Research Article

Examining the Racial Crossover in Mortality between African American and White Older Adults: A Multilevel Survival Analysis of Race, Individual Socioeconomic Status, and Neighborhood Socioeconomic Context

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We examine whether individual and neighborhood socioeconomic context contributes to black/white disparities in mortality among USA older adults. Using national longitudinal data from the Americans’ Changing Lives study, along with census tract information for each respondent, we conduct multilevel survival analyses. Results show that black older adults are disadvantaged in mortality in younger old age, but older black adults have lower mortality risk than whites after about age 80. Both individual SES and neighborhood socioeconomic disadvantage contribute to the mortality risk of older adults but do not completely explain race differences in mortality. The racial mortality crossover persists even after controlling for multilevel SES, suggesting that black older adults experience selective survival at very old ages. Addressing the individual and neighborhood socioeconomic disadvantage of blacks is necessary to reduce mortality disparities that culminate in older adulthood.

1. Introduction

Black older adults (ages 65+) have higher all-cause mortality rates than white older adults in the USA [1, 2]. A growing body of literature attempts to understand the mechanisms explaining these persisting race differences in mortality [3–5] so that we can understand how such race disparities might be reduced or eliminated. Substantial disparities in individual socioeconomic status (SES) by race have been observed in the USA, and research has shown that individual SES (e.g., income and education) partly mediates but does not eliminate the relationship between race and mortality [6–8]. In addition, neighborhood context has been identified as a key factor that contributes to race disparities in morbidity [9–11]. However, few studies have examined how neighborhood context contributes to race difference in mortality [12, 13], particularly at older ages.

The current study employees mixed effect survival analysis to investigate whether SES, measured at both the individual and neighborhood levels, explains black/white differences in mortality among older adults in the USA, using longitudinal national data.

A number of theoretical perspectives, including economic deprivation [14] and social disorganization theory [15, 16], suggest that neighborhood context is associated with health and mortality. Some studies have shown that living in a poor neighborhood is related to increased cause-specific mortality among adults [17–20], after controlling for individual SES variables. However, other studies have shown that there is no significant association between neighborhood context and all-cause mortality for older adults [11, 21, 22].

Very few studies have examined the contribution of neighborhood socioeconomic context to race disparities
in mortality for older adults, although some research has examined this question for younger and middle-aged adults. LeClere and colleagues found that the differential all-cause mortality rates between African American and non-Hispanic white men and women aged 18 and older were partly explained by individual-level SES and were further explained by neighborhood context. The differences in mortality between the black and white adults were completely explained by both individual SES and neighborhood combined [23]. However, some studies indicate that race differences in mortality persist among USA adults even after both individual SES and neighborhood context are controlled. For example, one study demonstrated that mortality for all racial/ethnic groups is related to individual SES and to neighborhood characteristics, among people aged 18–64. The race difference in mortality persisted after controlling for both individual and neighborhood level SES [24].

With regard to older adults, LeClere and colleagues showed that the percentage of female-headed families in the neighborhood was associated with heart disease mortality for women aged 65 and above. Individual SES and neighborhood context fully accounted for the heart disease mortality disparity between white and black older adults [12]. The above research provides some knowledge about the relationships among race, multilevel SES, and all-cause mortality, but the results are not consistent.

Moreover, this body of research has not fully considered the shape of the relationship between race and mortality at older ages. However, research suggests that there is a racial mortality crossover at older ages such that black older adults have higher mortality than white older adults in young older ages while white older adults have higher mortality than their black counterparts in very late old age [25–30]. It could be that the racial mortality crossover in late old age contributes to the inconsistent conclusions regarding the role of multilevel SES in explaining race difference in mortality among older adults. This is because if crossover effects exist, the effect of race on mortality for a younger subgroup will be positive while the effect of race will be negative for an older age subgroup. Combining age groups together in one “old age” group ages 65+ may result in the positive and negative effects canceling each other out.

