



## Original Contribution

# The Effect of Racial Residential Segregation on Black Infant Mortality

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Economic differences and proximal risk factors do not fully explain the persistent high infant mortality rates of African Americans (blacks). The authors hypothesized that racial residential segregation plays an independent role in high black infant mortality rates. Segregation restricts social and economic advantage and imposes negative environmental exposures that black women and infants experience. The study sample was obtained from the 2000–2002 US Linked Birth/Infant Death records and included 677,777 black infants residing in 64 cities with 250,000 or more residents. Outcomes were rates of all-cause infant mortality, postneonatal mortality, and external causes of death. Segregation was measured by using the isolation index (dichotomized at 0.60) from the 2000 US Census Housing Patterns. Propensity score matching methods were used. After matching on propensity scores, no independent effect of segregation on black infant mortality rates was found. Results show little statistical evidence that segregation plays an independent role in black infant mortality. However, a key finding is that it is difficult to disentangle contextual effects from the characteristics of individuals.

African Americans; infant mortality; prejudice; residence characteristics; risk factors

*Editor's note: An invited commentary on this article is published on page 000.*

While medical and societal advances have reduced infant mortality rates over the past 100 years, the disparity between African-American (herein referred to as black) and non-Hispanic white (herein referred to as white) infant mortality rates remains (1). Considerable research has been conducted trying to understand the etiology of poor black infant survival and the persistent disparity (2–20). Proximal risk factors contribute to the high black infant mortality rate but do not fully account for the racial differences.

One important risk factor for infant death is low birth weight (<2,500 g), which reflects both the infant's and mother's health. Blacks have twice the rate of low birth weight as whites (21–23). Yet, even among normal birth weight infants, black infant mortality rates are still twice as high as those for white infants (21–23). Proposed explanations for the persistent differences in infant mortality rate include income and education of the mother (24). However, infants born to college-educated blacks continue to have more than twice the infant mortality rate as those born to

college-educated whites (24, 25). It seems that other contextual factors must exist.

External causes of death, particularly during the postneonatal period, show the clearest causal path between harmful social and physical environmental exposures, such as racial residential segregation (herein referred to as segregation), and infant mortality rate. Although neonatal mortality, death in the first 27 days of life, is primarily attributed to biologic causes and is most clearly dependent upon the mother's prenatal exposures, postneonatal mortality, death at 28 days to 1 year, is historically attributed to environmental causes and infectious disease (26). Three specific causes of death during the postneonatal period are classified as "external" because of the etiology—sudden infant death syndrome, accident, and assault. Sudden infant death syndrome (27–29) is the third leading cause of infant mortality and the most common cause of postneonatal mortality. Blacks have rates of sudden infant death syndrome twice those of whites (21–23, 27, 29, 30). Accidental injury death (unintentional) is among the 10 most common causes of all infant mortality and is one of the top 3 leading causes of postneonatal mortality. Finally, compared with other racial groups, blacks have the highest rates of infant death due to homicide and

certain unintentional injury (31–33). Black assault (homicide) rates, the fifth leading cause of postneonatal mortality, are more than 3 times those for whites (34–37).

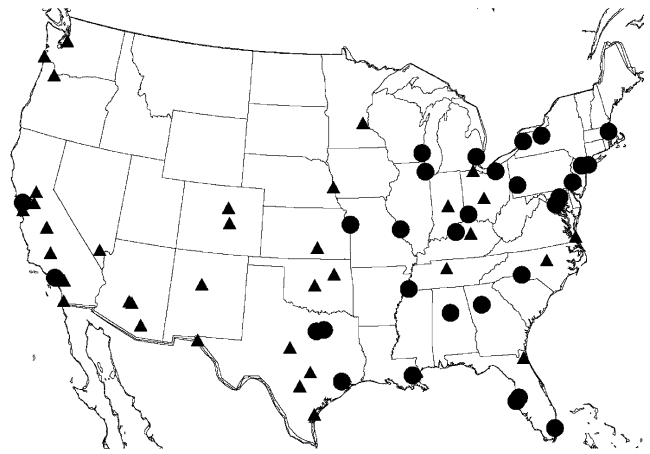
Risk factors for these external causes of death include demographics (race, marital status, socioeconomic status) (32, 33), low birth weight, prematurity, high parity, stomach (prone) sleeping position, soft bedding, and bed sharing (27, 29, 32, 38, 39). Several studies have focused on the social and physical environment to which black infants and mothers are exposed. Research suggests residual confounding by unmeasured social and cultural factors (33) or mediation by such factors as housing conditions (40). One hypothesis for the increased incidence of sudden infant death syndrome in the black community, especially the segregated community, is that community members are not receiving important health messages regarding sleep position because they are isolated from mainstream society (41). It was these hypotheses of differential social and environmental exposures (perhaps due to segregation) leading to infant death that we proposed to explore.

