Demography (2021) 58(6):2041–2063 Published online: 3 September 2021
DOI 10.1215/00703370-9463660

Inequality in Place: Effects of Exposure to Neighborhood-Level Economic Inequality on Mortality

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ABSTRACT This study contributes to the debate on whether income inequality is harmful for health by addressing several analytical weaknesses of previous studies. Using the Panel Study of Income Dynamics in combination with tract-level measures of income inequality in the United States, we estimate the effects of differential exposure to income inequality during three decades of the life course on mortality. Our study is among the first to consider the implications of income inequality within U.S. tracts for mortality using longitudinal and individual-level data. In addition, we improve upon prior work by accounting for the dynamic relationship between local areas and individuals' health, using marginal structural models to account for changes in exposure to local income inequality. In contrast to other studies that found no significant relation between income inequality and mortality, we find that recent exposure to higher local inequality predicts higher relative risk of mortality among individuals at ages 45 or older.

KEYWORDS Mortality • Inequality • Neighborhoods • Life course • Marginal structural models

Introduction

Income inequality in the United States has surged to levels not seen for a century (Burtless and Jencks 2003; Neckerman and Torche 2007). Since the 1970s, the variance in incomes of families with children increased by two thirds (Western et al. 2008) and the top percentile share of incomes doubled (Atkinson et al. 2011). Among the potential sequelae of growing inequality is increased mortality risk. As income inequality has risen in the United States, mortality rates have risen among some groups, and the U.S. trend in life expectancy has diverged from trends in other post-industrial democracies. A recent National Academy of Sciences report noted this circumstantial evidence and underscored controversial claims about the relationship between inequality and mortality as a top priority for research (Woolf and Aron 2013).

Although there already exists considerable work on income inequality and health (Beckfield 2004; Hu et al. 2015; Kawachi et al. 1997; Wilkinson and Pickett 2006), relatively little research considers the consequences of income inequality at a local

ELECTRONIC SUPPLEMENTARY MATERIAL The online version of this article (https://doi.org/10.1215/00703370-9463660) contains supplementary material.

or neighborhood level. Existing research on the impact of inequality on health usually considers aggregate inequality in relatively large geographic areas—nations (Beckfield 2004; Edwards and Tuljapurkar 2005; Kondo et al. 2009; Lynch et al. 2004; Shkolnikov et al. 2011; Vincens et al. 2018; Wilkinson 1992; Zheng 2012), states (Backlund et al. 2007; Deaton 2001; Kennedy et al. 1998; McLeod et al. 2004; Rasella et al. 2013), metropolitan areas (Deaton and Lubotsky 2003; Mellor and Milyo 2001), or counties (Fiscella and Franks 1997; Franzini et al. 2001; LeClere and Soobader 2000; Yang et al. 2017). In comparison, the consequences of income inequality in more local contexts are not well understood, especially when considering mortality as the outcome.

In addition, studies of inequality in local contexts are inconclusive. Although individuals' actual experiences of income inequality are likely shaped by their local area, in which inequality could be socially corrosive for communities or hold significant negative implications because of social comparisons (Jencks and Mayer 1990), the relation between local inequality and mortality often appears low or null (Franzini et al. 2001; Subramanian and Kawachi 2004; Wilkinson and Pickett 2006); these patterns are observed within several countries outside of the United States (Blakely et al. 2003; Clough-Gorr et al. 2015; Osler et al. 2002; Shibuya et al. 2002; Veenstra 2002). Furthermore, studies of the implications of local inequality for mortality often rely on ecological data (Brodish et al. 2000; Massing et al. 2004; McLaughlin and Stokes 2002; McLaughlin et al. 2001; Shi et al. 2005), which can lead to issues with individual-level inference (Gravelle 1998; Hernán 2012; Xie 2013). Finally, studies of inequality generally rely on regressions using fixed effects or lagged covariates (Beckfield 2004; Blakely et al. 2000; Daly et al. 2001; Subramanian and Kawachi 2006; Vincens et al. 2018), which can overlook causal mediators and overcontrol for the dynamic and recursive relationship between the local inequality and other time-varying covariates (Wodtke et al. 2011).

To address these limitations, we use detailed individual-level longitudinal data from the Panel Study of Income Dynamics (PSID) to estimate the effects of U.S. tract-level inequality on mortality over a period of up to three decades. Although the PSID is the longest-running nationally representative longitudinal survey of its kind, the data on mortality included in the PSID are rarely used, and to our knowledge have not been used to study the implications of local inequality. In our individual-level and time-sensitive analyses, we estimate marginal structural models with balanced or pseudorandomized observations across exposure levels (Sharkey and Elwert 2011; Wodtke 2013; Wodtke et al. 2011) to account for the dynamic relationship between local areas and individuals' health—in which some of the social conditions that predict mortality may be both causes of local income inequality (e.g., through selection) and consequences of income inequality. Attention to dynamic interplay is increasing in studies of the consequences of socioeconomic advantage for health and mortality under the life course approach (Elo 2009; Leventhal and Brooks-Gunn 2000; Mayer 2009; Wodtke et al. 2011) but is largely absent in studies of inequality. Our study shows that exposure to recent local inequality predicts higher mortality risk, even net time-varying characteristics such as family and neighborhood-level income, contradicting arguments that local inequality has few implications for health and mortality.

Theoretical Effects of Local Income Inequality on Mortality: Null, Indirect, and Direct Effects

When we consider the relationship between local income inequality and mortality, there are at least three possibilities. First, income inequality may be unrelated to mortality, which would suggest that local income inequality is not an important mortality risk. Second, income inequality may have negative implications for mortality *indirectly*, by which we mean through the implications of inequality for other time-varying covariates (such as individual or local socioeconomic status). Third, local income inequality may directly increase mortality risk if it increases stressful social comparisons or reduces neighborhood-level trust and social cohesion (without operating through known and observed mediators such as individual or local socioeconomic status).[1] In this section, we review existing studies that shed light on these three possibilities.

