Published in final edited form as:

Soc Sci Med. 2013 August ; 91: 58–66. doi:10.1016/j.socscimed.2013.03.003.

Investigating the relationship between neighborhood poverty and mortality risk: A marginal structural modeling approach

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Abstract

Extant observational studies generally support the existence of a link between neighborhood context and health. However, estimating the causal impact of neighborhood effects from observational data has proven to be a challenge. Omission of relevant factors may lead to overestimating the effects of neighborhoods on health while inclusion of time-varying confounders that may also be mediators (e.g., income, labor force status) may lead to underestimation. Using longitudinal data from the 1990 to 2007 years of the Panel Study of Income Dynamics, this study investigates the link between neighborhood poverty and overall mortality risk. A marginal structural modeling strategy is employed to appropriately adjust for simultaneous mediating and confounding factors. To address the issue of possible upward bias from the omission of key variables, sensitivity analysis to assess the robustness of results against unobserved confounding is conducted. We examine two continuous measures of neighborhood poverty – single-point and a running average. Both were specified as piece-wise linear splines with a knot at 20 percent. We found no evidence from the traditional naïve strategy that neighborhood context influences mortality risk. In contrast, for both the single-point and running average neighborhood poverty specifications, the marginal structural model estimates indicated a statistically significant increase in mortality risk with increasing neighborhood poverty above the 20 percent threshold. For example, below 20 percent neighborhood poverty, no association was found. However, after the 20 percent poverty threshold is reached, each 10 percentage point increase in running average neighborhood poverty was found to increase the odds for mortality by 89 percent [95% CI = 1.22, 2.91]. Sensitivity analysis indicated that estimates were moderately robust to omitted variable bias.

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Keywords

Marginal structural modeling; Neighborhood effects; Longitudinal analysis; Causal modeling; Sensitivity analysis

Introduction

Extant observational studies generally support the existence of a link between neighborhood context and health (Diez Roux & Mair, 2010; Kawachi & Berkman, 2003; Pickett & Pearl, 2001; Robert, 1999; Yen & Syme, 1999). Most of the existing literature has focused on neighborhood demographic or socioeconomic characteristics, particularly neighborhood poverty and disadvantage (Meijer, Rohl, Bloomfield, & Grittner, 2012; Riva, Gauvin, & Barnett, 2007; Robert, 1999). Other research has examined the social aspects of neighborhood environments (e.g., social capital, trust, and crime) in relation to health (Sampson, Morenoff, & Gannon-Rowley, 2002). More recently, attention has been paid to the built environment, such as housing conditions, ambient air quality, and urban form (Frank, Engelke, & Schmid, 2003). The various health outcomes that have been linked to neighborhood context include, among others, mortality (Leclerc, Rogers, & Peters, 1998; Sloggett & Joshi, 1998; Yen & Syme, 1999), infectious disease (Acevedo-Garcia, 2000, 2001), low birthweight (Morenoff, 2003; O'Campo, Xue, Wang, & Caughy, 1997; Roberts, 1997; Sastry & Hussey, 2003) cigarette smoking (Diez Roux, Merkin, Hannan, Jacobs, & Kiefe, 2003; Duncan, Jones, & Moon, 1999; Immo Kleinschmidt, 1995) and diet (Morland, Wing, & Roux, 2002). These associations between health and place generally remain statistically significant even after adjusting for various individual-level socioeconomic characteristics (c.f., Oreopoulos, 2003; Reijneveld & Schene, 1998; Andrew Sloggett & Joshi, 1994).

However, estimating the causal impact of neighborhood effects has proven to be a challenge. Since random assignment into different neighborhood context is cost-prohibitive and infeasible on a large scale, most neighborhood-effect studies have relied on observational data. And as with inferences from all observational studies, a constant concern with neighborhood effect estimates is that they are biased due to unobserved confounding. For example, if persons who are inherently less inclined to live a healthy lifestyle were also less likely to seek neighborhoods with the physical and social environment that supports physical activity and well-being, attributes that are negatively correlated to neighborhood disadvantage, then not accounting for this factor would lead to spurious neighborhood findings. Thus, estimates of significant neighborhood effects may be overestimating the true impact.

However, estimating neighborhood effects presents additional challenges because neighborhood exposure is dynamic. To the extent that neighborhood context varies across time, single-point measures which do not allow for any lagged or long-term effects of neighborhoods on health may also bias estimates. Cross-sectional analyses implicitly assume that either 1) neighborhood context throughout the life-course is relatively constant or 2) neighborhood context has an immediate impact on the health outcome(s) being investigated.

While cross-sectional neighborhood effect estimates are rarely explicitly interpreted as either a short-term or long-term exposure, evidence suggests that single-point estimates underestimate the long-term exposure to neighborhood context (Do, 2009). Additionally, an important consideration, which has only recently gained attention, is that factors such as income and employment status may themselves be affected by prior neighborhood conditions and are consequently confounders and mediators of neighborhood-health effects simultaneously. For example, exposure to poor socioeconomic environments during childhood and adolescence has been shown to detrimentally impact educational outcomes (Harding, 2003; Sampson, Sharkey, & Raudenbush, 2008; Wodtke, Harding, & Elwert, 2011). Given the strong linkage between educational attainment and adult health (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Cutler & Lleras-Muney, 2010; Kimbro, Bzostek, Goldman, & Rodriguez, 2008), adjusting for educational attainment prohibits recovering the indirect effect of neighborhoods on health via education, resulting in biased estimates of the total effect. Other similarly important potential mediators include marital status, income, and employment status, which, when collectively controlled for in neighborhood-health models, may result in non-trivial bias of neighborhood effects. Because these factors are in the causal pathway, a major concern in adjusting for these characteristics is the possible underestimation of the total effect. While it may be intuitive to interpret these estimates as the direct impact of neighborhoods that do not pass through these mediators, this is not the case. Models that include time-varying covariates that are simultaneous mediators and confounders produce biased estimates of the direct impact (Nandi, Glymour, Kawachi, & VanderWeele, 2012; Robins, 1999).

