternative measures of income inequality, each of which is sensitive to measurement error in a different way.

Another hypothesis concerns education. If the presence of a large black minority results in low levels of education for both blacks and whites, and if education is important for lowering mortality rates, we might find a spurious correlation between racial composition and education. We test this hypothesis by using the census data on individuals' education levels to calculate the fraction of the white population in each MSA whose highest level of education falls into various classes. Table 4 shows the results of the mortality regressions with education included using pooled MSA data from 1980 and 1990.

These results strongly support the view that people with higher education have lower mortality rates, but they do nothing to moderate the estimated effect of the fraction black on white mortality rates. The MSA results are consistent with other results using both regional and individual data; a college education, even some college education, is protective compared with only a high school education. (Though note that for men, postgraduate education adds nothing, and for women, those with postgraduate education are no more protected than high-school graduates or high-school drop outs.) But the main effect of the inclusion of the education variables is not on the estimated effect of racial composition, but on the estimated effect of income, which is now estimated to be mildly hazardous. Such findings are consistent

Table 4
Education, income, inequality and white mortality across MSAs in 1980 and 1990

	White males		White females	
Equivalent income	0.052	(2.1)	0.081	(3.3)
Gini coefficient	0.263	(2.0)	−0.084	(0.6)
Fraction black	0.388	(10.6)	0.227	(5.9)
No high school	0.059	(0.7)	0.060	(0.8)
Some college	−0.266	(3.0)	−0.204	(2.3)
College graduate	−0.530	(3.9)	−0.883	(6.1)
Post-graduate	−0.512	(4.2)	0.277	(1.5)
1990 dummy	−0.108	(10.3)	−0.020	(2.1)

Notes: Pooled data, 1980 and 1990, 574 observations. OLS regressions with the log odds of age-adjusted mortality as the dependent variable; age-adjustment is to the 1990 US population and is done separately for males and females. Equivalent income is the mean in the MSA of log income per adult equivalent at 1989 prices. The gini coefficient is calculated on an individual basis from income per equivalent adult over all races. The schooling variables are the fractions of people (white men or women, respectively) in the MSA whose highest education is as shown. The omitted category is high school graduate. Absolute *t*-values are shown in parentheses. All regressions are weighted by the square root of the relevant population.

with an earlier literature in economics, Grossman (1975), Fuchs (1989, 1993), and Garber (1989), which argues that it is education, not income, that is protective of health, as well as Ruhm (2000), who argues that business-cycle induced increases in income are hazardous to health. However, they stand in sharp contrast to analyses on individual level data, particularly those using the National Longitudinal Mortality Study, where income is importantly protective of health even conditional on education, see Elo and Preston (1996) and Deaton and Paxson (2001). The question of whether it is income, education, or some combination that is important for health matters a great deal for policy, especially for arguments about the role of fiscal policy in public health. However, income is not our main concern here, so we do no more than note the puzzle.

Another possible explanation for our main finding is that the provision of public services, especially health services, is poorer in places with a larger black population. Such an explanation would require that the provision of such services is in itself an important determinant of (white) mortality rates, something that is challenged by an extensive literature that imputes a small or negligible role to access to health care in explaining differences in mortality by socioeconomic status, see for example the review by Adler et al. (1994). Moreover, there is evidence against the proposition that health expenditures are indeed lower in places with a larger black minority. Alesina, Baqir, and Easterly (1999) argue on theoretical grounds that racial diversity is likely to decrease the political willingness to provide public goods, but in their empirical analysis find that local public expenditures, including expenditures on health, are *higher* in places where there is more ethnic fractionalization—which in the context of the US means in places where there is a large fraction of the population that is black.

Even so, expenditure may not be the relevant indicator, especially in localities where there is corruption and, more generally, the quality of health care may be only loosely related to the levels of expenditure, so that the issue can hardly be said to be closed. Fuchs et al. (2001) show that, among the elderly Medicare population, the effects of racial composition are largely confined to mortality through heart disease. The risk of mortality from heart disease varies greatly from one hospital to another, depending on whether or not the hospital is well-equipped and trained to deal with acute myocardial infarction, McClellan and Staiger (1999). If it is also true that hospitals are less likely to be so equipped in areas with a larger African American population, then the mortality risk would extend, not only to African Americans, but also to whites who experience a heart attack in an area with a large black population.

