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Neighborhood poverty and American Indian infant death: are the effects identifiable?

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Abstract

Purpose—Poor living conditions are posited as an underlying cause of American Indian (AI) infant mortality, which is unusually high in the post-neonatal period. We explore whether the effects of neighborhood poverty on AI infant death are identifiable using observational data.

Methods—Vital records for infants born between 1990 and 1999 to AI women in a metropolitan area (n = 4,751) are linked with tract-level poverty data. A counterfactual framework, an explicit causal contrast study design, and propensity score matching methods are employed. For each comparison, we create exchangeable groups by matching infants with the same probability of exposure to poverty when one was exposed and the other was not.

Results—Our results suggest that neighborhood poverty has little effect on AI infant death outcomes. Importantly, the study design makes transparent the challenge of identifying appropriate analytic comparison groups in studies of neighborhood poverty and health.

Conclusions—Collecting additional data will likely not overcome the fact that AI with a high probability of living in poverty rarely reside in low poverty neighborhoods. Yet, some of them must if a meaningful counterfactual comparison is to be made and the effects of neighborhood poverty on AI infant death are to be identified.

MeSH Keywords

Causality; Confounding Factors (Epidemiology); Epidemiologic Research Design; Indians; North American; Infant Mortality

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High rates of infant mortality continue to characterize many American Indian (AI) communities, both on reservations and in urban areas throughout the United States (US). However, little is known about the etiology of urban AI infant mortality, including potential mechanisms of risk.

Researchers have noted a number of individual-level risk factors for infant mortality in the general population. These are often associated with pregnancy complications and low birth

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weight, which are closely tied to neonatal mortality, death in the first 28 days after birth (1). Among AI, however, more infant deaths occur among average weight infants than for any other race group (2). Further, while two-thirds of infant deaths in the US occur in the neonatal period for all other race groups, nearly half of AI infant deaths still occur in the postneonatal (between 28 days and one year) period (2). Since postneonatal death has historically been attributed to social and environmental conditions (1), a relationship between social conditions (e.g. neighborhood poverty) and infant death is hypothesized.

Interest in the relation of poverty to infant mortality is hardly new. In the early 20th Century, many believed that poverty was related to the most common causes of infant death (3–6). Decades later researchers began to question this as published studies suggested an attenuation of the poverty-infant mortality relation (7–9). But the question remains. In contemporary reviews of AI infant mortality several authors *assert* an important role for poor living conditions (10–12) while others *conclude* that poor living conditions are key (13–15). However, only one study empirically evaluates the claim (16). The fact is that poverty's impact on health, especially infant mortality, is still not fully understood.

METHODS

We seek an answer to the general question: what is the effect of being born into a high poverty neighborhood compared with being born into a low poverty neighborhood on AI infant death. In other words, what would be the proportion of deaths among AI infants born into high poverty neighborhoods *if instead* they themselves had been born into low poverty neighborhoods? To answer this question we employ a three-step approach comprising: 1) a counterfactual framework, 2) explicit causal contrasts, and 3) propensity score matching methods.

This study relies on observational data containing individual-level linked birth/infant death (LBD) data from the Minnesota Department of Health, Center for Health Statistics, linked with neighborhood-level data from the 1990 Decennial Census of Population and Housing (STF3A). The data represent infants born to self-identified AI women residing in a metropolitan area of Minnesota from 1990 through 1999 (n = 4,751).

Outcome measure

Our outcome is infant death, which we subclassify to distinguish causes of death presumably amenable to medical versus social intervention. Using documented ICD-9 or ICD-10 codes for underlying cause of death, we categorize each infant death as either endogenous (internal or biologic in origin) or exogenous (external or socio-environmental in origin) using a previously developed classification scheme (17,18).

Exposure measure

The exposure of interest is neighborhood poverty, which is derived from 1990 US Census data. As there is no consensus on how poverty should be measured (19,20) nor how it should be operationalized for investigations of poverty and health (21), we choose the federal poverty threshold as defined in the 1990 Census data. Recent epidemiologic work on area-based socioeconomic measures and health disparities suggests that measures of economic deprivation (e.g. percent poverty) are the most robust at detecting social gradients in numerous health outcomes compared with single variable measures of neighborhood education and wealth or compared to composite indices (22).