Hence, researchers have longed faced a conundrum in which omission of these factors leads to upwardly biased effects while inclusion likely leads to downwardly biased estimates. To avoid the criticism of egregiously overestimating the effects of neighborhoods, studies have taken the conservative approach and conventionally included these simultaneous mediators and confounders in regression adjustments. However, with longitudinal data, appropriate adjustments for the effects of time-dependent confounders (e.g., income) to recover causal estimates of time-dependent treatments (e.g., neighborhood poverty) can be achieved by using marginal structural modeling (MSM) (Robins, Hernan, & Brumback, 2000).

While the usage of MSM strategies is increasing in epidemiologic research (e.g., Bodnar, Davidian, Siega-Riz, & Tsatis, 2004; Cook, Cole, & Hennekens, 2002), published work that has applied MSM strategies to investigate the effects of neighborhoods is still sparse. The few studies that have examined neighborhood effects on health outcomes via an MSM approach have found exposure to neighborhood disadvantage to increase the level of alcohol consumption and propensity for binge drinking, self-rated poorer health and decrease injection cessation among drug users (Cerdeira, Diez-Roux, Tchetgen, Gordon-Larsen, & Kiefe, 2010; Glymour, Mujahid, Wu, White, & Tchetgen Tchetgen, 2010; Nandi et al., 2010). Within the broader scope of neighborhood-effect studies in general, several studies have used MSM to examine the effects of childhood exposure to neighborhood deprivation on educational outcomes (Harding, 2003; Sampson et al., 2008; Sharkey & Elwert, 2011; Wodtke et al., 2011). These studies found childhood exposure to neighborhood deprivation to adversely affect high school graduation rates, cognitive ability, and verbal ability.

This paper seeks to address the above-mentioned issues that may bias neighborhood effect estimates in both directions. Using longitudinal data, we address the issue of possible downward bias due to adjusting for mediating factors by employing a marginal structural modeling strategy to investigate the link between neighborhood poverty and mortality risk. It then compares conventional naïve estimates to those recovered from marginal structural modeling. To address the issue of possible upward bias due to omitted variables, we then conduct a sensitivity analysis to assess the robustness of results against unobserved confounding.

Methods

Estimating treatment effects from MSMs is a two-stage process. In stage 1, the treatment assignment (here neighborhood poverty) is modeled. Since we treat neighborhood poverty as a continuous measure, we must estimate each person's probability density of having his/her own treatment at each time point, rather than as probabilities associated with a binary variable. The probability densities are then used to derive stabilized inverse probability of treatment (IPT) weights for each person (Robins et al., 2000). The higher the probability density of an individual to receive the observed treatment, the lower the weight the individual is assigned. Conversely, the lower the probability density that the individual received the observed treatment (based on observed covariates), the larger the weight the individual is assigned. Intuitively, this can be interpreted as upweighting individuals whose neighborhood exposure is underrepresented compared to what would have been observed through random assignment, and downweighting individuals whose neighborhood exposure is overrepresented. This weighting scheme transforms the covariate distributions of the sample population so that the weighted covariate distributions of the group across treatment levels become comparable. Essentially, the process creates a pseudopopulation in which treatment and the time-dependent confounders are no longer associated.

In stage 2, the causal parameter in this pseudopopulation is recovered by estimating a weighted regression model (observed population weighted by the stabilized IPT weight) on the outcome of interest. Under conditions of conditional exchangeability (i.e., treated assignment is effectively randomized, conditional on past treatment history and confounder history), this process will yield an unbiased estimate of the causal parameter of interest. Thus, by accounting for confounding without including these confounders in the structural part of the model, the IPT weight avoids the problems of biased estimation that arise when time-dependent confounders are inappropriately adjusted for via stratification or traditional regression approaches. This allows separating the time-dependent covariates confounder adjustment – which we want to incorporate in our modeling approach – from the mediation adjustment, which we want to avoid when assessing the total impact (direct and indirect) of the treatment on the outcome (Joffe, Ten Have, Feldman, & Kimmel, 2004; Robins et al., 2000).

Borrowing the same notation as described in Fewell et al. (2004), the stabilized IPT weight is defined as:

$$SW(t) = \prod_{k=0}^t \frac{f\{A(k) | \bar{A}(k-1), L(0)\}}{f\{A(k) | \bar{A}(k-1), \bar{L}(k)\}}$$

where $f\{*\}$ represents the conditional probability density function, $A(k)$ represents the time-varying treatment at time k , $\bar{A}(k-1)$ represents the treatment history up to time $(k-1)$, $L(k)$ represents an array of time-dependent covariates up to time k that are possible mediators as well as confounders, and $L(0)$ represents an array of baseline covariates. The denominator can be interpreted as a subject's conditional probability density of receiving his or her own observed treatment history up to time t , given time-dependent covariates, and baseline values. The numerator can be interpreted as a subject's conditional probability density of receiving his or her own observed treatment history up to time t , given baseline values $L(0)$.

