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 (PSID) 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 (MSMs) to account for changes in exposure to local income inequality. In contrast to other studies finding no significant relation between income inequality and mortality, we find that recent exposure to higher local inequality increases the relative risk of mortality among individuals at ages 45 or above.

KEY WORDS: mortality, inequality, neighborhoods, life-course, marginal structural models

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INTRODUCTION

Income inequality in the United States (U.S.) 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, Bloome, and Percheski 2008) and the top percentile share of incomes doubled (Atkinson, Piketty, and Saez 2011). Among the potential sequelae of growing inequality is increased mortality risk. As income inequality has risen in the U.S., mortality rates have risen among some groups, and the U.S. trend in life expectancy has diverged from life expectancy trends in other rich 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, Van Lenthe, and Mackenbach 2015; Kawachi et al. 1997; Wilkinson and Pickett 2006), relatively little research considers the consequences of income inequality at a local 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, Emmelin, and Stafström 2018; Wilkinson 1992; Zheng 2012), states (Backlund et al. 2007; Deaton 2001; Kennedy et al. 1998; McLeod, Nonnemaker, and Call 2004; Rasella, Aquino, and Barreto 2013), metropolitan areas (Deaton and Lubotsky 2003; Mellor and Milyo 2001), or counties (Fiscella and Franks 1997; Franzini, Ribble, and Spears 2001; LeClere and Soobader 2000; Yang, Matthews, and Park 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 context 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 due to 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 U.S. (Blakely, Atkinson, and O'Dea 2003; Clough-Gorr, Egger, and Spoerri 2015; Osler et al. 2002; Shibuya, Hashimoto, and Yano 2002; Veenstra 2002). In addition, studies of the implications of local inequality for mortality often rely on ecological data (Brodish, Massing, and Tyroler 2000; Massing et al. 2004; McLaughlin and Stokes 2002; McLaughlin, Stokes, and Nonoyama 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, Wilson, and Vasdev 2001; Subramanian and Kawachi 2006; Vincens et al. 2018), which can overlook causal mediators and over-control for the dynamic and recursive relationship between the local inequality and other time-varying covariates (Wodtke, Harding, and Elwert 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 information on mortality included in the PSID are rarely used, and to our knowledge has 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 pseudo-randomized 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 may increase 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).[2] In this section, we review existing studies that shed light on these three possibilities.

Although some scholars do not observe 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, and states). In a meta-analysis of 168 existing studies, Wilkinson and Pickett (2006) show 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, Blakely, and Kawachi 2003; Subramanian and Kawachi 2004, 2006). In contrast, fewer studies have considered whether income inequality within

[2] "Direct effects" refer to recent effects that are not mediated by our observed time-varying mediators – we theorize direct effects as a more essential part of inequality. In contrast, "indirect effects" go 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, it can be treated as a mediator as well. However, for theoretical reasons, we believe that social comparisons are closer to a direct effect – studies have not established an independent effect of social comparisons on mortality outside of studies of inequality, compared to numerous studies on the effects of our observed set of potential mediators (e.g., family and neighborhood inequality).

neighborhoods or *small* local areas (e.g., tracts, communities, parishes) matters for population health.

The few studies that consider small local areas have found little evidence that population health is 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 is most likely a result of Denmark's strong welfare system (Osler et al. 2002). Similarly, large-scale cross-sectional data from New Zealand reveal no association between inequality in census sub-regions and mortality (Blakely et al. 2003). Income inequality within coastal communities in British Columbia is also unassociated with mortality within those communities (Veenstra 2002). There is a only a weak cross-sectional association between income inequality and mortality at the prefecture level in Japan (Shibuya et al. 2002). A study of inequality in Swiss municipalities finds that inequality is actually associated with lower mortality (Clough-Gorr et al. 2015). Lastly, a longitudinal study within the U.S. context finds 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 to 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).[3] Taken together, existing studies point to the following null hypothesis: *income inequality in U.S. census tracts*[4] *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).[5] Estimating individual-level effects using solely aggregate-level data is also problematic when the distribution of epidemiological risk factors is profoundly different between populations affected (Hernán 2012) or when treatment effects are heterogeneous (Xie 2013). Since most studies of the inequality-health link use aggregate data (Macintyre, Ellaway, and Cummins 2002; Neckerman and Torche 2007; Truesdale and Jencks 2016), it remains unknown whether neighborhood income inequality is causally related to 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

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

[4] 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.