Recent literature suggests that segregation contributes to health disparities between blacks and whites because of the social, political, and economic disadvantage brought about by segregated communities (4, 41–44). Segregation is defined as the extent to which groups of individuals living in distinct areas within a region differ by race (45). The majority of blacks in the United States reside in residentially segregated communities, regardless of individual economic status (46). In fact, 80% of middle-class black households are located in predominantly black communities and within 4 blocks of impoverished neighborhoods. This proximity, regardless of individual economic status, may contribute to the higher rates of infant death for blacks living in areas with few contextual risk factors (4).

Our conceptual model of segregation's effect on infant mortality suggests that blacks, compared with whites, are differentially exposed to negative macrosocial conditions (neighborhood deterioration, lack of employment and educational opportunities, loss of tax base), social and material conditions (housing and air quality, limited social contacts, loss of community infrastructure), and individual-level factors (less physical activity, inadequate diet, lack of social support, stress, reduced access to medical care), placing infants at risk of dying because of the isolating effects of segregation (4–6, 10, 12, 13, 18, 20, 47, 48). Segregation reflects both the people who make up segregated communities and a theoretically larger toxic environment.

Although infant mortality has been studied extensively, we know of just 10 published articles on segregation and infant mortality (9–13, 16, 19, 20, 49, 50). All 10 concluded that segregation has a harmful association with infant mortality, varying from the Metropolitan Statistical Area to the borough level. The studies were all ecologic, with no ability to take into account individual characteristics. As our conceptual model above portrays, segregation reflects the individual characteristics and a larger contextual experience; thus, it is important to account for individual-level characteristics.

In this study, we asked whether there is a harmful, independent effect of segregation due to isolation on infant survival once individual-level characteristics are taken into



**Figure 1.** Study places by segregated (●) and nonsegregated (▲) status of residents, 2000 US Census Housing Patterns and Linked Birth/Infant Death records.

account. We used propensity score matching techniques to compare “exchangeable” infants born into segregated cities with those not born into segregated cities, thereby minimizing the effect of individual-level covariates. We then compared infant death outcomes between different levels of exposure to segregation.

## MATERIALS AND METHODS

Our data were derived from 2 sources: 1) Linked Birth/Infant Death records from the National Center for Health Statistics (outcome) (51) and 2) Housing Patterns from the US Census Bureau (exposure = segregation) (52). Linked Birth/Infant Death data are coded according to uniform coding specifications and are universally considered an accurate count of all infant births in the United States (53). The Linked Birth/Infant Death records are created by linking infant death records to birth records, with successful linkages made for nearly 99% of all infant deaths in 2000–2002 (21–23). Geographic identifiers are available in the Linked Birth/Infant Death data for cities with populations greater than 250,000 residents ( $n = 64$  (largely central) cities). Our segregation measure is at the level of *place*, or cities. *Place* is classified in both sources according to the Federal Information Processing Standards from the US Geological Survey, Geographic Names Information System (54). Linked Birth/Infant Death records were linked to city-level segregation data by Federal Information Processing Standards code, with 100% matching. Refer to Figure 1 for the location of study places by segregation status.