A third line of enquiry is to look at the results by region. In the state-level results with which we began, the correlations between mortality, income inequality, and fraction black had much to do with the South, where all three quantities tend to be higher than in the rest of the country. One of the main advantages of working with the MSA data is the ability to work *within* regions, and thus to eliminate the suspicion that the results are being driven by the South versus the rest of the US. There is also the hypothesis, advanced by Fuchs et al. (2001), that the mortality differences might come from selective migration. Migrants are typically healthier than those who stay behind so that, if they migrate from areas with larger to smaller minority black populations, they will increase mortality in the transmitting region and reduce it in the receiving region and, depending on initial conditions, may induce a correlation between white mortality rates and the fraction black. A serious examination of this hypothesis is beyond the scope of this paper, but to the extent that migrations are between regions, intraregional and interregional correlations are likely to differ.

Table 5 shows the results of running a stripped down regression—log odds of white male and female mortality on the mean of the logarithm of per equivalent income, the gini, the fraction black, and the 1990 dummy—for four regions of the US, the North-East, the South, the Mid-West, and the West. The effects of income inequality are inconsistent from region to region, and are more often estimated to be protective than hazardous. There is also some heterogeneity in the effects of income, with income less protective in the West than elsewhere. But the effects of the fraction black are consistently and significantly hazardous in all four regions, though the effects are about twice as large in the North-East and in the South than in the West and Mid-West. In any event, the effect on white mortality does not reflect some unmeasured difference between the South and the rest of the US.

Although there are more MSAs than states, there are not enough to allow us to run cross-MSA regressions state by state. However, it is possible to run the stripped-down regressions with the inclusion of dummy variables, one for each state; when MSAs cross state boundaries, we assign them to the state in which the majority of its population lives. Allowing for state effects allows us to control for unmeasured state-level factors that contribute to mortality rates and that are potentially correlated with the fraction of the population that is black. However, the fraction black remains a hazard to health in these regressions. For white males, and using the same regressions as in Table 5, the coefficient on the fraction black is 0.49 with a t-value of 8.7; for females, the coefficient is 0.48 with a t-value of 9.9. However, if we go one step further, and include dummies for each of the 287 MSAs, the coefficient on the fraction black

Table 5
Regional regressions of mortality across MSAs in 1980 and 1990

	North east				South			
	White males		White females		White males		White females	
Equivalent income	−0.242	(7.4)	−0.118	(4.0)	−0.209	(6.3)	−0.149	(4.3)
Gini coefficient	0.198	(0.8)	−0.363	(1.4)	−1.252	(5.5)	−0.833	(3.5)
Fraction black	0.401	(3.3)	0.498	(4.6)	0.438	(7.1)	0.386	(6.4)
1990 dummy	−0.113	(9.8)	−0.096	(7.3)	−0.075	(5.7)	−0.028	(2.1)
	Mid-west				West			
Equivalent income	−0.300	(7.3)	−0.172	(3.8)	−0.076	(1.5)	−0.079	(2.1)
Gini coefficient	−0.414	(1.5)	−0.682	(2.0)	0.662	(2.2)	0.141	(0.6)
Fraction black	0.993	(10.0)	0.920	(8.3)	0.843	(3.5)	0.956	(5.7)
1990 dummy	−0.129	(9.8)	−0.054	(3.2)	−0.149	(8.0)	−0.067	(4.7)

Notes: Pooled data, 1980 and 1990. OLS regressions with the log odds of age-adjusted mortality as the dependent variable; age-adjustment is to the 1990 US population as a whole, but is done separately for males and females. Variables as defined in previous tables. Each column represents a regression. There are 98 observations for the North East, 216 in the South, 158 in the Mid-West, and 102 in the West. The standard Census regions are: North-East: Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont; South: Delaware, District of Columbia, Florida, Georgia, Maryland, N. and S. Carolina, Virginia, W. Virginia, Alabama, Kentucky, Mississippi, Tennessee, Arkansas, Louisiana, Oklahoma, and Texas; Mid-West: Illinois, Indiana, Michigan, Ohio, Wisconsin, Iowa, Kansas, Minnesota, Missouri, Nebraska, N. and S. Dakota; West: Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Utah, Wyoming, Alaska, California, Hawaii, Oregon, and Washington. All regressions are weighted by the square root of the relevant population.