[5] 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 non-linear (an example of the "ecological fallacy").

area by failing to attract businesses or offering fewer relevant services, and socioeconomic advantage 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, Bound, and Ro 2014), such as inequality-effects. However, the potential for indirect effects should be analyzed with care because they could in theory also be confounders (for example 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 over-controlled. 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 over-control for indirect effects and may lead to bias (Wodtke et al. 2011).[6] We use Marginal Structural Models (MSM) to pseudo-randomize sequences of exposure to inequality relative to other time-varying covariates as well as regression-adjusted models.[7] 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 socio-economic position are believed to increase stress levels and chronic inflammation (Layte et al. 2019) and has 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, Adler, and Marmot 2003; Tang et al. 2016). Proponents of the status comparisons hypothesis generally assume that the latter explain the link between societal inequality, e.g. in terms of incomes, and health (Wilkinson and Pickett 2006, 2011). In line with this hypothesis, studies indicate 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 unfold, 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 2011). While some understandings of "class" are relative to wider society, stress due to comparing and being compared are also likely to occur in local areas, meaning that *local* inequality could theoretically increase mortality risk.

[6] The association between intra-generational 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.

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

Second, inequality may be socially corrosive. Social corrosion refers to the erosion of relations or expectations within groups as well as social and collective resources (Kawachi, Kennedy, and Wilkinson 1999; Neckerman and Torche 2007). Inequality could be socially corrosive in local context 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 (CPR) in out-of-hospital cardiac arrests (Iwashyna, Christakis, and Becker 1999) or in engagement 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 2011), 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, Baumer, and Rosenfeld 2004; Ross, Mirowsky, and Pribesh 2001; Wilkinson and Pickett 2011). 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 (Hypotheses 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).[8]

DATA AND METHODS

This study considers the consequences of inequality for mortality among respondents of the Panel Study of Income Dynamics (PSID) using data from 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 Panel Study of Income Dynamics (PSID) and the GeoLytics

[8] Evidence to support dynamic selection would suggest 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 provide both as intuition for why direct effects might exist.

Neighborhood Change Database (NCDB) (GeoLytics 2003). The PSID was first conducted in 1968 on a national sample of around 4,800 families.[9] These families, together with new families formed by sample members over time, were interviewed annually between 1968-1997 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 mid-life is rare. Thus, we follow the 4,774 individuals captured by the PSID from the point at which they are age 45 or older, until they die, are lost to follow up, or reach administrative end of follow-up (1997 wave of the PSID).

Individuals are captured by roughly 10 follow-ups before mortality, attrition, or end of follow-up.[10] 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 217 deaths are recovered from the restricted PSID mortality files. The mortality file contains, using survey responses and death certificate data, information on deaths of all individuals that 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

Measurement of neighborhood inequality, the exposure of interest, as well as other spatial covariates used in the study are derived from the Neighborhood Change Database (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.[11] 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 ten times the median income,[12] a conventional method of top-coding 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)

[9] 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 control for the design variables—in this case, family income – on which oversampling occurs (Wodtke, Harding, and Elwert 2016).

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

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

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

distribution and calculate Gini coefficients for each tract in each census year.[13] The Gini coefficient is a measure of statistical dispersion that represents the income 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.[14] We then divide census tracts into quintiles[15] based on 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 concerns inequality exposure in adulthood. While the long-term effects of local context during childhood or adolescence are likely to be important due to 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 1940[16]); and we expect recent birth cohorts have lower mortality than older birth cohorts (Masters, Hummer, and Powers 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 nonblack 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, Finch, and Crimmins 2015). Educational attainment is categorized by "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, South West 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 work force (no longer searching, retired, etc.). 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

[13] 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.
[14] The 5-year 2006-2010 Gini index for the U.S. 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 homogenous and given recent increases in inequality, the derived distribution of tract-level Gini indices in this paper are reasonable.
[15] 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.
[16] Results are robust to whether a handful of individuals born in 1951 and 1952 are included in the 1940s cohort or given their own category.

head, partner and other family members earned over the past year, reported in terms of 2010 dollars using the Consumer Price Index (CPI-U).[17]

Time-varying spatial covariates from census data are also included since neighborhood inequality is likely correlated with other contextual factors that could drive mortality (Huie, Hummer, and Rogers 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 non-metropolitan areas (McLaughlin et al. 2001). Dummy categories account for any item-specific nonresponse, but results are similar when we simply drop missing observations since nonresponse is rare.