The target population was defined as infants born to black mothers residing in US places with a total population of more than 250,000 residents in 2000–2002 ( $n = 677,777$  livebirths). The exposure of segregation (isolation index) used for this study was calculated by the US Census Bureau using blacks compared with whites as the referent population (52). To calculate segregation measures, the US Census Bureau defines white as those who reported their ethnicity as

non-Hispanic and race as white *only*, whereas the minority population of interest was considered present if individuals classified themselves as that minority group or any combination of races that also included the minority population of interest (52). This distinction is particularly relevant for whites because residential patterns, socioeconomic, and health outcomes vary considerably between non-Hispanic whites and Hispanic whites. However, using the same criteria for blacks does not substantially change conclusions regarding residential pattern (55). Categorizing infant deaths by using the same criteria as the US Census Bureau does assures that the exposure measurement reflects the actual comparison group.

## Measures

Our outcome of interest was infant death. The infant mortality rate represents the number of infant deaths up to 1 year of life per 1,000 livebirths. The postneonatal mortality rate represents the number of deaths occurring between 28 days and 1 year per 1,000 neonatal survivors. External postneonatal mortality deaths are a subset and were classified according to *International Statistical Classification of Diseases and Related Health Problems*, Tenth Revision, code. We used code R95 for sudden infant death syndrome, codes V01–X59 for accidents, and codes X85–Y09 for assault. A separate analysis of neonatal mortality was not conducted because neonatal mortality is largely due to maternal factors. Although neonatal mortality accounts for two-thirds of the infant mortality rate, we analyzed overall infant mortality rate for comparison with previous ecologic studies.

Segregation is a multidimensional construct representing 5 geospatial patterns—dissimilarity, isolation, clustering, centralization, and concentration (56). Housing patterns, or segregation index scores representing these 5 domains, are calculated by the US Census Bureau for only those places with 10 or more census tracts, a population of 10,000 or more, and 100 or more of the minority population of interest. Acevedo-Garcia et al. (43) recommends multilevel analysis of the effect of segregation, and Polednak (48) recommends examining geographies smaller than the Metropolitan Statistical Area level (48). Therefore, city-level data were chosen as the smallest unit of analysis available. The measure of segregation for this study was the isolation index, which indicates that negative health outcomes for blacks may be due to the isolation from mainstream society and has the clearest pathway to health outcomes and social spaces to which infants are exposed (43, 57, 58). The isolation index is a weighted average for a given metropolitan area, ranging from 0 to 1, with 1 indicating higher isolation (52). It captures the extent to which blacks are surrounded by other blacks or nonblacks. Here, the index is highly correlated ( $r = 0.90$ ) with the more commonly used dissimilarity index. The isolation index was dichotomized at 0.60 to emphasize the extreme isolation experienced by blacks in those cities and is supported by previous sociologic work (10, 45, 56, 59). Sensitivity analysis was conducted, altering the cutpoint between 0.50 and 0.70, with little substantive difference noted.

Covariates that we expected to differ between levels of segregation and that occurred prior to the infant's exposure to segregation were used for the predictive model in propensity score matching methods. The intent was to approximate experimental design, balancing individual-level covariates that may confound the relation between segregation and infant mortality. The hypothetical experiment would be to randomly assign women to reside in segregated cities or not after giving birth and then observe the difference in infant deaths. To compare the 2 hypothetical populations, we examined covariate balance of the characteristics of mother and infant. As such, the covariates used in this observational study were mother's age (years), education group (0–8, 9–11, 12, 13–15, and  $\geq 16$  years of schooling), and marital status (unmarried or not); number of previous births and number of infants at this birth; prenatal care utilization (adequate, intermediate, inadequate); father's age (years); birth weight ( $\leq 2,500$  g or  $> 2,500$  g); gestational age ( $< 37$  weeks or  $\geq 37$  weeks); and year of delivery. Because postneonatal mortality and external causes of death are attributed to social and environmental conditions, matching on these covariates enables "control" of maternal factors along the infant's life course. Hot deck imputation was used for "unknown or not stated" response categories in the Linked Birth/Infant Death records for birth weight, prenatal care utilization, and maternal education because heterogeneity within those response categories was expected (26, 60). The missing values fall within the acceptable imputation range of less than 10% missing (60). Hot deck imputation is the most common method in demographic surveys, is used by the US Census Bureau for correcting item nonresponse, and is a relatively simple procedure to implement (61).