becomes small and insignificantly different from zero. Unfortunately, this result is not very informative. When MSA dummies are included, we are essentially running a regression of the changes in the log odds of mortality against the changes in mean income and the racial composition of the MSAs. This regression suffers from a lack of precision because the fraction black does not change much over a decade. Beyond that, all we learn is that the fraction black is standing proxy for some constant or slowly changing factors that are important in the cross-section, but not in the time series. We learn nothing about what those factors might be. We also note that the analysis of changes on changes puts much more strain on the timing—which years of mortality to match with the 1979 and 1989 income and race data from the census—than is the case with the cross-sectional results.

Finally, we look at the age composition of mortality. Because the cause of death differs by age, locating the effects of racial composition in the age distribution may give some clue about the mechanisms involved. The age-specific regressions also protect us against potential artefactual effects associated with age-adjustment, which requires an essentially arbitrary choice of base population. Table 6 presents the estimated effects of income and of fraction black on the mortality rate in thirteen age groups. The form of these regressions differs from before. In a few of the smaller MSAs, there are no recorded deaths in the specific age groups in one or other of the years, and such observations cannot be included in a regression with the log-odds as dependent variable. Dropping them and running the standard regression produces results that are qualitatively similar to those in the table. Even so, we present the results of regressions using the mortality rate itself as the dependent variable. On the right hand side, in addition to the fraction black and the dummy for 1990, we include the mean of income per equivalent, rather than the mean of its logarithm. The table shows the coefficients for the fraction black and for income, by sex and age, with males on the left and females on the right. These are scaled so that the numbers in the left-hand panel are estimates of the effects on the white mortality rate per 1000 of moving from an MSA with zero to one with 100 percent black population, while those in the right-hand panel are the effects of an additional $1000 on the mortality rate per 1000. Average mortality rates across the MSAs (weighted by population) are shown for comparison.

These results do nothing to resolve the puzzle. The effects of racial composition, like those of income, are different at different ages, and vary largely in proportion to the level of mortality itself, so that the effect on the log odds would be roughly the same at all ages. With the exception of males aged 1 through 9, the fraction black is estimated as a significant risk to mortality at all ages. It is particularly high for 15–19 year old males, falling off for 15 years thereafter, but rising rapidly with age thereafter. The effect is always positive, and always significantly different from zero. Miller and Paxson

Table 6
Age, racial composition, and white mortality across MSAs in 1980 and 1990

Age group	Males					Females				
	Mortality	Fraction black $\times 10^3$		Mean income $\times 10^7$		Mortality	Fraction black $\times 10^3$		Mean income $\times 10^7$	
0 to 1	8.4	3.06	(2.9)	−2.25	(6.0)	6.5	2.31	(2.5)	−1.30	(3.8)
1–4	0.4	0.06	(0.5)	−0.18	(3.6)	0.4	0.30	(2.8)	−0.24	(5.1)
5–9	0.2	0.11	(1.4)	−0.09	(2.6)	0.2	0.14	(2.3)	−0.05	(1.8)
10–14	0.3	0.27	(3.3)	−0.15	(4.5)	0.2	0.24	(3.6)	−0.16	(5.4)
15–19	1.1	1.00	(5.0)	−0.38	(4.4)	0.4	0.48	(4.9)	−0.17	(3.8)
20–24	1.4	0.61	(2.4)	−0.14	(1.4)	0.4	0.27	(2.7)	−0.01	(0.3)
25–34	1.8	0.85	(3.9)	−0.15	(2.0)	0.6	0.32	(3.8)	−0.06	(1.9)
35–44	2.7	1.29	(3.7)	0.26	(2.4)	1.2	0.60	(4.6)	−0.12	(2.5)
45–54	5.4	4.09	(9.0)	−0.95	(7.4)	3.0	0.93	(4.1)	−0.32	(4.1)
55-64	14.5	9.96	(12.5)	−3.13	(14.4)	8.2	2.38	(5.6)	−0.65	(4.0)
65–74	33.7	19.72	(11.5)	−6.91	(12.4)	19.3	4.98	(4.9)	−1.65	(4.1)
75–84	78.1	29.93	(8.0)	−9.48	(7.3)	48.6	13.71	(6.6)	−2.12	(2.2)
85+	182.0	20.49	(2.4)	−0.85	(0.3)	144.1	23.25	(4.8)	0.09	(0.0)