Data

We use data from the 1990 to 2007 years of the Panel Study of Income Dynamics (PSID) merged with the 1990 to 2009 National Death Index. Begun in 1968, the PSID is composed of families of the baseline sample as well their descendants and individuals who marry into the families. Information was collected annually from 1968 to 1997, and biennially thereafter. Because of its self-replenishing design, the PSID is a nationally representative sample of the non-immigrant U.S. population. The PSID collects an extensive list of repeated measures of socioeconomic information as well as the residential location of respondents at the time of each interview. Thus, the PSID provides a wealth of socioeconomic information at both the individual and neighborhood characteristics throughout the life-course.

We use data from 1990 and odd years from 1991 to 2007 for a total of nine waves of socioeconomic and neighborhood information. The analytical sample is restricted to approximately 6500 respondents who are non-Hispanic black or white, ages 18 and over at baseline, and observed in baseline year 1990 and the immediate follow-up years of 1991 and 1993 with no missing covariates. The resultant sample consists of approximately 35,000 person-year observations with a median number of 8 observations per person.

Our outcome of interest is mortality risk between 1993 and 2009, operationalized as a binary indicator of death between each interview period. By 2009, approximately 15 percent of our baseline sample had died.

Our exposure of interest is neighborhood poverty, defined as the proportion of residents below the federal poverty level in a neighborhood. We use census tracts as proxies for neighborhoods and levels of neighborhood poverty are derived from the 1990 and 2000 decennial censuses, and 2005–2009 American Community Survey (ACS) five-year estimates. The 2005–2009 ACS estimates were designated as the level for 2007, the midpoint of the ACS data. Values for inter-census and ACS years were derived from linear interpolation. Census tract boundaries were consistently defined per 2000 definitions. In 1993, the initial year for which exposure to neighborhood poverty is considered, the

analytical sample consisted of approximately 4000 tracts with an average of 3 persons per tract.

Information from year 1990 is designated as baseline values and information from year 1991 is used to estimate the probability density of residing in his/her observed neighborhood poverty level in 1993. Hence, our neighborhood exposure of interest begins in 1993 and our first observed death is after the 1993 survey interview. We examine two neighborhood poverty specifications: a single-point-in-time level of neighborhood poverty measured in the wave immediately prior to the wave at which the outcome is observed (i.e., neighborhood poverty ($t - 1$)) and a running average of neighborhood poverty from 1993 to ($t - 1$). The single-point specification assumes that only the neighborhood poverty level in the wave immediately prior to the observed outcome is associated with mortality risk. The running average neighborhood poverty specification considers the relationship between more long-term average conditions and mortality risk and allows for more lagged effects of neighborhood poverty. Based on preliminary exploratory analyses (i.e., IPT weighted cubic spline plots) and commonly used neighborhood threshold of 20% (Harding, 2003; Sharkey & Elwert, 2011; Wilson, 1987; Wodtke et al., 2011), neighborhood poverty is specified as a continuous piece-wise linear spline with a knot at 20%. This allows for the slopes to differ before and after the 20% neighborhood poverty level and offers a more flexible, yet parsimonious and easily interpretable, parameterization of the relationship between neighborhood poverty and mortality risk.

Baseline characteristics, as measured in 1990, included race, gender, education level, wealth, self-rated health, age, marital status, female-headed household, family income to poverty ratio, labor force status, and neighborhood poverty. Time-varying characteristics, as measured between 1991 and 2007, considered as potential confounders as well as mediators include a subset of the (time-varying) variables included at baseline: marital status, female-headed household, family income poverty ratio, and labor force status (While education is theoretically a time-varying measure and can be considered a potential mediator, the PSID did not collect information on education for all adult respondents at each survey year. In addition, the median age at baseline is 43 years and only 5% of respondents were below 25 years old. Hence, in our analytical sample, very little variation was observed; only 4% of individuals were observed to change their education level within our analytical timeframe.).

Analytical strategy

To investigate the relationship between neighborhood poverty and mortality risk, we estimate a series of discrete-time hazard models via pooled logistic regressions. For both neighborhood poverty specifications, we estimate traditional naïve regression models that include baseline as well as time-varying covariates that could be in the causal pathway between neighborhood poverty and mortality risk:

$$\text{Logit } Pr(died(t)) = \text{neighborhood poverty}(t-1) + SES(t-1) + \text{baseline covariates}[Naïve model]$$

We then estimate comparable marginal structural models, which more appropriately address the issue of simultaneous confounding and mediating characteristics of time-varying socioeconomic factors. The conventional naïve models and MSMs are identically specified except that the MSMs do not include the time-varying characteristics and are weighted by the IPT weights:

$$\text{Logit } Pr(died(t)) = \text{neighborhood poverty}(t-1) + \text{baseline covariates [MSM model]}$$

Estimates from naïve and MSMs are then compared.

In summary, we estimate four models:

1. Naïve model that estimates the relationship between single-point neighborhood poverty and mortality risk.
2. Naïve model that estimates the relationship between running average neighborhood poverty and mortality risk.
3. Marginal structural model that estimates the relationship between single-point neighborhood poverty and mortality risk.
4. Marginal structural model that estimates the relationship between running average neighborhood poverty and mortality risk.

Inverse probability of treatment weighting

In the first stage of the marginal structural model, we estimate a person's probability density of residing in his neighborhood poverty level at time (t) as a function of race, gender, and prior level of neighborhood poverty. Since individuals were exposed to neighborhood conditions before the start of the treatment timeframe of interest (i.e., 1993), we also include characteristics of age, education, wealth, income to poverty ratio, marital status, female-headed household, labor force status, and self-rated health as measured in 1990 to maximize the comparability across individuals at baseline. Importantly, baseline 1990 neighborhood poverty is included as a pre-treatment control. Time-varying covariates that are possible mediators as well as confounders include: income, marital status, female-headed household, and employment status. The time-varying covariates are specified as running averages from 1991 to time (t). The IPT weights are then used as probability weights in the second stage outcome models for both the single-point and running average neighborhood poverty exposure specifications. Fig. 1 depicts the temporal structure of the first and second stage MSM strategy.