Krieger et al. recommend using area percent (of the population) below poverty with a priori cut points, including the policy-relevant designation of ≥ 20% below poverty as a federally defined poverty area (22). They use < 5%, 5–9%, 10–19% and ≥ 20% below poverty to

distinguish neighborhood poverty levels. For our study, these cut points were unsuitable due to the distribution of poverty among neighborhoods in which AI live: few Minnesota neighborhoods in which AI reside have less than 20% poverty. Accordingly, we define four categories of neighborhood poverty as < 5%, 5–19%, 20–39%, and ≥ 40% below poverty.

Analysis

We want to estimate the average effect of levels of exposure (poverty) on those who are in fact exposed. Each comparison is framed as an explicit causal contrast (23), for which we want to create exchangeable groups of exposed and unexposed (counterfactual) infants. In order to make a proper comparison, these two groups must be, on average, balanced on all relevant and available covariates (24). We use propensity score matching methods to create matched pairs comprising these two groups (25).

A propensity score is the conditional probability of being exposed/treated (26,27). The propensity score reduces the dimensionality of a large set of potential confounders to unity, which is conducive to simple pair matching. To estimate propensity scores, individual-level variables from the LBD data are used as predictors (covariates) of exposure (each category of neighborhood poverty) in a logistic regression model. Proper covariates are those that are predictive of the exposure of interest and occur prior to the outcome of interest.

Because social stratification surely plays a role in where AI live, we select all sociodemographic variables available in our dataset including maternal age and education, paternal race and education, and marital status. Other covariates expected to differ across exposure categories, and which occur prior to the infant being exposed to the neighborhood, include maternal smoking, prenatal care utilization, and numbers of previous births and child deaths. While conceding that the problem is difficult, we include maternal smoking, inadequate prenatal care utilization and previous child deaths – measures that may be the effects of living in poverty rather than predictors of it. Similarly, birth weight and number of children at this birth (singleton or twins) are also arguably improper covariates. Nevertheless, we include them since both are strongly related to the outcome of interest.

Propensity score estimation and matching are performed with the PSMATCH2 (28) module available for Stata statistical software (29). For each causal contrast specified, the probability of exposure (propensity score) is estimated and exposed infants are matched (with replacement) to unexposed infants on estimated propensity scores within a range of ± 0.01. In doing so, we match two infants with the same probability of exposure when in fact one of them was exposed and the other was not. After matching on propensity scores, we assess covariate balance across the two exposure groups using standardized differences (30). The goal is to achieve balance in observed covariates, just as we would expect if we had randomized subjects to exposures. This lets us be more comfortable with the assumption that these two groups are exchangeable (except for the exposure). We cannot, of course, assess and ensure balance on unmeasured confounders.

We estimate the average effect of the treatment on the treated (ATT), which is the average effect of the neighborhood poverty exposure on those who are in fact exposed. Conceptually, the ATT is a rate ratio derived from a comparison of the infant mortality rate (IMR) among infants born into high poverty neighborhoods (i.e., exposed) with what the IMR would have been had these same infants been born into low poverty neighborhoods (i.e., unexposed). There are a variety of methods for calculation of the standard error of propensity score matched effect estimates (31). We use the bootstrap, with 1000 repetitions, to estimate the standard error of the estimated ATT, which accounts for the propensity score estimation, the matching, and the ATT estimation. Finally, 95% confidence intervals are reported using a bias-corrected

confidence interval (recommended by Efron and Tibshirani, pg. 188) from the bootstrap procedure (32).

RESULTS

Table 1 shows selected characteristics and infant death outcomes for births to AI women by neighborhood poverty exposure group. Only 11% (n = 533/4,751) of births to AI women occur to those residing in low poverty (< 5%) neighborhoods, while nearly one-third (30%) are to women residing in high poverty (≥ 40%) neighborhoods. The columns represent the distribution of characteristics and infant outcomes within each neighborhood poverty exposure group. As expected, the characteristics of those in low poverty neighborhoods suggest higher social status and lower risk for infant death. Specifically, this group is slightly older, less parous, more adequately educated, more likely to be married, less likely to have inadequate or no prenatal care, and less likely to smoke than any of the other neighborhood poverty exposure groups.