Although some scholars have not observed an association between income inequality and population health (Beckfield 2004; Deaton and Lubotsky 2003; Lynch et al. 2004; McLeod et al. 2004; Mellor and Milyo 2001), the evidence base is relatively stronger at higher levels of aggregation (e.g., nations, regions, states). In a meta-analysis of 168 studies, Wilkinson and Pickett (2006) found a significant association between population health and income inequality within nations (Edwards and Tuljapurkar 2005; Lynch et al. 2004; Shkolnikov et al. 2011) and regional areas (Daly et al. 2001; Kaplan et al. 1996; Kawachi et al. 1997; Kennedy et al. 1998; Subramanian et al. 2003; Subramanian and Kawachi 2004, 2006). In contrast, fewer studies have considered whether income inequality within neighborhoods or *small* local areas (e.g., tracts, communities, parishes) matters for population health.

The few studies that considered small local areas found little evidence that population health was patterned by local inequality, especially for mortality as an outcome. A Danish study found that income inequality in parishes was not associated with mortality after adjusting for individual risk factors, but concluded that this was most likely a result of Denmark's strong welfare system (Osler et al. 2002). Similarly, large-scale cross-sectional data from New Zealand revealed no association between inequality in census subregions and mortality (Blakely et al. 2003). Income inequality within coastal communities in British Columbia was also unassociated with mortality within those communities (Veenstra 2002). And in Japan, only a weak cross-sectional association between income inequality and mortality was seen at the prefecture level (Shibuya et al. 2002), while a study of inequality in Swiss municipalities found that it was actually associated with lower mortality (Clough-Gorr et al. 2015). Lastly, a

[1] "Direct effects" refer to recent effects that are not mediated by our observed time-varying mediators. In contrast, "indirect effects" operate through observed mediators. A limitation of this terminology is that the existing literature offers but a blurry boundary between direct and indirect. For example, if social comparisons were observed over time, they can be treated as a mediator as well. However, for theoretical reasons, we theorize social comparisons as part of a direct effect—studies have not established an independent effect of social comparisons on mortality outside of studies of inequality, while numerous studies have identified the effects of our observed set of potential mediators (e.g., family and neighborhood inequality).

longitudinal study within the U.S. context reported no association between mortality and inequality in counties between the mid-1970s and late 1980s (Fiscella and Franks 1997). Theoretically, it is possible that inequality within local communities is not detrimental (e.g., for social cohesion or relative deprivation) compared with inequality at broader scales (Franzini et al. 2001; Subramanian and Kawachi 2004; Wilkinson and Pickett 2006). It could also be that, even net of mean levels of advantage, there are positive spillovers within local areas of having more advantaged neighbors that nullify negative consequences of inequality (Jencks and Mayer 1990:118).[2] Taken together, these studies point to the following null hypothesis: *income inequality in U.S. census tracts*[3] *is not associated with individual mortality* (Hypothesis 1A).

Alternatively, local inequality could shape mortality. To understand whether this is the case, it is essential to measure individual-level characteristics and individual-level mortality directly. Failing to do so renders individual-level inference vulnerable (Gravelle 1998).[4] Estimating such effects using solely aggregate-level data is also problematic when the distribution of epidemiological risk factors is profoundly different between the populations affected (Hernán 2012) or when treatment effects are heterogeneous (Xie 2013). Since most studies of the inequality–health link employ aggregate data (Macintyre et al. 2002; Neckerman and Torche 2007; Truesdale and Jencks 2016), it remains unknown whether neighborhood income inequality could increase the risk of mortality.

In understanding the inequality–mortality link, it is possible that the implications of local inequality are indirect. For example, if local income inequality limits socioeconomic gains in that area by failing to attract businesses or offering fewer relevant services, and socioeconomic disadvantage increases mortality risk, then the effects of inequality on mortality risk may be mediated by absolute disadvantage. There is currently no consensus on whether residential mobility is selected on factors related to health, such as absolute advantage, in ways that would bias estimates of place-effects (Geronimus et al. 2014), such as inequality-effects. However, the potential for indirect effects should be analyzed with care because they could in theory also be confounders (e.g., if individuals with lower socioeconomic status are more likely to be selected into unequal neighborhoods). Since such confounders may also be mediators of an indirect effect of inequality on mortality, as discussed above, they should not be overcontrolled. Although traditional individual-level longitudinal data analyses in health research leverage repeated observations to account for fixed (Beckfield 2004) or lagged effects (Blakely et al. 2000; Daly et al. 2001; Subramanian and Kawachi 2006), these strategies overcontrol for indirect effects and may lead to bias

[2] Some scholars argue that inequality can be beneficial when it leads to some affluence in otherwise homogeneously poor neighborhoods. This can introduce positive spillovers for infrastructure and services (Joseph et al. 2007; Nandi et al. 2006; Wilson 1987) or lead to positive network effects on health behaviors (Fan et al. 2016).

[3] Census tracts are small county subdivisions designed to approximate neighborhoods (Bischoff and Reardon 2014). While tracts are not interchangeable with neighborhoods, we proxy local inequality using tract inequality because tracts are the smallest unit of analysis for which nationally representative census data are available with comparable boundaries and variables over time. There are currently around 73,000 tracts, with roughly 4,200 families in each.

[4] According to Gravelle (1998), positive correlation between population mortality and inequality can arise at aggregate even if inequality has no effect on individual risk of mortality if the relationship between income and health is nonlinear (an example of the "ecological fallacy").

(Wodtke et al. 2011).[5] We use marginal structural models (MSM) to pseudorandomize sequences of exposure to inequality relative to other time-varying covariates as well as regression-adjusted models.[6] In turn, we hypothesize that: *local inequality could indirectly increase the risk of mortality* (Hypothesis 1B).