Estimation

Marginal structural models (MSMs) are a class of models[18] 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 probability of treatment weighted (IPT) weighted estimators. IPT weighting is a statistical 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 pseudo-population, 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, e.g. the effect of neighborhood disadvantage on high school completion (Wodtke et al. 2011), but have 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, Morenoff, and Gannon-Rowley 2002) and (2) introduces possible bias in the estimation of effects of exposure confounders (Greenland 2003; Pearl 2011). MSM estimation avoids these issues by reweighting observations based on the distribution of exposures among different covariate levels rather than 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 $\bar{a}_k = (a_1,...,a_k)$, where $\bar{a}_k$ 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 $Y_{\bar{a}_k}$ be a binary indicator for

[17] Results are not sensitive to the use of 2010 dollars versus raw dollars unadjusted for inflation.
[18] 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. 1999; Wodtke et al. 2011).

mortality at wave *k* given survival up to wave *k* and a (either observed or counterfactual) exposure trajectory. We wish to estimate the effect of one exposure trajectory $\bar{a}_k$ compared to another possible trajectory $\bar{a}'_k$, given by,

$$E(Y_{\bar{a}_k} - Y_{\bar{a}'_k}) = E(Y_{\bar{a}_k}) - E(Y_{\bar{a}'_k}) = \mathrm{P}(Y_{\bar{a}_k} = 1) - \mathrm{P}(Y_{\bar{a}'_k} = 1) \quad (1)$$

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

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*:

$$logit\left(\mathrm{P}\left(Y_{\bar{a}_{k=K}} = 1\right)\right) = \theta_0 + \theta_1\left(\sum_{k=1}^{K} \mathrm{a}_k / K\right) \quad (2)$$

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:

$$logit\left(\mathrm{P}\left(Y_{\bar{a}_{k=K}} = 1\right)\right) = \theta_0 + \theta_1 a_K \quad (3)$$

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 (ignorability assumption):

$$Y_{\bar{a}_k} \perp A_k \mid \bar{L}_k, \bar{A}_{k-1} \quad (4)$$

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

$$logit\left(\mathrm{P}(Y_K = 1 \mid \bar{\mathrm{Y}}_{k-1} = 0,\ \bar{\mathrm{A}}_k,\ \bar{\mathrm{L}}_k)\right) = \alpha_0(k) + u(\bar{\mathrm{A}}_k) + \epsilon(\bar{\mathrm{L}}_k) \quad (5)$$

where $\alpha_0(k)$ are wave-specific intercept terms, $u(\bar{\mathrm{A}}_k)$ are parametrization of exposure histories, and $\epsilon(\bar{\mathrm{L}}_k)$ 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 Inverse Probability Treatment (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 constructing of IPT and attrition weights, please refer to the technical 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 are followed from baseline (approximately 45 years old) and until death, attrition, or 1997. This means that individuals born in the 1920s are followed for around 20.25 years and up to 27 years (between the 1970 and the 1997 waves of the PSID). Individuals born in the 1930s are followed on average for 15.07 years and up to 17 years (between the 1975 and 1997 waves of the PSID). Individuals born later than the 1940s are followed for around 4.89 years and up to 7 years. The data capture 115, 77, and 21 cases of mortality among the individuals born in the 1920s (N = 894), the 1930s (N = 1,123), and the 1940s (N = 2,757), respectively. The average ages at death among those who died are 72.91, 66.94 and 57.39, respectively. Naturally, the more recent cohort has not had an opportunity to experience mortality in older ages yet. Birth cohorts are similar in gender composition, though later birth cohorts have a greater proportion White, higher educational attainment, and are more likely to live in the South. All later analyses adjust for differences in baseline covariates.

[Table 1 About Here]

Time-varying variables (income, employment, and neighborhood characteristics) are reported over person-year observations in Table 1. Individuals in later cohorts are more likely to be in the work force, typically have higher family incomes, and are more likely to live in more advantaged

neighborhoods with higher populations. The racial composition of neighborhoods appears similar across cohort subsamples.

Inequality Exposure Patterns

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

This study estimates the effects of exposure to inequality using an ordinal quintile variable, where the 1st quintile corresponds to the least and the 5th quintile corresponds to the most unequal neighborhoods. The range of Gini coefficients that fall into each ordinal bin is presented in the top rows of Table 2.

[Table 2 About Here]

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 homogenous 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 Pickett 2006). Income inequality in the U.S., measured by the Gini coefficient, ranges between 0.43 to 0.60 in the most unequal neighborhoods (see Table 2). The latter is equal to levels of income inequality in the most unequal U.S. states.[19]

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 1st 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.