The expected social gradient across increasing levels of neighborhood poverty is clear and strong for all social indicators. For example, 80% of birth mothers in the low poverty group have adequate education for age. With increasing neighborhood poverty level comes a marked decrease in the proportion of mothers with adequate education (68%, 54%, and 46%, respectively); a pattern that remains consistent for all other social indicators, as well as infant death outcomes. Indeed, infant mortality rates rise as the level of neighborhood poverty rises, such that the crude all-cause infant mortality rate is over three times higher for infants born into high poverty neighborhoods compared with infants born into low poverty neighborhoods (IMR = 23.3 and 7.5, respectively). Evident differences between exposure groups underscore the importance of achieving balance on the differentially distributed characteristics.

Propensity scores are estimated for three separate causal contrasts representing increasing levels of neighborhood poverty exposure: a) 5–19% poverty compared with < 5% poverty, b) 20–39% poverty compared with < 5% poverty, and c) ≥ 40% poverty compared with < 5% poverty. Figure 1 graphically depicts the overlap for each of the three causal contrasts specified. Each graph represents the distribution of the propensity scores for being born into the specified neighborhood poverty exposure for those who were in fact exposed (neighborhood poverty level) and for those who were unexposed/counterfactual (< 5% poverty). The bars to the left are propensity scores for the exposed, those to right for the unexposed. Generally, the overlap shown suggests comparability across the two exposure groups for the first panel. In other words, there is adequate overlap in the propensity score distributions for the two groups such that we should be able to find a suitable (counterfactual) match for most of the exposed subjects. However, in the second and third panels it appears that very few unexposed infants have a high probability of being in a higher poverty neighborhood. Thus, a small number of unexposed infants (those born into low poverty neighborhoods) who have high probabilities of being born into higher poverty neighborhoods will likely serve as the unexposed (counterfactual) match for more than one (or even many) exposed infants.

Table 2 presents the effect estimates of increasing levels of neighborhood poverty on infant death outcomes. The first section shows the results for effects of poverty on endogenous-cause deaths. Few statistically meaningful effects of neighborhood poverty on endogenous cause deaths are observed. Specifically, we only detect significant effects for 20–39% poverty. In this case, the infant mortality rate for AI infants born into neighborhoods with moderately high poverty was four times higher than the rate would have been if instead these infants had been born into neighborhoods with low poverty (RR = 4.0, 95% CI = 1.8, 19.0). The second section of Table 2 presents the effect estimates of neighborhood poverty on exogenous-cause AI infant

death. We are unable to detect any statistically meaningful effects of neighborhood poverty here.

We conducted a post-hoc sensitivity analysis using a Mantel-Haenszel bounds procedure to assess the sensitivity of our effect estimates (33). The sensitivity results indicate that an unmeasured confounder would have to triple or quadruple the odds of differential exposure (gamma) in order for the effects of poverty on infant death to become significant (data not shown).

DISCUSSION

The empirical evidence does not present a strong case for an independent effect of neighborhood poverty on AI infant death outcomes in one urban area. We observe some direct effect of poverty on endogenous-cause deaths and no effect on exogenous-cause deaths. Specifically, our analyses suggest that infants born into neighborhoods with moderately high poverty had endogenous-cause death rates 4.0 times higher than they would have been had they been born into low poverty neighborhoods. This was unexpected as our hypotheses about the effects of neighborhood poverty were drawn from widely held beliefs that poor social conditions are linked to poor health outcomes and further, that exogenous-cause infant deaths would be linked directly to poor social and/or environmental conditions (i.e. poverty).

Since endogenous-cause infant deaths are typically biologic in origin, the effect of neighborhood poverty on endogenous-cause deaths should be indirect, mediated through maternal health. Plausible effects of poverty on maternal health that may translate to infant health have been well documented. For instance, residence in a high poverty neighborhood may diminish access to adequate nutrition, to culturally-appropriate and quality health care, or to reliable modes of communication and transportation (34–36). It is also possible that some or all of these things are available, but that circumstances in a high poverty neighborhood constrain one's options to choose only one in lieu of the others (37). Further, living in a high poverty neighborhood may increase maternal exposure to psychosocial stressors such as crime and violence, which may result in physiologic responses or detrimental coping mechanisms either of which could lead to an unhealthy in utero environment (38–40). No data were available with which to explore these additional possibilities.