Lastly, neighborhood inequality could directly shape mortality, via at least two theoretical mechanisms. The first is social comparison (i.e., relative deprivation), which refers to the idea that individuals aim to "rank high in comparison with the rest of community in point of pecuniary strength" (Veblen 1899:39–40). If, however, individuals do not consider themselves as "ranking" favorably to their relevant comparison group, this may lead to so-called "status anxiety." Status anxiety and lower subjective socioeconomic position are believed to increase stress levels and chronic inflammation (Layte et al. 2019) and have been widely associated with adverse consequences for health and mortality, including poor self-rated health (Ostrove et al. 2000), obesity (Goodman et al. 2003), depression (Kahn et al. 2000; Muramatsu 2003), and cardiovascular illness (Singh-Manoux et al. 2003; Tang et al. 2016). Proponents of the status comparisons hypothesis generally assume that the latter explain the link between societal inequality—for example, in terms of incomes—and health (Wilkinson and Pickett 2006, 2010). In line with this hypothesis, studies have indicated that local inequality diminishes well-being, reduces self-reported happiness (Alderson and Katz-Gerro 2016; Firebaugh and Schroeder 2009), and induces anxiety (Hildebrand and Van Kerm 2009), as well as morbidity (LeClere and Soobader 2000; Massing et al. 2004; Soobader and LeClere 1999). If inequality exacerbates chronic stress through these means, it is reasonable to expect a host of implications, including for mortality. The many pathways through which stress leads to morbidity and mortality are only beginning to be understood, but stress compromises immune and cardiovascular systems to such an extent, and increases vulnerability to so many diseases, that it has been likened to more rapid aging (Wilkinson and Pickett 2010). While some understandings of "class" are relative to wider society, stress due to comparing and being compared is also likely to occur in local areas, meaning that *local* inequality could theoretically increase mortality risk.

Second, inequality may be socially corrosive. Social corrosion refers to the erosion of relations or expectations within groups as well as of social and collective resources (Kawachi et al. 1999; Neckerman and Torche 2007). Inequality could be socially corrosive in local contexts by reducing social cohesion and civic engagement, which are necessary to create and sustain public goods and resources, within local areas (Kawachi et al. 1999; Nandi et al. 2006). Weaker community life, less trust, and less civic engagement reduce the ability of local areas to provide public goods and resources that are protective to health (Kawachi et al. 1999). This in turn shapes mortality risk; for instance, local areas can differ in their provision of cardiopulmonary resuscitation in out-of-hospital cardiac arrests (Iwashyna et al. 1999) or in engagement

[5] The association between intragenerational changes in income and occupation and local inequality requires further research. However, since causal implications are plausible in both directions (dynamic selection), our methodology ensures that the estimates of the inequality–mortality link are robust to these processes.

[6] The existence of indirect effects would explain why studies have found no relationship between local inequality and mortality, but contradicts the idea that local inequality does not matter.

with public infrastructure in reporting hazardous or unsafe environments (Sampson and Raudenbush 2001). Neighborhood corrosion can not only reduce social engagement, increase the risk of accidental death, and weaken community life (Wilkinson and Pickett 2010), it can also trigger hostility and violence, which are especially relevant in local areas, between individuals who are in close proximity (Lynch et al. 2004; Messner et al. 2004; Ross et al. 2001; Wilkinson and Pickett 2010). Overall, these ideas suggest that: *living in U.S. census tracts with greater income inequality increases mortality directly* (Hypothesis 1C).

To summarize, Hypothesis 1A presents a null hypothesis in which local inequality does not matter, as many observers have concluded. Hypothesis 1B presents an alternative hypothesis in which inequality can causally but indirectly increase mortality (dynamic selection). In testing for these hypotheses, we improve upon existing studies by differentiating between indirect (Hypothesis 1B) and null effects (Hypothesis 1A) and newly consider whether local inequality could directly increase mortality risk in other ways—such as through social comparison or neighborhood corrosion (Hypothesis 1C).[7]

Data and Methods

This study considers the consequences of inequality for mortality among respondents of the Panel Study of Income Dynamics, using data from the 1970–1997 annual survey waves. As the longest-running longitudinal survey of its kind, the PSID allows for almost three decades of follow-up on neighborhood inequality, during the same period when income inequality increased in the United States. Our primary aim is to determine whether neighborhood inequality increases individual risk of mortality. In sensitivity analyses, we consider differential vulnerability to inequality by assessing whether neighborhood contexts may matter more for the less advantaged (Daly et al. 1998; Dowd et al. 2011; Kahn et al. 2000), and we discuss whether findings are age sensitive by repeating analyses for older individuals. Analyses are not repeated for younger individuals because there are not enough cases of mortality for these samples.

Analytic Sample

We combine data from the PSID and the GeoLytics Neighborhood Change Database (NCDB) (GeoLytics 2003). The PSID was first conducted in 1968 on a national sample of around 4,800 families.[8] These families, together with new families formed by

[7] Evidence to support dynamic selection suggests that indirect effects play a critical role in explaining the effects of inequality but does not rule out direct effects. Lack of evidence of dynamic selection also does not necessarily imply lack of direct effects of inequality. Finally, note that we do not attempt here to disentangle the social corrosion and relative deprivation arguments, but we provide both as intuition for why direct effects might exist.

[8] The PSID oversampled low-income families. If the goal of the analyses were to generate descriptive statistics that are representative of the survey population, then survey weights would be necessary. However, since our main goal is to estimate the moderated effects of inequality, we do not use survey weights. In these analyses, sampling weights are unnecessary and inefficient, because the models already sufficiently

sample members over time, were interviewed annually between 1968 and 1997 and biennially thereafter. Data on neighborhood context and other covariates are unavailable after 1997. The PSID provides a unique opportunity for substantial longitudinal analyses, but thus far, follow-ups rarely span an entire life course, and mortality before midlife is rare. Thus, we follow the 4,774 individuals captured by the PSID from the point at which they were age 45 or older until they died, were lost to follow-up, or reached administrative end of follow-up (the 1997 wave of the PSID).

Individuals are captured by roughly 10 follow-ups before mortality, attrition, or end of follow-up.[9] Multivariate analyses account for issues of right-censoring due to differential attrition. Results are not sensitive to the inclusion versus omission of censoring weights. Data on 213 deaths were recovered from the restricted PSID mortality files. The mortality file, using survey responses and death certificate data, contains information on deaths of all individuals who were interviewed as part of PSID and died after their inclusion in the study. Here we note that the mortality follow-up data now included in the PSID are rarely used, and to our knowledge have not been used at all to estimate the effects of income inequality.