Because the neighborhood poverty is non-normally distributed with values within the (0,1) interval, we specify its distribution as a beta density function and fit a beta regression model to estimate the probability density evaluated at a person's level of neighborhood poverty at time (t). Specifying neighborhood poverty as beta distributed allows for a flexible parameterization of any continuous probability distribution defined on the interval (0,1) and is thus more flexible than, for example, a lognormal transformation (Comparisons of AIC statistics between the lognormal and beta regressions indicated that the beta specification

resulted in a better model fit.). IPT weights are then constructed from the predicted probability densities and truncated at the 1 and 99 percentiles to mitigate the influence of outliers (Cole & Hernan, 2008).

We account for attrition by applying censoring weights. Censoring at time (t) is specified as binary and a series of logistic regression models were estimated, analogous to the IPT weight models, to construct the censoring weights. A respondent was considered censored at time (t) if they were not observed in and did not die by the next survey year. For 2007, respondents were censored if death was not observed by 2009. Censoring was considered an absorbing state. That is, once a respondent is considered censored, he/she is considered to have permanently attrited. At each survey period, approximately 3% of the sample was censored due to nonresponse or attrition. The exception is for 1995, when 27% of the respondents were censored due to the PSID reducing the size of the families sampled in 1997.

The final MSM weight, which adjusts for both time-varying confounders as well as attrition, is the product of the IPT weight, the censoring weight, and the PSID longitudinal survey weight. In contrast, the weighting for the naïve models is composed of only the PSID longitudinal survey weights. For both strategies, we use Huber–White sandwich estimators to obtain robust standard errors (Brumback, Bouldin, Zheng, Cannell, & Andresen, 2010).

Sensitivity analysis

The MSM strategy is designed to minimize bias due to issues of over-adjustment of confounders that may also be considered as mediators and recovers the causal estimate of treatment under the assumption of conditional exchangeability and no unmeasured confounders. However, because unobserved confounding is not directly addressed, any unobserved differences in individuals that are also correlated to health and neighborhood characteristics are not captured and may bias neighborhood effect estimates. In the context of an MSM framework, omitted variable bias may result when a factor that is correlated with neighborhood poverty level and mortality risk is not included in the first stage prediction model of treatment assignment. The resulting IPT weights would not have accounted for the omitted factor and the unobserved confounding may result in biased estimates of neighborhood effects.

The magnitude of bias due to unmeasured confounders may or may not be large. If the magnitude is relatively small, then inferences from the point estimates may be fairly robust to omitted variable bias. However, if the magnitude is large, then the inferences may be extremely sensitive to possible unmeasured confounding. Although it is impossible to estimate the magnitude of bias due to unobserved confounding from observational data, we can assess the required strength of the hypothetical bias to alter the inferences from our neighborhood-effect results through sensitivity analysis.

We first assume the existence of an unmeasured time-varying confounder with a constant partial correlation level, δ , to neighborhood poverty, and a partial correlation level, α , to mortality, after accounting for the variance of all the baseline variables included in the model. Hence, the δ and α represent conditional associations after adjusting for the baseline

characteristics. We then conduct a series of simulations of 100 iterations to re-estimate the IPT weights and neighborhood effect estimates from the outcome model that included the additional correlation factor. By systematically investigating the impact of different combinations of correlations, the level of correlations required to alter the neighborhood-effect estimates to various magnitudes and to reduce any significant neighborhood-effect findings to statistical insignificance can be estimated. We choose the range of correlation levels from the levels observed in the covariates that were included in our IPT weight models (e.g., income, education) in order to put the magnitude of correlation levels in context (McCaffrey, Ridgeway, & Morral, 2004; Ridgeway, 2006).

Results

Descriptive statistics (see Table 1), as measured at baseline, show that individuals in poor neighborhoods (defined here as those with greater than 20% poverty rate), compared to those in non-poor neighborhoods, are more likely to be black, female, less educated, unmarried, unemployed, a female-headed household, and earn a substantially lower income with lower net worth. The proportions of residents in poor neighborhoods who report poor health are double those of non-poor neighborhoods.

The crude relationships between neighborhood poverty and mortality, adjusted only for baseline age, race, and sex, suggest that both single-point and running average neighborhood poverty increases mortality risk up to the 20% neighborhood poverty level (Table 2). The increased risk then levels off after the 20% threshold.

After adjusting for all baseline and time-varying covariates, naïve estimates for the single-point neighborhood specification indicated no association between neighborhood poverty and mortality risk for either segment, and the point estimates were close to the null (Table 3). However, results from the marginal structural model strategy, which were weighted by the IPT weights (see Table 4 for IPT weight distribution by year) showed a different picture. While MSM estimates still indicated no statistically significant effect of neighborhood poverty on mortality for poverty levels below 20%, a 58% increased odds (95% CI = [1.20, 2.10]) was demonstrated for each 10 percentage point increase in neighborhood poverty after the 20% threshold.

The patterning of results for the running average neighborhood specification paralleled those for the single-point neighborhood specification. That is, results from the fully adjusted naïve models uncovered no connection between running average neighborhood poverty and mortality risk. Again, while the marginal structural model estimates indicated no significant association existed between a range of 0–20% running average neighborhood poverty, a statistically significant increased odds of mortality [OR = 1.89; 95% CI = [1.22, 2.91]] above the 20% poverty level was found (Table 5).