There are two primary competing explanations for racial mortality crossover effects [27]. If black older adults are more likely to misreport their age as older than it really is, this would result in more young black older adults being categorized in the oldest old groups. Alternately, black older adults may experience selective mortality. Since black adults are more likely to die at younger ages, the black survivors at very older age should be very robust. Unfortunately, it is difficult to detect age reporting errors in survey data. In any case, the potential existence of the racial mortality crossover effect means that, when studying race differences in mortality among older adults, it is necessary to consider the interaction of race and age.

There have been theoretical debates on the expected patterns of race disparities in mortality over the life course. The double jeopardy hypothesis suggests that blacks are faced with a double burden on health with aging and therefore should experience worse health status and mortality compared to whites at older ages [31]. Another perspective hypothesizes persistent inequality—that race differences in health are set at earlier ages and are then relatively stable throughout the aging process [32]. These two perspectives suggest that crossover effects should not exist. A third perspective describes a different story in which some factors that affect health at younger ages are not as strongly associated with changes in health at older ages. In particular, social factors may be less important than biological factors at older ages. For example, the research by House et al. [33] suggests that the relationship between SES, other risk factors, and health may be buffered by biological robustness (in early adulthood), biological frailty (in later old age), or the existence of social welfare programs, particularly at older ages. This third perspective suggests that the race difference will reduce or even crossover at older ages.

Empirical studies provide some support for the third perspective and have shown that the health disadvantage of blacks not only disappears but crosses over such that black older adults have lower mortality ratios at very old ages (around age 80) [27, 34, 35]. If this crossover effect happens, it may obscure the true relationships among race, multilevel SES and mortality among older adults in different age groups. Given this consideration, we examine the potential for a race crossover effect in mortality among older adults and reexamine the contribution of multilevel SES to mortality and its variation by age among older adults.

Our study extends prior work in a number of ways. The existing literature has contrary findings on the effects of neighborhood context on mortality and the magnitude of the effects of neighborhood context on explaining race differences in mortality among older adults. A number of limitations to prior work may cause this contradiction. First, as described above, most studies examine all older adults combined, rather than examining age-specific changes among older adults. Second, most studies examined disease-specific mortality, but it is also important to understand how multilevel SES is associated with all-cause mortality. Third, most previous studies used a single indicator or separate indicators of neighborhood context, whereas we combine a number of neighborhood variables to create a more comprehensive neighborhood disadvantage index.

Fourth, our statistical approach represents an improvement on prior work in the USA because we use multilevel survival analyses to account for the multilevel nature of the data while modeling mortality using the greater precision of survival analysis. Most prior work in the USA has not adjusted for the complex survey design effects that result from the multistate sampling method of the data or the nested nature of the individual-level data within neighborhoods, leading to potentially incorrect variance and parameter estimates [36–38]. Some studies considered the correlated data structure and used multilevel logistic regression to estimate the association between multilevel SES and mortality [11, 22, 39, 40]. But in these studies, censoring issues were not addressed leading to potential errors in estimates. Recently, a few studies employed multilevel survival
analysis, which combines the advantages of mixed effects model and Cox proportional hazard model to estimate the effects of multilevel SES on cause-specific mortality [17–19, 41] and all-cause mortality [42]. However, these studies were based in European countries. We utilize this method using a USA sample.

In sum, results regarding the effects of neighborhood SES on mortality and its contribution to race differences in mortality among older adults are mixed. Some national studies provided evidence that multilevel SES is associated with mortality and contributes to explaining race differences in mortality [12, 23, 24, 43]. However, there is still a debate on the magnitude of their contribution to race differences in mortality, particularly at older ages. We build from and attempt to improve upon prior work by introducing an interaction between race and age. We also use multilevel survival models to appropriately estimate the associations between race, the interaction of race and age, individual SES, neighborhood SES, and all-cause mortality over 16 years among older adults in a nationally representative sample of adults in the USA.