Inequality Exposure

Measurements of neighborhood inequality—the exposure of interest—as well as of other spatial covariates used in the study, are derived from the NCDB. The NCDB contains nationally representative tract-level data from the 1970–2000 censuses with tract boundaries and measures defined consistently over time (GeoLytics 2003). First, we compute inequality exposures during census years. Inequality exposure and other contextual variables for intercensal years are calculated using linear imputation.[10] When computing inequality measures, we focus on average family rather than household income because financial dependents, for instance children, are nested in families, whereas households sometimes contain unrelated adults. Family incomes are provided by the NCDB as counts within income bins, including an unbounded top category. We impute an upper bound by top-coding at 10 times the median income,[11] a conventional method that generates conservative (lower) estimates of Gini coefficients (Burtless and Jencks 2003). In sensitivity analyses following the main results, we show that results are robust to using other measures of inequality.

Within each income bin, we impute family incomes from a uniform (uninformative) distribution and calculate Gini coefficients for each tract in each census year.[12] The Gini coefficient is a measure of statistical dispersion that represents the income

control for the design variables—in this case, family income—on which oversampling occurs (Wodtke et al. 2016).

[9] For example, although a few individuals who reached age 45 in 1996 were observed only for a year, others who reached age 45 in 1970 were observed for 27 years (our analyses account for differences in birth cohort and sample attrition).

[10] Linear interpolation is appropriate given the clear secular trend in inequality over this period, and the necessity of retaining intercensal observations for estimation.

[11] Results remain similar even when substantially more conservative (lower) top bounds are imputed.

[12] The effects of inequality remain similar when means of income bins are assigned to families within the bin, but resultant Gini coefficients are lower because this artificially restricts variation.

distribution of an area's residents, where larger coefficients indicate higher levels of income inequality. The average Gini coefficient over all tracts in the data is 0.38.[13] We then divide census tracts into quintiles[14] using the national distribution of the inequality coefficients and create a time-varying ordinal treatment variable, coded 1 through 5, that records the neighborhood quintile in which an individual resides at each wave. Given our sample, our study examines inequality exposure in adulthood. While the long-term effects of local context during childhood or adolescence are likely to be important because of embeddedness in local context at an early age (Leventhal and Brooks-Gunn 2000; Wodtke et al. 2011) and the enduring effects of childhood health (Montez and Hayward 2014), our sample only captures exposure to inequality starting in middle-aged adulthood.

Covariates

Time-invariant baseline covariates include race, gender, educational attainment, birth cohort, and region of birth. Birth cohorts are coded in 10-year dummy variables (1920s, 1930s, and 1940s);[15] we expect recent birth cohorts to have lower mortality than older birth cohorts (Masters et al. 2012). Findings are robust to different groupings of birth cohort (and remain similar when a continuous measure for birth year is included instead). Race is coded 1 for Black and 0 for non-Black, and we expect a positive coefficient on race (Masters et al. 2014). Gender is coded 1 for female and 0 for male; we expect a negative coefficient (Beltrán-Sánchez et al. 2015). Educational attainment is categorized as less than high school, high school graduate, and at least some college, where we expect a negative association between educational attainment and mortality (Link and Phelan 1995). Geographic regions consist of six census regions (New England, Mid-Atlantic, Midwest, South, Southwest, and West), where we expect a positive coefficient on the indicator for South (Fenelon 2013).

Time-varying individual-level covariates in this analysis, measured at each wave k, include employment status and total family income. Given the importance of socioeconomic status as a fundamental cause of health, it is especially important to control for the effects of income and employment on mortality (Link and Phelan 1995; Miech et al. 2011). Employment status is coded as a three-category dummy variable, with categories indicating full employment, any unemployment in the previous year, and lack of presence in the workforce (e.g., no longer searching, retired). Income is measured as the log transformation (commonly used on skewed distributions such as income distributions) of the sum of taxable income of the family head, partner, and

[13] The five-year 2006–2010 Gini index for the United States as a whole is 0.467, and county-level Gini indices ranged from 0.64 to 0.21 (Bee 2012). Given that tracts are designed to be more homogeneous, and given recent increases in inequality, the derived distribution of tract-level Gini indices in this paper is reasonable.

[14] MSM estimation requires dichotomization as IPT estimates perform poorly with continuous treatments. We follow Wodtke et al. (2011) in taking a five-level treatment in the main results. In addition, although stronger effects are found when more than five categories are used, we limit exposure to neighborhood inequality to five levels for the sake of interpretability.

[15] Results are robust to whether a handful of individuals born in 1950–1952 are included in the 1940s cohort or given their own category.

other family members earned over the past year, reported in terms of 2010 dollars using the Consumer Price Index (CPI-U).[16]

Time-varying spatial covariates from census data are also included because neighborhood inequality is likely correlated with other contextual factors that could drive mortality (Huie et al. 2002). We include mean neighborhood family income (reported as a log-transformation and in terms of 2010 dollars); the racial composition of tracts in terms of proportion of Black residents, which is sometimes argued to be detrimental to health (Deaton 2001; McLeod et al. 2004); and total tract population to proxy health and epidemiological differences between metropolitan and nonmetropolitan areas (McLaughlin et al. 2001). Dummy categories account for any item-specific nonresponse, but results are similar when we simply drop missing observations as nonresponse is rare.

Estimation

Marginal structural models are a class of models[17] used for the estimation, from observational data, of the causal effect of a time-dependent exposure in the presence of time-dependent covariates that may be simultaneously confounders and mediators of the effect. The parameters of MSM models can be estimated using inverse probability of treatment (IPT) weighted estimators. IPT weighting is a technique for calculating statistics standardized to a population, in this case a counterfactual population that balances treatment assignment across prior confounders by giving more (or less) weight to individuals with covariate histories that are underrepresented (or overrepresented) in their current treatment group. In the weighted pseudopopulation, treatment at each wave is independent of time-varying confounders; in other words, exposure to different neighborhood contexts behaves as if it were sequentially randomized with respect to prior observed covariates.

MSM estimation has been successfully applied to questions using PSID data, for example, the effect of neighborhood disadvantage on high school completion (Wodtke et al. 2011), but has not been applied to studies of inequality and health. For a review of the limitations of conventional regression-based estimates of time-varying exposures with adjustments for time-varying covariates, see Wodtke et al. (2011). Essentially, controlling for time-varying covariates both (1) removes the causal effect of prior exposures through time-varying covariates (Sampson et al. 2002) and (2) introduces possible bias in the estimation of effects of exposure confounders (Greenland 2003; Pearl 2009). MSM estimation avoids these issues by reweighting observations on the basis of the distribution of exposures among different covariate levels rather than by stratifying analyses by covariates.