## Analytic approach

We used propensity score matching methods to improve exchangeability by balancing covariates across exposure groups (segregated and nonsegregated), thus reducing observable bias while maintaining support of the data (62, 63). The difference in disease frequency under the 2 exposure distributions is an estimate of the independent causal effect of segregation on black infant mortality (64).

For each infant, we used logistic regression to estimate the predicted probability of an infant's exposure to segregation given the observed covariates and interaction terms. We matched infants with the same predicted probability of exposure (i.e., propensity score) to segregation—only some were actually exposed and some were not—by using the PSMATCH2 module (65) of the Stata statistical software program (66). Since matching is the most exact method for balancing covariates (63), exposed infants were matched 1:1 with replacement to unexposed infants with the same predicted probability of exposure to segregation within a range of  $\pm 0.01$ . Standardized differences were calculated to assess covariate balance across exposure groups, with a goal of a standardized difference of less than 10% indicating sufficient balance (67). The estimated number of deaths that would have occurred among infants born in segregated places if instead they had been born in nonsegregated places

is the average effect of the treatment on the treated (65). Bootstrap methods were used to calculate appropriate standard errors. Sensitivity analysis was conducted by using Rosenbaum's test bounds (68) as a way to assess hidden bias.

## RESULTS

There were 677,777 black infants born between 2000 and 2002 in the 64 US places with a population greater than 250,000. A total of 9,003 black infant deaths occurred (overall infant mortality rate = 13.3 per 1,000 livebirths).

Table 1 displays the crude black infant mortality rates by maternal characteristics for the study population as a 3-year average. Twenty-nine places were classified as segregated (isolation index  $\geq 0.60$ ), and 35 places were classified as nonsegregated (isolation index  $< 0.60$ ). The mean percentage of the population living below the poverty level was 21.6% in segregated cities compared with 14.5% in nonsegregated cities. Of the total population, we found that 42.8% of residents in segregated cities are black compared with 12.2% in nonsegregated cities.

Propensity score matching yielded 100% matching of infants exposed to segregation to 1 control in nonsegregated cities. Table 2 shows covariate imbalance of select covariates as defined for the predictive model, level of imbalance prior to and after matching, and percentage bias reduction for all black infants. Because the sample size for this study was quite large and small differences may present as statistically different, we instead focused on the standardized difference. Only 1 covariate level was greater than 10% prior to matching (mother's education). All covariates were balanced ( $< 10\%$ ) after matching. Sensitivity analysis using Rosenbaum's test bounds (68) yielded tight confidence bounds around the log odds of differential assignment due to *unobserved* factors and the very small Hodges-Lehmann point estimate, revealing that unmeasured confounding was inconsequential.

There were 1.12 excess infant deaths per 1,000 livebirths among black infants due to living in a segregated city compared with a nonsegregated city, although the difference was not statistically significant. In fact, no statistical difference in the number of infant deaths per 1,000 livebirths was found for black infants residing in segregated cities compared with those born in nonsegregated places regarding overall infant death (rate difference = 1.12,  $Z = 1.35$ ,  $P = 0.178$ ), postneonatal deaths (rate difference = 0.99,  $Z = 1.63$ ,  $P = 0.103$ ), or external causes of death (rate difference = 0.53,  $Z = 1.39$ ,  $P = 0.164$ ). Refer to Table 3 for the rate difference in the number of infant deaths per exposure category per 1,000 livebirths.

## DISCUSSION

We estimated the independent effect of living in a segregated place on black infant mortality, focusing on the postneonatal period. We hypothesized that segregation has a harmful effect on black infant survival due to macrosocial, social and environmental, and individual-level risk exposures. However, we found limited statistical evidence that

**Table 1.** Infant Mortality Rates per 1,000 Livebirths for US Black Infants by Maternal and Infant Characteristics, Linked Birth/Infant Death Records, 2000–2002