Sensitivity results against unobserved confounding are presented in Table 6 for the MSM running average neighborhood poverty model. Through simulation, we estimated the neighborhood effect estimates adjusted for a hypothetical confounder with various combinations of partial correlation levels (controlling for all other baseline variables

included in the model) within the range of -0.25 to 0.25 for mortality and -0.20 to 0.20 for neighborhood poverty. Since the MSM results recovered a statistically significant neighborhood poverty effect only for poverty levels above 20%, results presented in Table 6 reflect the effect estimates only for the second segment in the spline model (i.e., above 20% average running neighborhood poverty). We are mainly interested in a positive/positive or negative/negative correlation combination that would induce a positive bias of our model estimates. For example, the sensitivity analysis suggests that in the presence of a hypothetical confounder with correlation levels of -0.10 for mortality and neighborhood poverty, MSM analysis would still have yielded a statistically significant effect estimate of $OR = 1.75$, 95% $CI = [1.12, 2.74]$ for a 10 percentage point increase in running average neighborhood poverty above 20%. However, a confounder with higher partial correlation levels of -0.15 and -0.20 to mortality and neighborhood poverty, respectively, would change our inferences to statistical non-significance ($OR = 1.55$, 95% $CI = [0.95, 2.54]$). To put this in context, the partial correlation levels observed in our data for education are -0.02 and -0.11 with mortality and neighborhood poverty, respectively. Thus, had an additional confounder with similar correlation levels of education been included in our model, MSM results would still have yielded statistically significant estimates (approximately $OR = 1.84$, 95% $CI = [1.20, 2.85]$). Similarly, an omitted confounder that is positively associated with both neighborhood poverty and mortality risk with a partial correlation level similar to poor health (0.12 and 0.03 to death and neighborhood poverty, respectively) would still have also yielded statistically significant estimates.

Discussion

In this study, we used longitudinal data and applied a marginal structural modeling strategy to investigate the relationship between neighborhood poverty and mortality risk. As aforementioned, there has been much debate in the literature over the role of confounding and mediating factors in neighborhood-health studies. Given that place may determine many of the control variables (e.g., employment status) used in conventional models, it is difficult to tease out what is truly a confounding effect from a mediating effect. Conventional regression adjustments attempt to control for confounding by including both the neighborhood context and all possible confounders as covariates. But when there exist time-varying covariates that are simultaneously mediators and confounders, standard regression adjustments may result in biased estimates of neighborhood effects. The extent of the bias in neighborhood effects on health due to over-adjustment of mediating factors has not been fully explored.

While estimates from traditional naïve models and MSMs are not generally directly comparable because the former recovers a conditional estimate while the latter recovers a marginal estimate, marginal and conditional estimates are equivalent from models that estimate risk ratios (Kaufman, 2010). Because the risk of mortality in each time period is rare (approximately 2–3%), the odds ratio recovered can be considered as approximate risk ratios. Hence, we can explicitly compare results from the naïve and MSM strategies.

We found no evidence from the traditional naïve strategy that neighborhood context influences mortality risk based on single-point or running average measures of

neighborhood poverty. The null findings from our naïve models are somewhat in contrast to the results from the majority of studies in the extant literature that examined the relationship between neighborhood socioeconomic status (variously defined) and overall or cause-specific mortality (Meijer et al., 2012; Pickett & Pearl, 2001; Riva et al., 2007). While it is difficult to directly compare findings due to differences in definitions and specifications of neighborhood poverty/disadvantage being investigated and a high heterogeneity in results across studies, meta-analysis does indicate that among studies that examined small areas, defined as those with than 7000 inhabitants (Census tracts have between 2500 and 8000 residents), there is a statistically significant increased overall relative risk in mortality for residents in lower-SES neighborhoods, compared to those in higher-SES neighborhoods ($RR = 1.11$; 95% $CI = [1.08, 1.14]$) (Meijer et al., 2012). However, most studies were cross-sectional and did not adjust for an extensive list of individual-level socioeconomic characteristics. Hence, they are susceptible to unobserved confounding, which may result in biased estimates that were stronger than warranted.

In contrast to the null findings from the naïve models, results from the MSM strategy for both the single-point and running average measures of neighborhood poverty reveal statistically significant detrimental effects indicating that mortality risk is sensitive to increases in neighborhood poverty in high poverty areas. That is, increases in neighborhood poverty do not appear to influence mortality risk as long as neighborhood poverty remains under 20%. However, after the 20% threshold is reached, statistically significant increases in the odds of mortality is observed as neighborhood poverty rises. The estimate for the running average neighborhood poverty measure was stronger than the single-point measure, $OR = 1.89$ versus 1.54 , respectively. The pattern of results suggests three main conclusions: 1) conventional naïve regression estimates underestimate the total direct and indirect impact of neighborhood poverty and their correlates on mortality risk, 2) single-point neighborhood measures that may more readily capture relatively brief exposures are more weakly connected to mortality risk than long-term exposures, and 3) the relation between neighborhood poverty and mortality risk is nonlinear such that the risk of mortality is not increased until neighborhood poverty reaches a certain threshold.

The relative magnitudes between single-point and long-term estimates from the MSM results are consistent with the notion that transient exposures to specific neighborhood context exert a weaker influence than habitual exposure. Empirical evidence from studies that compare single-point versus long-term measures of neighborhood context also supports this intuitive perception (e.g., Do, 2009). The larger effect estimates from the MSM strategy are consistent with our hypothesis that conventional regression adjustments over-adjusts for factors that are mediators and lie in the causal pathway between neighborhood context and health.