Below, we use the counterfactual framework to formally define the moderated exposures of interest. Let the sequence of observed or potential inequality exposures (treatment variables) experienced through wave k for a given individual be

[16] Results are not sensitive to the use of 2010 dollars versus raw dollars unadjusted for inflation.

[17] They are "marginal" because they model the marginal distribution of potential outcomes and "structural" because causal models are referred to as structural in the treatment-effects literature (Robins et al. 2000; Wodtke et al. 2011).

$\overline{a}_k=(a_1, \ldots, a_k)$, where $\overline{a}_k$ represents the exposure trajectory up to wave k. For each subject, baseline is defined to be the PSID wave, indexed by $k\in\{0,1, \ldots, K\}$, in which a subject is first observed. Let $Y_{\overline{a}_k}$ be a binary indicator for mortality at wave k given survival up to wave k and an (either observed or counterfactual) exposure trajectory. We wish to estimate the effect of one exposure trajectory $\overline{a}_k$ compared to another possible trajectory $\overline{a}_k'$, given by

$$E(Y_{\overline{a}_k}-Y_{\overline{a}_k'})=E(Y_{\overline{a}_k})-E(Y_{\overline{a}_k'})=P(Y_{\overline{a}_k}=1)-P(Y_{\overline{a}_k'}=1), \tag{1}$$

where $P(Y_{\overline{a}_k}=1)$ is the probability of mortality in wave k given trajectory $\overline{a}_k$, and $P(Y_{\overline{a}'_k}=1)$ is the analogous probability given the alternative trajectory $\overline{a}_k'$.

Thus, we follow Wodtke et al. (2011) and South and Crowder (2010) in taking a parsimonious specification of duration-weighted exposure by averaging ordinal wave-specific treatments up to wave k:

$$logit\Big(P(Y_{\overline{a}_k=K}=1)\Big)=\theta_0+\theta_1(\textstyle\sum_{k=1}^{K}a_k/K), \tag{2}$$

where K indicates the number of follow-ups in which the exposure is observed for a given person-time observation. To estimate recent effects of exposure, we consider

$$logit\Big(P(Y_{\overline{a}_{k=K}}=1)\Big)=\theta_0+\theta_1 a_K, \tag{3}$$

where the probability of mortality at wave $k=K$ is only a function of most recent exposure. These equations are *marginal structural models.*

Note that the effects of exposure are identifiable only if we assume that at each wave, exposure is random (independent from potential outcomes) given observed covariates and exposure histories (the ignorability assumption):

$$Y_{\overline{a}_k}\perp A_k\mid \overline{L}_k, \overline{A}_{k-1}, \tag{4}$$

where $\overline{L}_k$ represents observed covariate history up to wave k. Let $A_k\in\{1,2, \ldots, 5\}$ encode the history of exposure status at the kth wave since start of follow-up, such that $A_k=1$ denotes residence in the first quintile, or tracts with the most homogeneous family income distributions, and $A_k=5$ denotes residence in the fifth quintile, or the most unequal tracts. $\overline{A}_{k-1}$ encodes exposure history up to wave k–1.

To estimate the effects of the exposure, the conventional regression-adjusted approach involves fitting a discrete-time logit model of the form

$$logit\Big(P(Y_K=1\mid \overline{Y}_{k-1}=0, \overline{A}_k, \overline{L}_k)\Big)=\alpha_0(k)+u(\overline{A}_k)+\epsilon(\overline{L}_k), \tag{5}$$

where $\alpha_0(k)$ are wave-specific intercept terms, $u(\overline{A}_k)$ are parameterizations of exposure histories, and $\epsilon(\overline{L}_k)$ are parameterizations of confounder histories. In our analyses, we simply consider most recent exposure and confounder values in estimating regression-adjusted estimates of inequality. However, given the known limitations of these approaches, we also weight by IPT to estimate the marginal effects of exposure to inequality.

When using IPT-weighted estimators, treatment at each wave is independent to prior confounders. Conditioning on covariate history, therefore, is no longer necessary because the weights achieve randomization (an unadjusted regression model

that excludes time-varying covariates can be fit to estimate marginal effects). MSM coefficients can be interpreted as the implications of exposure given that the sequence of exposure is no longer related to other time-varying covariates. It is important to clarify that MSMs with IPT weights make the same assumptions on no confounding as regression techniques. However, MSMs relax additional unrealistic assumptions about time-varying covariates made by regressions. As a separate issue, we correct for potential nonrandom attrition using weights. All analyses use stabilized weights analogous to those derived for selection into treatment but adjusting for differential probability of remaining in the study through the end of follow-up (Robins et al. 2000). For more information on the construction of IPT and attrition weights, please refer to the technical online appendix. Finally, when we report results of MSMs, we use standard errors estimated from 1,000 bootstrap samples.

There are several possible outcomes for these analyses. First, if neither regression-adjusted nor MSM estimates on any exposure are significant, then there is no evidence that local inequality matters net of individual and local characteristics (supporting Hypothesis 1A). Second, if MSM and regression-adjusted estimates on recent exposure are significant, this provides the strongest evidence that local inequality can directly increase mortality risk, because the inequality–mortality link is *not* necessarily driven in part by indirect effects and selection on time-varying covariates (supporting Hypothesis 1C). Third, if the MSM estimates on duration-weighted exposures are also significant (or if only MSM estimates are significant), time-varying covariates are likely both confounders and mediators (Wodtke 2013; Wodtke et al. 2011), and indirect effects are an essential part of the story (supporting Hypothesis 1B).