The underlying cause of exogenous death among AI infants continues to elude. The lack of significant findings for neighborhood poverty and exogenous death may in part be explained by the deaths that were classified as exogenous. Specifically, 51% of the exogenous deaths were attributed to Sudden Infant Death Syndrome. If SIDS has a strong biologic or genetic component, over half of the deaths in the exogenous-cause death category would seemingly be misclassified. It is also possible that, all other things equal, there is simply no discernable effect of neighborhood poverty on infant death outcomes for the population of urban AI in our study.

Importantly, our findings highlight the relatively small pool of unexposed (counterfactual) matches from which we had to choose when using the 'ideal' low poverty group as the counterfactual comparison. The notably high infant mortality rates estimated for the unexposed matched group underscore the implications of having relatively few infants with a high probability of high poverty exposure available as a counterfactual match. Unexposed infants with a high probability of exposure served as a counterfactual match for numerous exposed infants. Multiple matches occurred as follows: a) for 5–19% compared with < 5% poverty, unexposed infants were matched between 0 and 36 times (mean = 2.8, SD = 3.8); b) for 20–39% compared with < 5% poverty, unexposed infants were matched between 0 and 36 times

(mean = 2.3, SD = 4.5); and c) for ≥ 40% compared with < 5% poverty, unexposed infants were matched between 0 and 66 times (mean = 2.6, SD = 6.6).

Study limitations

First, our data are observational and observational designs are fraught with inferential obstacles. Of special note is our inability to account for potential unobserved or unobservable differences; it is possible that unmeasured confounders between groups explain any detected difference in infant death.

Second, linked birth/infant death data are the gold standard for infant mortality research, but they are not perfect. Importantly, key variables are not available in vital records. Family income or poverty data are not routinely collected on the birth record. Thus, we are unable to assess the effects of family poverty on infant death, which may in fact be more important. More complete documentation of paternal characteristics, as well as data on parents' employment, occupation, and health insurance status may be useful in more fully accounting for the sociodemographic makeup, which could produce a more accurate propensity score estimation model. Additionally, incorporating maternal health data into the infant death analyses may provide important clues linking neighborhood poverty to endogenous death outcomes. Moreover, nearly half of the infant deaths (49%) occurred to hospital inpatients. As expected, 78% of endogenous-cause deaths were among inpatients, while only 15% of exogenous-cause deaths occurred to hospital inpatients. However, we are unable to ascertain from the data available whether these infants ever left the hospital after birth. To better assess the direct effects of exposure to neighborhood poverty on infant death we need a measure of duration of infant exposure to the neighborhood.

Third, the urban AI population is unique. This is a heterogeneous population representing numerous tribes with different histories, cultural practices, and traditions. Many are highly mobile, moving frequently from urban to reservation homes. Likewise, there are a multitude of cultural strengths inherent in this community that affect infant health, either directly or by buffering the negative effects of poverty. Data limitations do not allow us to account for mobility, tribal affiliation, cultural strengths, levels of assimilation, or exposure to tribal public health, as well as the overall diversity of this urban AI population. Moreover, lack of data sources for examining urban AI health and healthcare has been noted as a serious deficiency in more fully understanding AI health (41–43).

Finally, we intentionally limit our investigation to infants born to AI women living in one metropolitan area, since relocation within this area seems possible. The complexity of social constructs like race/ethnicity make hypothetical experiments suggesting that AI are exchangeable with, say, non-Hispanic whites less tenable (44). Restricting our sample to infants born to AI women seems to make an exchangeability assumption more reasonable. However, this important design strength severely limits the number of infants available for matching from the unexposed (counterfactual) group. Note that collecting more observations will not overcome the fact that AI with a high probability of living in poverty rarely reside in low poverty neighborhoods – a situation recently termed "structural confounding"(45).

Conclusion

The effect of neighborhood poverty on AI infant death is not identifiable given the available data. Moreover, identification of neighborhood effects on health outcomes, generally, is difficult both conceptually and inferentially. Given the methodological difficulties in estimating "neighborhood effects" with a (multilevel) regression model (46), we employed a counterfactual framework, explicit causal contrasts, and propensity score matching methods. Since the causal pathways from poverty to infant death likely differ by cause of death, we

estimated the independent effects of neighborhood poverty on endogenous-cause and exogenous-cause infant deaths. Contrary to expectation, our findings suggest that neighborhood poverty has little independent effect on AI infant death outcomes. However, our study design makes quite transparent that social structure severely limits the pool of potential matches from the unexposed (counterfactual) group due to the characteristics of urban AI birth mothers who reside in low poverty neighborhoods. Collecting more data will not overcome the fact that AI with a high probability of living in poverty rarely reside in low poverty neighborhoods; and some of them must if we are to estimate meaningful neighborhood effects on health.