The shape of the nonlinear relation found between neighborhood poverty and mortality risk indicates that as long as neighborhood poverty and its correlates are below a certain threshold, its impact on mortality risk is negligible – at least within the exposure duration measured in this study. However, above that threshold, the social and economic challenges, such as higher crime rate and lower employment and quality educational opportunities, begin to exert a toll on residents' health, above individual-level circumstances. We

examined whether our results were sensitive to the knot point specification by also placing knots at 10% and 15%; estimates below the knot point remained statistically non-significant and estimates for neighborhood poverty above the knot points were $OR = 1.73$, 95% $CI = [1.17, 2.57]$ and $OR = 1.86$, 95% $CI = [1.25, 2.75]$, respectively. We also specified knot points at two locations, 10% and 20%, to further examine the relationship between neighborhood poverty below 20% and mortality. Consistent with previous results, no statistically significant relationship with mortality was found for the 0–10% and 10–20% neighborhood poverty ranges. A statistically significant relationship, though expectedly attenuated, was found for a linear specification without any knots ($OR = 1.56$, 95% $CI = [1.01, 2.40]$).

The patterning of results suggest that policy aimed at redistributing resources in order to minimize the number of neighborhoods that exceed a certain level of poverty and disadvantage may be particularly effective and efficient to reduce the mortality deficit found in higher poverty areas. The implications for this are particularly salient given the increasing spatial concentration of poverty in the United States. Approximately 23% of the U.S. population during the period of 2006–2010 lived in poor neighborhoods characterized by poverty rates over 20%, compared to 19% in the beginning of the decade (Bishaw, 2005, 2011). Further, 8.7 million Americans resided in extreme poverty neighborhoods where at least 40% of residents are below the poverty rate (Kneebone, Nadeau, & Berube, 2011). This represents a one third increase from the year 2000 overall, with suburbs experiencing a much higher growth of 54 percent in extreme poverty neighborhoods. Accompanying this spread of extreme poverty neighborhoods into suburbs were demographic shifts in the composition of its residents; compared to 2000, residents of extreme poverty neighborhoods were more likely to be white and high school or college graduates (Bishaw, 2005, 2011; Kneebone et al., 2011). Hence, the challenges and health consequences faced by residents in poor and extreme poverty neighborhoods are increasingly less isolated to racial/ethnic minorities and the uneducated.

Important for policy recommendations, sensitivity analysis suggests that our results are moderately robust to unobserved confounding such that our inferences would still hold in the presence of an additional confounder as strong as education or poor health. Further, our results were robust to whether we truncated the IPT weight separately at each year or pooled together. However, results were somewhat sensitive to the level of truncation. A 2.5% truncation expectedly attenuated the estimate and yielded results (though still suggestive of a positive relation) that were statistically non-significant ($OR = 1.25$, 95% $CI [0.94, 1.67]$).

Nonetheless, there is a strong reason to believe that the total effect of neighborhoods on health is even more pronounced than the results from this study. The MSM estimates recovered in this study reflect the effect of adult neighborhood poverty exposure within the 15-year analytical timeframe. Hence, it does not estimate the effect of exposure throughout the entire life-course. Because childhood environment influences individuals' socioeconomic trajectories, using conditions observed in adulthood as baseline factors negates any childhood neighborhood impacts on multiple health determinants (e.g., educational attainment, adaptation of behavioral norms, employment prospects, etc.). Consequently, results from this study may only capture a portion of life-course

neighborhood influences on health. Analyses from data with childhood or young adulthood information that can serve as baseline measures would allow a substantially longer timeframe for which the impact of neighborhood can play out.

In addition to this limitation, it is important to note that three key assumptions of the marginal structural model may not be fully met. The first of these – *neighborhood-level SUTVA* – assumes that the poverty rates in one neighborhood does not affect the mortality risk for individuals in another neighborhood (Vanderweele, 2008). To the extent that neighborhood conditions in adjacent neighborhoods may have spill over effects (e.g., high crime rates which coincide with neighborhood poverty may spill over to adjacent neighborhoods), this assumption may not hold. The second assumption is consistency, which implies that the observed outcome at the observed exposure is equal to the potential outcome associated with the observed exposure. This assumption is reasonable in drug trials, where the treatment within a given arm is consistently applied, but in observational studies may be more problematic, especially when, as here, there may be many mechanisms by which a neighborhood reaches a given poverty level. Because the PSID is a nationally representative sample, the extensive geographic and temporal coverage of the data compel us to rely on administrative data for neighborhood context. This limits our ability to investigate specific features of neighborhoods that would have allowed us to better ensure that the consistency assumption holds. A third key assumption is positivity: each subject in the sample is presumed to have a non-zero probability of having any level of exposure (Here, because the exposure is continuous, positivity implies that the probability distribution function of neighborhood poverty rate associated with each subject is greater than 0 for all rates inclusive of 0 and 1.). Modeling neighborhood poverty rate using a beta distribution ensures this assumption will be met in the model, but to the extent that the model fails – e.g., if white high SES respondents have no chance of living in a neighborhood with extremely high poverty rates – the positivity assumption may not be met in the data. Similarly, trimming large weights (which belong to subjects predicted to have an extremely low probability of living in a neighborhood with a given level of poverty) to stabilize estimates may attenuate on average the estimated effect of the exposure.