[Table 3 about Here]

The weights presented in Table 3 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 presented in Table A-1). Unadjusted

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

models are estimated using a logistic regression predicting mortality 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 regression predicting mortality conditioning on baseline covariates and time-varying covariates. These models indicate that exposure to local inequality contributes to mortality, controlling for time-covariates. Finally, we estimate the effects of exposure to neighborhood inequality using marginal structural models with inverse probability of treatment and attrition weights. Coefficients in Table 4 are the increase in the log-odds of mortality.

[Table 4 about Here]

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 2nd quintile of neighborhood inequality relative to the 1st quintile. The odds of mortality increase by approximately exp(0.67) = 1.95 times for individuals who are exposed to the 5th quintile relative to the 1st quintile of neighborhood inequality. There are also effects of exposure to the 4th quintile relative to the 1st quintile – an increase in log odds of mortality of 0.75. The effects of inequality on mortality risk appear non-monotonic, and we discuss this pattern further in the discussion section.

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 here support Hypothesis 1C, of direct effects of recent inequality on mortality, which are robust to selection on time-varying covariates such as absolute 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.

[Table 5 about Here]

Coefficients on the control covariates (presented in Table A-1), show that birth cohort, gender, education, and employment affect mortality risk. Unsurprisingly, being female, in a more recent cohort, having at least a high school education, and being in the work force are protective against mortality. Race was unpredictive of mortality net of the other covariates.[20] Outside of local inequality, we discovered that neighborhood characteristics, such as average family income and proportion black, and geographic area (the indicator for Southern state), did not have an association with mortality. Given the strong role of the impact of absolute socioeconomic status, and 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.

Sensitivity Analysis

The above results robust to various specifications of the functional forms of the regressions used to estimate the inverse probability of treatment weights (see Equation 6 in the technical

[20] 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.

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 GBMs (generalized boosted models) to estimate IPT weights. We provide the results of this sensitivity analysis in Table A-2.

This strategy led to substantively similar results as the main results which used logistic regression, in which the 2nd, 4th, and 5th inequality quintile predicts higher risk of mortality compared to the lowest inequality quintile. Further analyses are necessary to understand this non-monotonic 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 non-monotonicity. 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 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 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 similar patterns in the relationship between recent income inequality and mortality for white respondents as for the sample as a whole. The effects of inequality were not statistically significant for black respondents. These results are shown in Table A-3 in the 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 vs. whites, or the smaller sample of black respondents. Future research should investigate these possibilities given the mixed evidence in existing literature on whether the effects of neighborhood conditions on health depend on race (Noah, Yang, and Wang 2018) or are independent from race (Subramanian and Kawachi 2006). Such investigation likely requires data other than the PSID,[21] however what we can say definitively here is that our findings are *not* driven by black respondents.

[21] We are cautious about interpreting results from stratified analyses due to the relatively small numbers of mortality captured by the PSID.

We also repeat the analyses but 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 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-aged 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 measuring income inequality at the state rather than tract level (Table A-4).[22] Consistent with existing 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 theorized 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 existing literature has led to an incomplete picture on the effects of inequality.

DISCUSSION

We apply an individual-level and longitudinal framework to determine whether there exist causal effects of local income inequality on mortality, combining individual-level data from the PSID with information on tract-level income inequality over a period of thirty years, and applying a MSM approach that pseudo-randomizes 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) finding a significant association between income inequality and health, and contradicts others (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 use of aggregate data, which raises concerns regarding statistical assumptions on the exchangeability of conditions (Hernán 2012), especially when the distribution of risk factors can differ between populations affected. The few existing studies that use both individual-level and longitudinal data do 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 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

[22] The analyses in Table A-4 do not include tract-level inequality.

selection on observed time-varying covariates is unlikely to play a large role, but that instead there may a direct effect of recent inequality of mortality.

Our findings contradict the idea that neighborhoods are too homogenous 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 to 0.60 in the most unequal neighborhoods, meaning that local income inequality in the U.S. 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,[23] and net our observed time-varying covariates.

The contributions of our study should be assessed in the context of its limitations. Due to data restrictions, we can only consider exposure to inequality after the age of 45 –although there was little evidence of indirect or cumulative effects after this age, cumulative inequality may be more harmful 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 does 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). Cause-specific mortality data could help disentangle stress mechanisms from mechanisms relating to hazards within communities. In our sensitivity analyses, we found a possible non-monotonic effect of inequality on mortality – cause-specific mortality data could also help get at the reasons for this pattern. Finally, while our study may not generalize 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 mortality of individuals.