Results

Descriptive statistics for covariates are given separately by birth cohort in Table 1. Individuals were followed from baseline (the wave in which they were approximately 45 years old) until death, attrition, or 1997. This means that individuals born in the 1920s were followed for an average of 20.25 years and up to 27 years (between the 1970 and 1997 waves of the PSID); individuals born in the 1930s were followed on average for 15.07 years (and up to 22 years between 1975 and 1997 waves); and individuals born in the 1940s (or up to 1952) were followed on average for 4.89 years (and up to 12 years between the 1985 and 1997 waves). The data capture 115, 77, and 21 cases of mortality among individuals born in the 1920s ($n=894$), the 1930s ($n=1{,}123$), and the 1940s ($n=2{,}757$), respectively. The average ages at death were 72.91, 66.94, and 57.39 years, respectively. Naturally, the more recent cohort had not had an opportunity to experience mortality in the older ages. Birth cohorts were similar in gender composition, though later cohorts had a greater proportion of Whites and higher educational attainment, and were more likely to live in the South. All later analyses adjust for differences in baseline covariates.

Time-varying variables (income, employment, and neighborhood characteristics) were reported over person-year observations. Individuals in later cohorts were more likely to be in the workforce, typically had higher family incomes, and were more likely to live in more advantaged neighborhoods with higher populations. The racial composition of neighborhoods appeared similar across cohort subsamples.

Table 1 Sample characteristics by birth cohort, Panel Study of Income Dynamics and Neighborhood Change Database

Covariate	Birth Cohort			Full Sample
	1920–1930	1930–1940	1940–1952	
Baseline Covariates				
Educational attainment				
Less than high school	43.39	37.78	17.82	25.21
High school	37.19	37.31	36.23	36.60
More than high school	19.42	24.91	45.95	38.19
Gender				
Male	44.85	42.21	48.31	46.23
Female	55.15	57.79	51.69	53.77
Race				
Black	31.43	37.43	27.17	30.33
White	68.57	62.57	72.83	69.67
Southern	32.92	36.41	38.26	37.05
Time-Varying Covariates				
Log family income	10.78	10.84	11.01	10.87
Employment				
Employed	60.79	66.12	78.66	67.53
Unemployed	6.93	7.63	6.97	7.18
Not in workforce	32.28	26.25	14.37	25.29
Neighborhood population	3,709	3,906	4,134	3,897
Neighborhood log family income	10.97	10.99	11.14	11.03
Neighborhood proportion Black	0.21	0.24	0.20	0.22
Mortality				
Number of follow-ups	20.25	15.07	4.89	10.16
Number of observed deaths	115	77	21	213
Age-at-death	72.91	66.94	57.39	67.46
N	894	1,123	2,757	4,774

Sources: 1970–1997 annual waves of the Panel Study of Income Dynamics and the Neighborhood Change Database.

Inequality Exposure Patterns

Although tracts are delineated to be relatively socioeconomically homogeneous, there remains substantial tract-level inequality, as well as variation in such inequality. Gini coefficients averaged 0.38 with a standard deviation of 0.06 (and the distribution over all person-wave observations was approximately normal).

This study estimates the effects of exposure to inequality using an ordinal quintile variable, where the first quintile corresponds to the least unequal neighborhoods, and the fifth quintile corresponds to the most unequal neighborhoods. The ranges of Gini coefficients that fall into each ordinal bin are presented in Table 2.

The cutoffs for the ranges are calculated using all person-wave observations, where coefficients of 0 and 1 indicate minimum and maximum possible inequality. Note that Table 2 contradicts the idea that neighborhoods are too homogeneous for there to be a detectable effect on health or mortality, which has sometimes been suggested (Franzini et al. 2001; Subramanian and Kawachi 2004; Wilkinson and

Table 2 Neighborhood inequality indices

Inequality Exposure	Quintile Range
Quintile	
First (least unequal)	0.07–0.32
Second	0.32–0.35
Third	0.35–0.39
Fourth	0.39–0.43
Fifth (most unequal)	0.43–0.60
Number of Observations	4,744

Note: Data show approximately 48,510 person-wave observations on the individuals observed in the analytic sample. Inequality exposures are in terms of Gini coefficients, calculated at the tract level.

Pickett 2006). Income inequality in the United States, measured by the Gini coefficient, ranged between 0.43 and 0.60 in the most unequal neighborhoods. The latter is similar to levels of income inequality in the most unequal U.S. states.[18]

Exposure Weights

Stabilized IPT weights adjust for the dynamic and recursive relationship between neighborhood inequality exposure, where the exposure both influences and is influenced by time-varying covariates. We also compute stabilized attrition weights to adjust for nonrandom loss to follow-up. Weights are truncated at the first and 99th percentiles to improve efficiency and avoid disproportionate influence from outlying observations (Cole and Hernán 2008; Sharkey and Elwert 2011). Table 3 shows descriptive statistics for the stabilized IPT, attrition, and final weights used in the main analyses. The weights presented are well-behaved as they are centered around 1 and exhibit small variances. The attrition weights exhibit similar desirable properties.

Inequality Effects Estimates

Table 4 shows unadjusted, conventional regression-adjusted, and stabilized IPT-weighted estimates for the effect of recent exposure to different neighborhood inequality contexts on the probability of mortality (coefficients on other variables are presented in Table A-1, online appendix). Unadjusted models are estimated using a logistic regression predicting mortality while including only baseline covariates as controls. We observe an association between neighborhood inequality and mortality in unadjusted models, but these could be driven by time-varying confounders. Regression-adjusted models are estimated using logistic regressions predicting mortality conditioning on baseline covariates and time-varying covariates. These models indicate that exposure to local inequality contributes to mortality, controlling for

[18] Income inequality ranges from 0.48 to 0.52 in the 10 most unequal states (U.S. Census Bureau 2017).

Table 3 Stabilized treatment and attrition weights

Weight	Mean	SD	Percentile			
			1st	25th	75th	99th
Stabilized Treatment Weight (SW)	1.00	0.05	0.64	0.99	1.01	1.69
Stabilized Attrition Weight (CW)	1.00	0.05	0.76	1.00	1.00	1.26
SW × CW	0.99	0.07	0.56	0.99	1.01	1.58

Note: Descriptive statistics are for stabilized IPT, attrition, and final weights, calculated over $n = 45{,}174$ person-wave observations on 4,459 individuals present in the 1970–1997 annual waves of the PSID.

time-varying covariates. Finally, we estimate the effects of exposure to neighborhood inequality using marginal structural models with inverse probability of treatment and attrition weights. The coefficients in Table 4 are the increase in the log-odds of mortality.