Despite these limitations, this study represents a marked improvement over conventional neighborhood-health studies. We are able to at least partially adjust for bias related to incorrect adjustments of time-varying factors that are simultaneously mediators and confounders and show that conventional estimates can severely underestimate the impact of neighborhoods on health. Because policy inferences and anticipated impacts of interventions rely on estimates of causal versus associational connections, addressing these sources of bias are important to make appropriate policy recommendations. Indeed, the conventional estimates would imply that neighborhood poverty and its correlates have little to no impact on mortality risk while the MSM estimates indicate otherwise. Results from this study suggest that a more substantial investigation, using data throughout the life-course beginning at childhood may yield even stronger evidence to the impact of neighborhoods on health. Whether any or all of these patterns are applicable to other health outcome measures await future research.

Acknowledgments

We thank Bei Jiang for her help in preparing the data for analyses and conducting the preliminary analytical models. Funding for this project was provided by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (grant #R21 HD058715).

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Fig. 1.

Marginal structural modeling temporal framework, Panel Study of Income Dynamics, 1990–2009. $L(90)$ reflects baseline covariates used to predict neighborhood poverty exposure at time (t). $L(\text{mean}91\text{to}(t))$ reflects time-varying covariates, specified as running averages up to time (t), used to predict neighborhood poverty exposure at time (t). $Npov(t)$ reflects neighborhood poverty measured at time t . This is the outcome variable in the 1st stage and the independent variable in the 2nd stage. All models in the 1st stage included baseline and prior neighborhood poverty to predict $Npov(t)$.

Table 1PSID sample characteristics (percentages or means) at 1990 baseline by neighborhood poverty category.^a

Characteristics	High poverty neighborhood (>20% poverty)	Low poverty neighborhood (20% poverty)
<i>N</i>	1855	4619
Proportion of baseline sample	17.22	82.78
Gender		
Female	60.17	54.79
Race		
White	58.93	93.34
Black	41.07	6.60
Age	48.64 (16.83)	49.44 (16.49)
Income poverty ratio		
1	23.93	5.94
>1 & 2	21.04	12.87
>2 & 3	19.58	17.36
>3	35.46	63.84
Wealth category		
0	16.40	5.61
Quartile 1: >0 & 12,560	25.86	14.56
Quartile 2: >12,560 & 46,560	24.91	19.40
Quartile 3: >46,560 & 136,600	19.84	27.30
Quartile 4: >136,600	12.98	33.13
Education (years)		
11	27.54	13.12
12	39.49	37.19
13–15	19.08	23.82
16	7.05	13.59
17	6.85	12.27
Labor force status		
Employed	54.32	63.55
Unemployed	6.79	2.97
Retired	18.95	19.02
Keeps house	14.03	11.85
Other	5.91	2.61
Marital status		
Married	53.39	71.34
Never married	17.20	8.06
Widowed	12.39	8.69
Divorced/Separated	17.02	11.91
Female-headed household	13.44	4.02
Self-rated health		
Excellent	13.87	21.65

Characteristics	High poverty neighborhood (>20% poverty)	Low poverty neighborhood (20% poverty)
Very good	27.09	35.48
Good	32.18	28.68
Fair	18.86	10.39
Poor	8.01	3.80

^aStandard deviation in parenthesis. All proportions and means reflect statistics that were weighted by the PSID longitudinal survey weight.

Table 2Crude estimates for neighborhood and mortality, adjusted for baseline age, race, and sex.^a

Neighborhood poverty	OR [95% CI]
<i>Single-point</i>	
neigh pov for range [0 to 20%]	1.53 [1.13, 2.06]
neigh pov for range [20 to 100%]	1.02 [0.91, 1.14]
<i>Running average</i>	
neigh pov for range [0 to 20%]	2.02 [1.48, 2.76]
neigh pov for range [20 to 100%]	1.03 [0.92, 1.16]

^aOR reflects a hypothetical change of 10 percentage points.

Table 3Single-point-in-time neighborhood poverty results.^a

Neighborhood poverty	OR [95% CI]
<i>Naive model</i>	
neigh pov for range [0 to 20%]	0.95 [0.77, 1.16]
neigh pov for range [20 to 100%]	0.88 [0.73, 1.06]
<i>MSM model</i>	
neigh pov for range [0 to 20%]	0.89 [0.48, 1.64]
neigh pov for range [20 to 100%]	1.58 [1.20, 2.10]

^aOR reflects a hypothetical change of 10 percentage points.

Table 4

Distribution of IPT weights truncated at the 1 and 99 percentiles by year.

Year	Mean	Standard deviation
1993	1.31	4.26
1995	1.69	6.06
1997	2.04	7.67
1999	2.31	8.60
2001	2.56	9.28
2003	2.88	10.04
2005	3.13	10.68
2007	3.34	11.23

Table 5Running average neighborhood poverty results.^a

Average neighborhood poverty	OR [95% CI]
<i>Naive model</i>	
neigh pov for range [0 to 20%]	1.00 [0.80, 1.25]
neigh pov for range [20 to 100%]	0.89 [0.73, 1.09]
<i>MSM model</i>	
neigh pov for range [0 to 20%]	1.04 [0.40, 2.72]
neigh pov for range [20 to 100%]	1.89 [1.22, 2.91]

^aOR reflects a hypothetical change of 10 percentage points.