2. Materials and Methods

2.1. Data. Wave 1 (W1) of the Americans Changing Lives (ACL) study was conducted in 1986 through face-to-face interviews in the homes of 3,617 adults. The sample was created using a multistage, stratified area probability sample (ACL) study was conducted in 1986 through face-to-face interviews in the homes of 3,617 adults. The sample was based in European countries. We utilize this method with 16+ years schooling and mean family income were reversed) to create the Neighborhood SDI. A higher score means greater neighborhood disadvantage.

Gender is coded 1 for men and 0 for women. Race is a dummy variable (Black = 1; White = 0).

The dependent variable at each wave is all-cause mortality. Mortality was tracked over time and was confirmed with the National Death Index (coded to the year of death) through wave 4 (2001) [33].

2.3. Statistical Analysis. Survival analysis is used to examine predictors of time until death. The basic Cox proportional hazards regression is expressed as [46, 47]

\[
h(t | Z) = h_0(t) \exp \left( \sum_{k=1}^{p} \beta_k Z_k \right). \tag{1}
\]

\[h(t | Z)\] is the hazard rate at time \(t\) for an individual covariates vector \(Z\). \(h_0(t)\) is an arbitrary baseline hazard rate. \(\beta_k\) is the estimated parameter vector that represents the direction and magnitude of the association between \(Z_k\) and \(h(t | Z)\) compared to baseline hazard rate. If we assume that censoring time and event for the \(j\)th participants are independent given by \(Z_k\), the parameters in (1) could be estimated by maximizing the partial likelihood function in

\[
L(\beta) = \prod_{i=1}^{D} \exp \left[ \sum_{j \in R(t_i)} \frac{\sum_{k=1}^{p} \beta_k Z_{ij}}{\sum_{k=1}^{p} \beta_k Z_{jk}} \right]. \tag{2}
\]

In (2), let \(t_1 > t_2 \ldots < T_D\) denote the ordered event times. \(Z_{ijk}\) is the \(k\)th covariate associated with individual whose death time is \(t_i\), \(R(t_i)\) represents the \(k\)th covariate associated with individuals who are still in the risk set at a time just prior to \(t_i\). The numerator in (2) represents the information about death where the denominator includes all information about individuals who have not yet died.

When neighborhood socioeconomic context is included in analysis, we model the association between individual survival times within neighborhood. There is an unobservable random effect shared by subjects within a neighborhood. In this case, Cox proportional hazard mixed effects model (PHMM) is appropriate [46, 48, 49]. To simplify the model, one time-invariant neighborhood socioeconomic context variable is included in the analysis. The hazard function for the \(j\)th individual in \(i\) neighborhood can be expressed as [50, 51]

\[
h_{ij}(t | Z) = h_0(t) \exp (\beta Z_{ij} + b_i \omega_i). \tag{3}
\]

\(Z_{ij}\) is the vector covariate and \(\beta\) is the vector of regression coefficient. \(\omega_i\) is a vector of covariates that have random effects. This equation captures the random effects of the cluster and enables covariate by cluster interactions. Due to the interaction of clustered and individual covariates, \(\omega_i\) is a subset of \(Z_{ij}\) [51, 52].
on mortality are presented in Table 2. Model 1 in Table 2 shows that black older adults had a higher mortality rate compared to the norm [53, 54]. For example, the variance is 0.0051 in Model 4. Its standard error is 0.07. This suggests that an individual neighborhood has an average 7 percent higher or lower mortality level compared to the norm. This relatively large variation between neighborhoods suggests that it is appropriate to model neighborhood-level variance in mortality analyses among older adults.

### Table 1: Descriptive statistics (unweighted): percentage distribution or mean (standard deviation in parentheses) at baseline for independent variables (respondents aged 65 and above at baseline, N = 1211).