Looking at the IPT-weighted results, the odds of mortality in any wave increase by about $\exp(0.71) = 2.03$ times for individuals who are exposed to the second quintile of neighborhood inequality relative to the first quintile. The odds of mortality increase by approximately $\exp(0.67) = 1.95$ times for individuals who are exposed to the fifth quintile relative to the first quintile. There are also effects of exposure to the fourth quintile relative to the first quintile—an increase in log-odds of mortality of 0.75. The effects of inequality on mortality risk appear to be nonmonotonic, and we discuss this pattern further in the Discussion.

All three models in Table 4 show significant effects of neighborhood inequality. Thus, Hypothesis 1A (a null effect) is unsupported by these analyses. In addition, results of regression-adjusted and MSM estimates on recent exposure support Hypothesis 1C (of direct effects of recent inequality on mortality), which are robust to selection on time-varying covariates such as absolute socioeconomic status (SES). We did not find support for Hypothesis 1B (of indirect effects that are mediated by observed time-varying covariates that are also confounders). The analyses on the duration-weighted exposures, which had very little effect on mortality, are shown in Table 5.

Coefficients on the control covariates (see Table A-1, online appendix) show that birth cohort, gender, education, and employment affect mortality risk. Unsurprisingly, being female, being in a more recent cohort, having at least a high school education, and being in the workforce are protective against mortality. Race was unpredictive of mortality net of the other covariates.[19] Outside of local inequality, we discovered that neighborhood characteristics, such as average family income, the proportion of Black residents, and geographic area (the indicator for living in a Southern state), did not have an association with mortality. Given the strong role of the impact of absolute socioeconomic status, race, and ethnicity for mortality in neighborhood effects research, we further discuss the effects of race and SES for the inequality–mortality link in sensitivity analyses.

[19] When we removed the indicator for Southern state and the indicators for SES (i.e., family income, education, inequality), then being Black raises the risk of mortality.

Table 4 Logistic models of mortality by neighborhood inequality exposure, using three strategies of adjusting for selection

Gini	Unadjusted	Regression-Adjusted	IPT-Weighted
First	—	—	—
Second	0.83***	0.72**	0.71**
	(0.24)	(0.25)	(0.25)
Third	0.52	0.37	0.46
	(0.26)	(0.27)	(0.26)
Fourth	0.73**	0.51*	0.75**
	(0.25)	(0.26)	(0.26)
Fifth	0.60*	0.38	0.67*
	(0.26)	(0.27)	(0.27)

Notes: Effects are log-odds ratios of mortality risk. Positive coefficients indicate increased risk. All models adjust for baseline covariates: birth cohort, race, gender, southern region, and educational attainment. Time-varying covariates are employment, family income, neighborhood average family income, neighborhood population size, and neighborhood proportion Black. The first model does not adjust for time-varying covariates; the second model includes time-varying covariates as regression controls; and the third model uses time-varying covariates to inform IPT weights.

*$p < .05$; **$p < .01$; ***$p < .001$ (for two-sided tests of no effect)

Sensitivity Analysis

The foregoing results were robust to various specifications of the functional forms of the regressions used to estimate the inverse probability of treatment weights (see Eq. (6) in the technical online appendix). Models with nonlinear transformations and second-order terms on continuous variables, different groupings of categorical variables, and interactions led to similar results. Since these permutations did not substantively alter the results, we report models using lowest-order terms and omit interactions for interpretability and parsimony. As an additional robustness check, we use generalized boosted models to estimate IPT weights. We provide the results of this sensitivity analysis in Table A-2 (online appendix).

This strategy led to results that were substantively similar to the main results, which used logistic regression, in which the second, fourth, and fifth inequality quintiles predict higher risk of mortality compared with the lowest inequality quintile. Further analyses are necessary to understand this nonmonotonic pattern. We speculate that if relative deprivation operates more strongly at lower levels of income inequality, while social corrosion and mechanisms related to violence, accidents, or physical hazards operate more strongly at higher levels of income inequality, this could produce nonmonotonicity. Thus, we suggest that future research should further interrogate the role of social corrosion and/or social comparison mechanisms. Our contribution is to show that income inequality has an effect on mortality that is not driven by mediation through observed mortality risks, such as measures of absolute SES, and that is robust to various methods of estimating the IPT weights.

To assess the sensitivity of analyses to our choice of analytic subsample, we conduct several additional analyses to consider the effects of absolute SES, race, and age on the relationship between recent inequality and mortality. First, we conduct

Table 5 Logistic models of mortality by duration-weighted neighborhood inequality exposure, using three strategies of adjusting for selection

Gini	Unadjusted	Regression-Adjusted	IPT-Weighted
First	—	—	—
Second	0.04	–0.13	–0.01
	(0.22)	(0.23)	(0.23)
Third	0.41	0.24	0.40
	(0.21)	(0.22)	(0.22)
Fourth	0.09	–0.16	0.17
	(0.24)	(0.25)	(0.24)
Fifth	–0.26	–0.45	–0.20
	(0.28)	(0.29)	(0.28)

Notes: Effects are log-odds ratios of mortality risk. Positive coefficients indicate increased risk. All models adjust for baseline covariates: birth cohort, race, gender, southern region, and educational attainment. Time-varying covariates are employment, family income, neighborhood average family income, neighborhood population size, and neighborhood proportion Black. The first model does not adjust for time-varying covariates; the second model includes time-varying covariates as regression controls; and the third model uses time-varying covariates to inform IPT weights.

analyses of recent inequality exposure separately for those who are above and below average at baseline when comparing to their local SES. Figure A-1 (online appendix) shows the predicted probability of mortality (varying SES and inequality while holding other covariates at their mean or modal categories). There remains a significant negative effect of income inequality on mortality regardless of absolute SES. We recommend that future studies consider the ways in which income inequality has an effect on mortality that goes beyond relative deprivation arguments (i.e., through social corrosion).