Characteristic	Black
Infant mortality	13.3
Postneonatal mortality	4.7
Sex	
Male	14.8
Female	11.7
Plurality	
Single	11.9
Multiple	51.2
Birth weight, <sup>a</sup> g	
<2,500	71.8
$\geq 2,500$	4.3
Period of gestation, weeks	
<32	193.4
32–36	11.2
37–41	4.6
$\geq 42$	5.4
Trimester that prenatal care began <sup>a</sup>	
First	12.7
After the first	10.4
No prenatal care	51.0
Age of mother, years	
<20	14.2
20–24	12.7
25–29	13.0
30–34	13.1
35–39	13.9
40–54	16.1
Educational attainment of mother, <sup>a</sup> years	
0–8	14.3
9–11	15.0
12	13.3
13–15	12.2
$\geq 16$	11.2
Livebirth order	
1	13.0
2	11.6
3	12.6
4	14.6
$\geq 5$	18.6
Unknown	50.3
Marital status	
Married	10.9
Unmarried	14.2

<sup>a</sup> Includes imputed values.

**Table 2.** Select Covariate Imbalance Across Exposure Groups Prior to and After Matching for All US Black Study Infants, 2000–2002

	Racial Residential Segregation, mean	Mean Value Prior to Matching	Mean Value After Matching	Standardized Difference Prior to Matching	Standardized Difference After Matching	% Bias Reduction
Mother's age	25.487	25.067	25.377	6.8	1.8	73.9
Total birth order	2.998	2.960	2.891	1.3	3.8	–183.3
Livebirth order	2.235	2.258	2.195	–1.8	3.1	–71.6
Maternal education, years						
0–8	0.289	0.028	0.025	0.6	32.4	–311.7
9–11	0.254	0.221	0.252	7.7	0.5	93.5
12	Reference					
13–15	0.217	0.240	0.219	–5.4	–0.5	90.7
≥16	0.103	0.107	0.104	–1.5	–0.4	70.0
Adequate prenatal care						
Adequate	Reference					
Intermediate	0.259	0.239	0.255	4.5	0.9	80.7
Inadequate	0.115	0.093	0.111	6.9	1.2	82.6
Unmarried	0.742	0.663	0.750	17.5	–1.7	90.2
Birth weight, g						
<2,500	Reference					
≥2,500	0.866	0.871	0.876	–1.5	–3.1	–105.8
Gestational age						
Less than normal	0.177	0.168	0.167	2.5	2.7	–4.8
Normal	Reference					

segregation independently caused excess deaths of black infants when comparing exchangeable populations of black infants born in segregated and nonsegregated cities.

Our descriptive findings confirm that blacks have a high infant mortality rate regardless of maternal characteristics. Compared with nonsegregated cities, segregated cities, as defined in this study, innately have higher populations of black residents but also have a higher number of residents living in poverty. A vast literature describes the effects of poverty on infant survival and the joint relation between segregation and poverty (4, 42, 47, 57, 69–72). Economic deprivation often accompanies segregation, accentuating the common mechanisms of each. Middle-class blacks are not immune to the joint effect because the majority of middle-class black households are located within 4 blocks of impoverished neighborhoods and thus are still exposed to degrees of deprivation (4, 42, 47, 57, 69–72).

Our findings contradict previously published research on racial residential segregation and infant mortality (9–13, 16, 19, 20, 49, 50). Yankauer's landmark study in 1950 (13) demonstrated the steady and significant increase in nonwhite infant mortality as the proportion of nonwhite infant births per area increased. In a follow-up study, he found that, although infant mortality rate declined overall, the disadvantage for the nonwhite population remained unchanged and perhaps worsened (12).

After a gap of nearly 40 years, LaVeist (19, 20, 49) conducted a series of studies focused on 176 cities with populations greater than 50,000 residents and 10% or more black residents using the dissimilarity index. His studies consistently found that segregation was positively associated with black infant mortality rate and postneonatal mortality rate after adjusting for socioeconomic factors and geographic region. Polednak (10, 11, 48) conducted

**Table 3.** Independent Effect of Racial Residential Segregation on Black Infant Deaths per 100,000 Livebirths, US Cities With a Population >250,000, 2000–2002

	Rate Difference	SE	Z	P Value	95% CI
Infant mortality	1.12	0.83	1.35	0.178	–0.51, 2.74
Postneonatal mortality	0.99	0.60	1.63	0.103	–0.20, 2.17
External causes of death	0.53	0.38	1.39	0.164	–0.22, 1.27

Abbreviations: CI, confidence interval; SE, standard error.