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Abbreviations

AI
: American Indian

ATT
: Average Treatment effect on the Treated

CI
: confidence interval

ICD (9, 10)
: International Classification of Diseases (Ninth Revision, Tenth Revision)

LBD
: Linked Birth/Infant Death data

SD
: standard deviation

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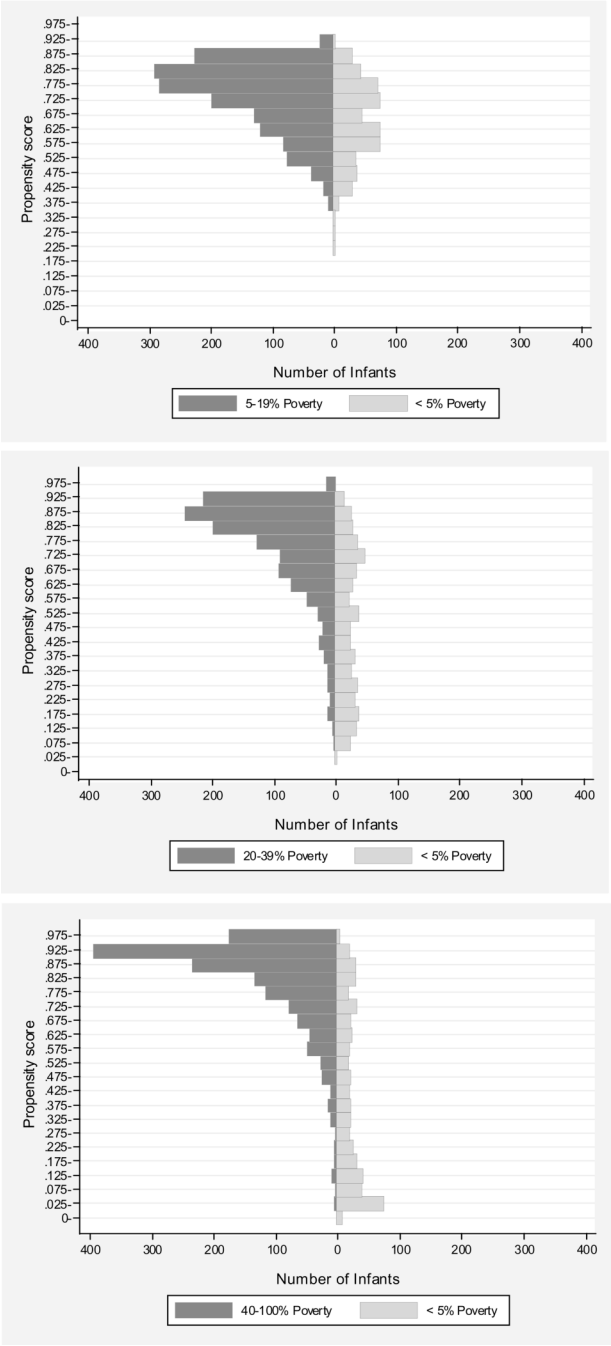

Figure 1.
Propensity score overlap by level of poverty for births to American Indian women residing in a 7-county metropolitan area, MN 1990 – 1999

Table 1

Selected Characteristics of births to American Indian women residing in a 7-county metro area by neighborhood tract poverty, Minnesota 1990 – 1999