Notes: Pooled data, 1980 and 1990. Each number comes from an OLS regression with probability of death on the left-hand side and the fraction black, mean income per equivalent in 1989 prices, and a dummy for 1990 on the right-hand side. The coefficients on fraction black are multiplied by 1000 and are therefore the effect of a unit change (from 0 to 1.0) on the mortality rate per 1000. The coefficient on income is multiplied by 10,000,000, and so represents the effects of an additional $1000 of per equivalent income on the mortality rate per 1000. All regressions are weighted by the square root of the relevant age and sex specific population.

(2001) further show, using PUMA level data, that the fraction black is correlated with the death from a range of diseases; for example, for white males aged 25 to 64, the effect is present for death from infectious disease, cancer, homicide, and cardiovascular disease, but not for diabetes nor accidents.

Fig. 3 shows scatter-plots between the fraction black and the mortality of white males at selected ages using the MSA data for 1990; once again, the diameters of the circles are proportional to population size. Note that each plot has its own scale for the vertical axis. The figures provide an immediate visual counterpart to the results in Table 6, and they also establish that the correlations do not depend on one or two peculiar MSAs. Even in the three central panels, where there is one large MSA in the upper-right (New York City), the significance of the positive correlation is not affected by its exclusion or down-weighting.

Conclusions

Cross-section regressions across American states and cities show that, conditional on racial composition, income inequality does not raise the risk of mortality. The fraction of the population that is black is a significant risk-factor for mortality, not only for the population as a whole—which would follow mechanically from the fact that blacks have higher mortality rates than whites—but for both blacks and whites separately. Our empirical results provide no evidence that the association between the fraction black and white mortality is the result of confounding. The effect is robust to conditioning on education, it is present for all age-groups except boys aged 1 to 9, and it is present within geographical regions of the country.

The literature on income inequality and health has postulated that income inequality generates psychosocial stress that is directly harmful to health. More specifically, the negative effects of income inequality on trust are frequently identified as a plausible mechanism (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997; Putnam, 2000, Chapter 20). Yet the argument that income inequality corrodes trust and social capital works as well or better for ethnic composition. In particular, Alesina and La Ferrara (2000) analyze individual-level data from the census and the General Social Survey (the same source used in aggregate form by Kawachi et al.) to show that individuals are less likely to report that they trust their neighbors when they live in an MSA with either high income inequality or a large fraction of African Americans, but that when both variables are entered together, only the ethnic composition has any significant effect. Just as in our mortality analysis, area racial composition drives out income inequality as an explanation of trust, so that to the extent that lack of trust is a mechanism that raises the risk of mortality, the data implicate racial composition, not income inequality. Such an account also reconciles the American and Canadian evidence; Ross et al. (2000) show that there is no correlation between income inequality and mortality in Canadian provinces or cities.