3 studies by using standard Metropolitan Statistical Areas and the dissimilarity index. Segregation was found to have an independent association with infant mortality and was the most important predictor of infant mortality. Bird (8) assessed whether state-level structural variables modify the relation between states' infant mortality for blacks and whites and found that racial composition, education, poverty, and segregation contributed to black infant mortality at the state level. Finally, Guest et al. (16) looked at both infant mortality and working-age mortality in Chicago, Illinois, by using the isolation index and found that segregation predominantly increases black mortality through differential socioeconomic patterns but also appears to have an independent "deleterious" effect.

These previous studies contributed to the scientific literature, but all were ecologic. Ecologic study designs may suffer the ecologic fallacy (73). Our use of individual-level covariates advanced the work in this area and may account for the different results. Our use of propensity score matching methods offers a potentially more rigorous design. We also conducted a comparable analysis by using traditional logistic regression and found no significant difference in infant mortality rate between blacks living in segregated and nonsegregated cities. The value of propensity score matching methods is that it makes explicit the exchangeability between comparison groups. Additionally, to our knowledge only 1 study (16) previously used the isolation index. The choice of segregation dimension may matter in health research (43).

We do not claim that segregation has no effect on the black infant mortality rate but rather that it is difficult, if not impossible, to disentangle the individual effects from contextual effects. Our results suggest that the effects of segregation as defined here are not larger than the characteristics of the individuals when comparing exchangeable populations or that they are not independent from the individuals who live in the segregated cities. There is no group without the specific individuals who constitute it (74), and distinct categories of contextual versus compositional effects themselves are contrived distinctions (75). What differentiates a segregated city from a nonsegregated city is the cumulative effect of the people and the policies, both formal and informal, that are in place.

There are limitations to our approach. The choice of covariates predicting segregation may be on the causal path, and separating the characteristics of the mother and infant ignores a life course perspective. We attempted to address both of these issues by focusing primarily on postneonatal mortality, which is more distal to conditions of birth. Measurement of segregation, as with many social measures, is a function of the cutpoints chosen, the measurement tool used, and the assumptions of the meaningfulness of segregation at and near the cutpoints. Another limitation is restriction of the analysis to the city level. It could be that the relation between segregation and birth outcomes varies depending on the size of the geographic area and number of residents. Patterns of segregation are seen at the Metropolitan Statistical Area level, resulting in possible selection bias because urban dwellers may differ from suburban dwellers. At the same time, the city-level segregation may

not accurately reflect the experiences of blacks within the city. Analysis at the census tract level may provide a different estimate of individual-level exposures. We were unable to account for heterogeneity of exposure, both within city and over time. We were able to account for the city in which the infant was born but not if that infant then moved, possibly altering the infant's social and environmental exposures.

Finally, propensity score methods also have limitations. Even though they enabled us to more closely mimic experimental design, minimize emphasis on *P* values for model specification, and make inference more transparent than traditional regression techniques do, the 2 most important limitations are unmeasured confounding and poor overlap. Propensity score methods do not account for unmeasured confounding, as would perfect randomization. There is no direct way to test unmeasured confounding, but our sensitivity analysis showed it to be largely inconsequential here. Subjects without a match, no overlap or propensity scores, were dropped from the analysis, leading to selection bias. Despite these limitations, our data were of high quality, measurement of outcomes was reliable, and the analytic approach was rigorous. This study offers an important advancement to both methods and the understanding of contextual effects on health.

In conclusion, future work is needed in many areas, including understanding the effect of segregation at different geographic levels (i.e., Metropolitan Statistical Area), further exploring the mechanisms, understanding the experience and exposures of black women and infants in nonsegregated areas, and incorporating historical migratory patterns in the making of segregation and its effect on health. The infant mortality rate continues to require public health and policy attention. Although this study suggests no *independent* effect of racial residential segregation on black infant mortality rate, we emphasize the difficulties separating composition from context and cautious interpretation.

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