	Neighborhood % Population Below Poverty				
	< 5% (n = 533)	**5–19% (n = 1,542)**	**20–39% (n = 1,261)**	**40–100% (n = 1,415)**	**Total (n = 4,751)**
Characteristics of Mother					
Maternal age					
Mean age, (sd)	25.50 (6.07)	24.07 (5.94)	23.74 (5.85)	23.91 (5.94)	24.10 (5.95)
Number of previous births					
Mean number now living, (sd)	1.10 (1.25)	1.34 (1.50)	1.67 (1.66)	2.08 (1.97)	1.62 (1.70)
Mean number now dead, (sd)	0.03 (0.19)	0.04 (0.23)	0.05 (0.25)	0.06 (0.27)	0.05 (0.24)
Number at this birth					
Singleton	524 (98%)	1,506 (98%)	1,239 (98%)	1,389 (98%)	4,658 (98%)
Twins	9 (2%)	36 (2%)	22 (2%)	26 (2%)	93 (2%)
Maternal education					
Adequate education for age	428 (80%)	1,045 (68%)	685 (54%)	650 (46%)	2,808 (59%)
Inadequate education for age	80 (15%)	433 (28%)	507 (40%)	685 (48%)	1,705 (36%)
Unknown	25 (5%)	64 (4%)	69 (5%)	80 (6%)	238 (5%)
Marital status					
Unmarried (single, cohabiting)	318 (60%)	1,189 (77%)	1,094 (87%)	1,265 (89%)	3,866 (81%)
Married	215 (40%)	353 (23%)	167 (13%)	150 (11%)	885 (19%)
Prenatal care utilization					
Intensive use	29 (5%)	75 (5%)	40 (3%)	36 (3%)	180 (4%)
Adequate use	114 (21%)	232 (15%)	137 (11%)	102 (7%)	585 (12%)
Medium use	250 (47%)	686 (44%)	550 (44%)	513 (36%)	1,999 (42%)
Inadequate use	38 (7%)	179 (12%)	228 (18%)	343 (24%)	788 (17%)
No prenatal care	6 (1%)	37 (2%)	42 (3%)	72 (5%)	157 (3%)
Unknown	96 (18%)	333 (22%)	264 (21%)	349 (25%)	1,042 (22%)
Smoked during pregnancy					
Yes	141 (26%)	532 (35%)	571 (45%)	686 (48%)	1,930 (41%)
No	339 (64%)	866 (56%)	582 (46%)	626 (44%)	2,413 (51%)
Unknown	53 (10%)	144 (9%)	108 (9%)	103 (7%)	408 (9%)
Characteristics of Father					
Paternal education					
Adequate education for age	300 (56%)	536 (35%)	254 (20%)	193 (14%)	1,283 (27%)
Inadequate education for age	37 (7%)	123 (8%)	105 (8%)	117 (8%)	382 (8%)
Unknown	196 (37%)	883 (57%)	902 (72%)	1,105 (78%)	3,086 (65%)
Race of father					
White	223 (42%)	376 (24%)	181 (14%)	114 (8%)	894 (19%)
American Indian	100 (19%)	322 (21%)	256 (20%)	336 (24%)	1,014 (21%)
Other race	47 (9%)	111 (7%)	66 (5%)	69 (5%)	293 (6%)
Unknown	163 (31%)	733 (48%)	758 (60%)	896 (63%)	2,550 (54%)
Characteristics of Infant					
Low birth weight	17 (3%)	119 (8%)	134 (11%)	114 (8%)	384 (8%)
Average birth weight	516 (97%)	1,423 (92%)	1,127 (89%)	1,301 (92%)	4,367 (92%)
Infant Death Outcomes					
Number (Rate per 1000 live births)					
All-cause infant death	5 (9.4)	25 (16.2)	22 (17.4)	33 (23.3)	85 (17.9)
Endogenous-cause death	3 (5.6)	12 (7.8)	13 (10.1)	17 (12.0)	45 (9.5)
Exogenous-cause death	2 (3.8)	13 (8.4)	9 (7.3)	16 (13.3)	40 (8.4)

Table 2

Effect estimates of neighborhood tract poverty on American Indian infant deaths by cause of death category, Minnesota 1990 – 1999

Causal contrast	Number of Matched Pairs	Infant deaths per 1000 births		ATT_{ratio} †	95% CI‡
		Exposed to poverty	Unexposed* to poverty (< 5%)		
Endogenous-cause					
5–19% vs. < 5%	1491	7.4	5.4	1.4	0.4, 16.0
20–39% vs. < 5%	1246	9.6	2.4	4.0	1.8, 19.0
40–100% vs. < 5%	1400	10.7	4.3	2.5	0.6, 19.0
Exogenous-cause					
5–19% vs. < 5%	1491	8.0	13.4	0.60	0.1, 2.8
20–39% vs. < 5%	1246	7.2	19.3	0.38	0.1, 4.0
40–100% vs. < 5%	1400	11.4	22.1	0.52	0.1, 12.5

*Unexposed (counterfactual) matches.

†ATT_{ratio} is a rate ratio representing the Average Treatment effect on the Treated.

‡95% bias-corrected confidence interval obtained from 1,000 bootstrap replications.