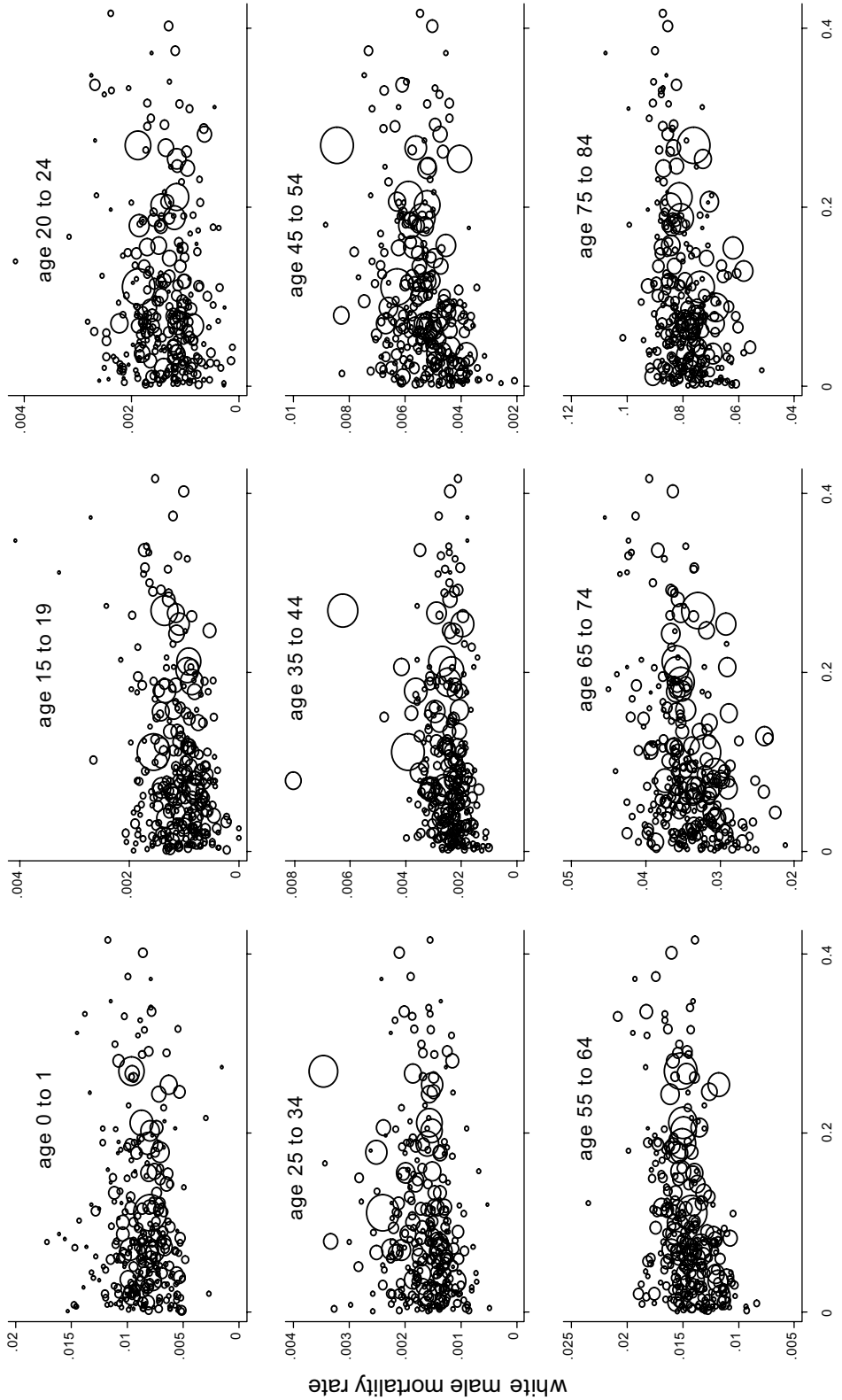

Fig. 3. Income inequality and mortality across US MSAs, 1990 (circles have diameter proportional to population).

If income inequality is a mask for the effects of racial composition on trust and thence on mortality, then there should be no relationship between income inequality on mortality in Canada, where race lacks the social salience that it carries in the United States.

Acknowledgements

We are grateful to Sam Bowles, Anne Case, Susan Dynarski, Vic Fuchs, Sandy Jencks, Adriana Lleras-Muney, Jeff Milyo, Christina Paxson, Robert Putnam, Richard Wilkinson, and two anonymous referees for helpful comments and discussions during the preparation of this paper. We gratefully acknowledge financial support from the John D. and Catherine T. MacArthur Foundation through its Network on Poverty and Inequality in Broader Perspectives, and from the National Institute on Aging through a grant to the National Bureau of Economic Research.

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