When repeating the analyses separately for White and for Black respondents, we find patterns in the relationship between recent income inequality and mortality for White respondents that are similar to those for the sample as a whole. The effects of inequality were not statistically significant for Black respondents (Table A-3, online appendix). We speculate that this could be due to a truly weaker connection between local income inequality exposure and mortality among Black respondents, more variation in income inequality among White respondents, different covariance between SES and income inequality for Blacks versus Whites, or the smaller sample of Black respondents. Future research should investigate these possibilities given the mixed evidence in the literature on whether the effects of neighborhood conditions on health depend on race (Noah et al. 2018) or are independent of race (Subramanian and Kawachi 2006). Such investigation likely requires data other than that from the PSID,[20] however, we can say definitively that our findings are *not* driven by Black respondents.

We also repeat the analyses for an older sample. When following individuals from at least 65 years old until death, attrition, or 1997, we do not obtain the same results. No

[20] Because of the relatively small numbers of mortality captured by the PSID, we are cautious about interpreting results from stratified analyses.

coefficient on inequality has a significant impact on mortality. It seems that compared to the health and mortality of older adults, the health and mortality of working-age and middle-aged adults are more sensitive to inequality and socioeconomic circumstances (Backlund et al. 2007; Lynch et al. 2004), as well as place-effects (Geronimus et al. 2014).

Lastly, we repeat our analyses but measure income inequality at the state level rather than the tract level (Table A-4, online appendix).[21] Consistent with the literature, we observe larger effects of income inequality at the state level than we had previously observed at the tract level. However, while the mechanisms of social corrosion and relative deprivation have previously been proposed at the state level, we theorize that at least one of these mechanisms might also apply at the local level, and we find more direct evidence of inequality effects such as these (i.e., effects of recent exposure that are not mediated by the observed time-varying covariates) at the tract level than at the state level. In sum, our sensitivity analyses show that the strong focus on larger levels of aggregation in the literature has led to an incomplete picture on the effects of inequality.

Discussion

We apply an individual-level and longitudinal framework to determine whether local income inequality has causal effects on mortality, combining individual-level data from the PSID with information on tract-level income inequality over a period of 30 years, and applying a MSM approach that pseudorandomizes exposure trajectories to inequality relative to time-varying covariates. We show that recent exposure to neighborhood-level inequality predicts higher mortality risk, and is robust to family-level income, mean neighborhood-level income, and other time-varying covariates. In general, our study supports previous studies (Backlund et al. 2007; Hildebrand and Van Kerm 2009; Kaplan et al. 1996; Kawachi et al. 1997) in finding a significant association between income inequality and health, and contradicts other studies (Beckfield 2004; Deaton 2001; Kravdal 2008; Mellor and Milyo 2001), especially reports of a null or small effect within local areas (Blakely et al. 2003; Clough-Gorr et al. 2015; Fiscella and Franks 1997; Franzini et al. 2001; Osler et al. 2002; Shibuya et al. 2002; Subramanian and Kawachi 2004; Veenstra 2002; Wilkinson and Pickett 2006).

Our study is the first to look at the effects of local income inequality on mortality using individual-level data over a long period. In general, there is surprisingly limited evidence to suggest that income inequality shapes health (Truesdale and Jencks 2016). Yet this may be due to the scope of analyses for studies of local inequality and the use of aggregate data, which raise concerns regarding statistical assumptions on the exchangeability of conditions (Hernán 2012), especially when the distribution of risk factors can differ between the populations affected. The few studies that used both individual-level and longitudinal data did not properly account for dynamic relationships between local context and risk factors for health and mortality; traditional strategies for accounting for time-varying covariates remove potential indirect causal

[21] The analyses in Table A-4 (online appendix) do not include tract-level inequality.

pathways (Wodtke et al. 2011). In using MSMs to make individuals more exchangeable (by generating a counterfactual that strengthens inference), we take seriously the possibility of both direct and indirect effects of local inequality for mortality risk. However, our results show that dynamic selection on observed time-varying covariates is unlikely to play a large role, but that instead there may a direct effect of recent inequality on mortality.

Our findings contradict the idea that neighborhoods are too homogeneous for there to be an effect on health or mortality (Franzini et al. 2001; Subramanian and Kawachi 2004; Wilkinson and Pickett 2006). As the data from the NCDB reveal, the Gini coefficient of income inequality ranges between 0.43 and 0.60 in the most unequal neighborhoods, meaning that local income inequality in the United States can be as high as state-level income inequality (U.S. Census Bureau 2017). While our sensitivity analyses show that state-level inequality has a strong effect on inequality, our main analyses show that there is a significant effect of recent exposure to tract-level inequality on mortality, above and beyond state-level inequality,[22] and net our observed time-varying covariates.

The contributions of our study should be assessed in the context of its limitations. Because of data restrictions, we can only consider exposure to inequality after the age of 45; although we saw little evidence of indirect or cumulative effects after this age, cumulative inequality may have stronger implications prior to adulthood. When the data are available, future research should consider whether there are particular life stages or subpopulations for which sustained exposure is more harmful. In addition, while our data do not provide enough cases of mortality to separate analyses by cause of mortality, we call for future work to disentangle mechanisms by analyzing cause-specific mortality data (Miech et al. 2011). Such mortality data could help disentangle stress mechanisms from mechanisms relating to hazards within communities. In our sensitivity analyses, we found a possible nonmonotonic effect of inequality on mortality—and cause-specific mortality data could also help get at the reasons for this pattern. Finally, while our study may not be generalizable outside the United States, our analytical approach can be applied anywhere that local-area estimates of inequality can be used together with longitudinal data on the mortality of individuals. ■

Acknowledgments The authors express their gratitude to Geoffrey T. Wodtke for his generous help with the application of the marginal structural models. The authors also thank the participants of the "Contextual Factors and Inequalities in Health and Mortality" session at the 2017 annual meeting of the Population Association of America, as well as the participants of the "Health and Mortality" session at the American Sociological Association annual conference in 2018, for their very helpful comments and suggestions. The authors are grateful for research funding from the Center for Population and Development Studies at Harvard University.

[22] Local-level inequality is far from a proxy for state-level inequality. In our main analyses, we do not account for state-level inequality because it occurs almost entirely between tracts rather than within tracts. The correlation between state-level and tract-level inequality is very low, and ANOVA analyses show that only around 7.6% of the variation in tract-level inequality comes from differences between states. For these reasons, even when we do account for state-level inequality in the analysis, the effects of local inequality remain significant.

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