[23] 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 state level inequality 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 is the analysis, the effects of local inequality remain significant.

TABLES AND FIGURES

TABLE 1: SAMPLE CHARACTERISTICS

Birth Cohort	1920 - 1930	1930 - 1940	1940 - 1952	Full Sample
Baseline Covariates				
Educational Attainment				
Less than HS	43.39	37.78	17.82	25.21
High School (HS)	37.19	37.31	36.23	36.60
More than HS	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	27.07	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 work-force	32.28	26.25	14.37	25.29
NBH Population	3,709	3,906	4,134	3,897
NBH Log Family Income	10.97	10.99	11.14	11.03
NBH Proportion Black	0.21	0.24	0.20	0.22
Mortality				
Num. Follow Ups	20.25	15.07	4.89	10.16
Num. Observed Deaths	115	77	21	217
Age at Death	72.91	66.94	57.39	67.46
N (Sample Size)	894	1,123	2,757	4,774

Source: Using 1970-1997 annual waves of the Panel Study of Income Dynamics

TABLE 2: NEIGHBORHOOD INEQUALITY INDICES

Inequality Exposure, Quintile Ranges	
1st (least unequal)	(0.07, 0.32)
2nd	(0.32, 0.35)
3rd	(0.35, 0.39)
4th	(0.39, 0.43)
5th (most unequal)	(0.43, 0.60)
Observations	N = 4,744

Note: Over 48,510 person-wave observations on the sample of individuals present in the 1970-1997 annual waves of the Panel Study of Income Dynamics. Inequality exposures are in terms of Gini-Coefficients, calculated at the tract level.

TABLE 3. STABILIZED TREATMENT AND ATTRITION WEIGHTS

			Percentiles			
	Mean	SD	1st	25th	75th	99th
Weights						
[1] Stabilized treatment weight (SW)	1.00	0.05	0.64	0.99	1.01	1.69
[2] Stabilized attrition weight (CW)	1.00	0.05	0.76	1.00	1.00	1.26
[3] SW X CW	0.99	0.07	0.56	0.99	1.01	1.58

Note: Descriptive statistics 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.

TABLE 4: LOGISTIC MODELS OF MORTALITY GIVEN NBH INEQUALITY EXPOSURE USING THREE STRATEGIES OF ADJUSTING FOR SELECTION

	Unadjusted	Regression Adjusted	IPT Weighted
Gini			
1st	--	--	--
2nd	0.83***	0.72**	0.71**
	(0.24)	(0.25)	(0.25)
3rd	0.52	0.37	0.46
	(0.26)	(0.27)	(0.26)
4th	0.73**	0.51*	0.75**
	(0.25)	(0.26)	(0.26)
5th	0.60*	0.38	0.67*
	(0.26)	(0.27)	(0.27)

Notes: (1) Effects are log odds ratios of mortality risk. Positive coefficients indicate increased risk. (2) All models adjust for baseline covariates: birth cohort, race, sex, southern region, and educational attainment. (3) Time-varying covariates are employment, family income, NBH average family income, NBH population size, NBH proportion black. Model 1 does not adjust for time varying covariates. Model 2 includes time varying covariates as regression controls. Model 3 use time-varying covariates to inform IPT weights (4) *p<0.05 **p<0.01 and ***p<0.001 for two-sided tests of no effect.

TABLE 5: LOGISTIC MODELS OF MORTALITY GIVEN DURATION-WEIGHTED NBH INEQUALITY EXPOSURE

	Unadjusted	Regression Adjusted	IPT Weighted
Gini			
1st	--	--	--
2nd	0.04	-0.13	-0.01
	(0.22)	(0.23)	(0.23)
3rd	0.41	0.24	0.40
	(0.21)	(0.22)	(0.22)
4th	0.09	-0.16	0.17
	(0.24)	(0.25)	(0.24)
5th	-0.26	-0.45	-0.20
	(0.28)	(0.29)	(0.28)

Notes: (1) Effects are log odds ratios of mortality risk. Positive coefficients indicate increased risk. (2) All models adjust for baseline covariates: birth cohort, race, sex, southern region, and educational attainment. (3) Time-varying covariates are employment, family income, NBH average family income, NBH population size, NBH proportion black. Model 1 does not adjust for time varying covariates. Model 2 includes time varying covariates as regression controls. Model 3 use time-varying covariates to inform IPT weights (4) *p<0.05 **p<0.01 and ***p<0.001 for two-sided tests of no effect.

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