Table 6

Sensitivity analysis for running average neighborhood poverty MSM model: simulated results (odds ratio effect estimates and 95% confidence intervals) adjusted for hypothetical unobserved confounding by various levels of correlations to mortality and neighborhood poverty.^a

α :corr(OV, mortality) ^b	δ :corr (OV, neighborhood poverty) ^c										
	-0.20	-0.15	-0.10	-0.05	0.05	0.10	0.15	0.20	0.15	0.10	0.05
-0.25	1.39 [0.82, 2.35]	1.47 [0.89, 2.42]	1.58 [0.99, 2.55]	1.73 [1.10, 2.72]	2.09 [1.39, 3.15]	2.30 [1.57, 3.39]	2.55 [1.76, 3.70]	2.80 [1.95, 4.04]	2.55 [1.76, 3.70]	2.30 [1.57, 3.39]	2.09 [1.39, 3.15]
-0.20	1.46 [0.88, 2.44]	1.54 [0.95, 2.50]	1.63 [1.03, 2.61]	1.75 [1.12, 2.75]	2.04 [1.35, 3.10]	2.21 [1.49, 3.28]	2.39 [1.63, 3.50]	2.58 [1.77, 3.76]	2.39 [1.63, 3.50]	2.21 [1.49, 3.28]	2.04 [1.35, 3.10]
-0.15	1.55 [0.95, 2.54]	1.61 [1.00, 2.58]	1.69 [1.07, 2.67]	1.78 [1.14, 2.79]	2.00 [1.31, 3.05]	2.11 [1.41, 3.17]	2.24 [1.51, 3.32]	2.37 [1.61, 3.50]	2.24 [1.51, 3.32]	2.11 [1.41, 3.17]	2.00 [1.31, 3.05]
-0.10	1.65 [1.03, 2.65]	1.69 [1.07, 2.68]	1.75 [1.12, 2.74]	1.81 [1.16, 2.83]	1.96 [1.28, 3.00]	2.03 [1.34, 3.07]	2.10 [1.40, 3.16]	2.18 [1.46, 3.27]	2.10 [1.40, 3.16]	2.03 [1.34, 3.07]	1.96 [1.28, 3.00]
-0.05	1.77 [1.12, 2.77]	1.78 [1.15, 2.78]	1.81 [1.17, 2.81]	1.84 [1.19, 2.87]	1.92 [1.25, 2.95]	1.95 [1.27, 2.98]	1.98 [1.30, 3.02]	2.02 [1.33, 3.08]	1.98 [1.30, 3.02]	1.95 [1.27, 2.98]	1.92 [1.25, 2.95]
-0.025	1.83 [1.18, 2.84]	1.83 [1.19, 2.83]	1.84 [1.20, 2.85]	1.86 [1.20, 2.88]	1.90 [1.23, 2.93]	1.91 [1.24, 2.94]	1.92 [1.26, 2.95]	1.94 [1.27, 2.99]	1.92 [1.26, 2.95]	1.91 [1.24, 2.94]	1.90 [1.23, 2.93]
0.025	1.97 [1.29, 3.00]	1.94 [1.28, 2.95]	1.92 [1.26, 2.93]	1.89 [1.23, 2.92]	1.87 [1.20, 2.89]	1.84 [1.19, 2.85]	1.82 [1.17, 2.83]	1.81 [1.16, 2.83]	1.82 [1.17, 2.83]	1.84 [1.19, 2.85]	1.87 [1.20, 2.89]
0.05	2.05 [1.36, 3.09]	2.00 [1.32, 3.02]	1.96 [1.29, 2.97]	1.91 [1.24, 2.94]	1.85 [1.19, 2.87]	1.81 [1.16, 2.81]	1.77 [1.13, 2.77]	1.75 [1.11, 2.76]	1.77 [1.13, 2.77]	1.81 [1.16, 2.83]	1.85 [1.19, 2.87]
0.10	2.21 [1.50, 3.28]	2.12 [1.43, 3.16]	2.04 [1.36, 3.07]	1.95 [1.27, 2.99]	1.82 [1.16, 2.83]	1.74 [1.11, 2.74]	1.68 [1.06, 2.67]	1.64 [1.02, 2.63]	1.68 [1.06, 2.67]	1.74 [1.11, 2.74]	1.82 [1.16, 2.83]
0.15	2.40 [1.65, 3.50]	2.26 [1.54, 3.32]	2.13 [1.43, 3.17]	1.99 [1.30, 3.03]	1.78 [1.14, 2.79]	1.69 [1.07, 2.67]	1.60 [1.00, 2.58]	1.54 [0.94, 2.52]	1.60 [1.00, 2.58]	1.69 [1.07, 2.67]	1.78 [1.14, 2.79]
0.20	2.59 [1.80, 3.74]	2.42 [1.67, 3.50]	2.22 [1.51, 3.27]	2.03 [1.34, 3.08]	1.76 [1.12, 2.76]	1.63 [1.02, 2.61]	1.53 [0.94, 2.49]	1.46 [0.88, 2.42]	1.53 [0.94, 2.49]	1.63 [1.02, 2.61]	1.76 [1.12, 2.76]
0.25	2.79 [1.96, 3.99]	2.58 [1.80, 3.69]	2.32 [1.59, 3.39]	2.07 [1.37, 3.12]	1.73 [1.10, 2.73]	1.59 [0.99, 2.55]	1.47 [0.89, 2.42]	1.38 [0.82, 2.34]	1.47 [0.89, 2.42]	1.59 [0.99, 2.55]	1.73 [1.10, 2.73]

Bold face type reflects statistically significant estimates.

^a OR reflects a hypothetical increase of 10 percentage points in neighborhood poverty for the second segment in the spline model (i.e., above the 20% running average neighborhood poverty level) with the addition of a hypothetical confounder with partial correlation levels α and δ .

^b α :corr (OV, mortality) reflect the partial correlation level between the hypothetical omitted variable and mortality.

^c δ :corr (OV, neighborhood poverty) reflect the partial correlation level between the hypothetical omitted variable and neighborhood poverty.