<table>
<thead>
<tr>
<th>Variables</th>
<th>Range</th>
<th>Percentages or mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (%)</td>
<td>0-1</td>
<td>31.23</td>
</tr>
<tr>
<td>Black (%)</td>
<td>0-1</td>
<td>28.98</td>
</tr>
<tr>
<td>Age (years)</td>
<td>65–96</td>
<td>73.13 (6.34)</td>
</tr>
<tr>
<td>Income (Dollars)</td>
<td>1–10</td>
<td>3.18 (2.16)</td>
</tr>
<tr>
<td>Education</td>
<td>0–17</td>
<td>9.99 (3.75)</td>
</tr>
<tr>
<td>Neighborhood SDI</td>
<td>−3.22–3.26</td>
<td>0 (1)</td>
</tr>
</tbody>
</table>

Neighborhood SDI: Neighborhood Socioeconomic Disadvantage Index.

2.4. Analytic Strategy. A series of Cox proportional hazard models will be presented to examine the association between race, multilevel SES, and mortality. Models 1 to 4 in Table 2 use Cox proportional hazards regression to estimate the contribution of both individual SES and neighborhood SDI to race differences in mortality when neighborhood-level variance is ignored. Mixed effect Cox analyses are shown in Models 1b to 4b in Table 2 in order to examine how multilevel SES explains race disparity in mortality when we consider the neighborhood-level variance. Finally, the interaction between race and age is introduced in Models 5 to 7 to investigate whether race differences in mortality differ by different age groups after multilevel SES is controlled.

3. Results

Table 1 presents the descriptive information for the demographic and socioeconomic measures at baseline for 1211 older adults aged 65 and above who reported their race as either white or black. About one third of participants were male, about 45% were married at baseline, and the average age was 73. The mean years of education are about 10. The average family income was in the range of $15,000 to $19,999 in 1986.

The effects of individual SES and neighborhood SDI on mortality are presented in Table 2. Model 1 in Table 2 shows that black older adults had a higher mortality rate (e**0.0167 = 1.18) than white older adults. The probability of dying (at an earlier age or by the end of the study period) for black older adults is on average 18% higher than the probability of death for white older adults. When only individual SES measures were added in Model 2, race differences in mortality disappeared and family income is negatively related to mortality. Model 3 examines the effects of neighborhood SDI on mortality. First, we find that neighborhood SDI is positively associated with mortality. This model suggests that, if the neighborhood socioeconomic disadvantage index increases by one standard deviation, the probability of death increases by 10% (e**0.091 = 1.0953). Race differences in mortality persist after controlling for neighborhood SDI. Both individual SES and neighborhood SES were included simultaneously in Model 4. Model 4 indicates that both family income and neighborhood SDI are significantly related to mortality. However, there is no remaining statistically significant race difference in mortality.

Models 1b–4b in Table 2, using mixed effects Cox model, demonstrate similar results. Both individual SES and neighborhood SDI are related to mortality. Race differences in mortality are fully mediated by individual SES. Looking at Models 2–4 and 2b–4b together, we conclude that individual SES is stronger than neighborhood SDI in explaining race differences in mortality.

The interaction of race by age was added in Models 5–7 in Table 2 in order to investigate whether race disparities in mortality vary by age among older adults. Model 5 indicates that there is a significant interaction of race and age, which means that race differences in mortality are not constant across age for older adults. The positive coefficients for race and age and the negative coefficient for the interaction of race and age suggest that there is a diminishing or crossover effect of race rather than theories suggesting a double jeopardy hypothesis. The positive coefficient of race suggests that black adults experienced a greater risk of death at young old ages. The negative coefficient of the interaction of race and age shows that black older adults’ risk of mortality increases at a slower rate than white older adults’ risk. We compute the turning point age where black and white older adults have an equal risk of mortality after controlling other covariates in Model 5. The formula is 3.028*1 + 0.106*age – 0.038*age = 3.028*0 + 0106*age – 0.038*0 and we get age = 79.68. This means that after around age 80, black older adults had lower risk of dying than whites.

In Model 6, we include individual SES measures to examine their effects on mortality and their contribution to race differences in mortality by age. First, as in Models 1–4 and Models 1b–4b, family income is negatively associated with mortality. Second, including individual SES measures reduced but did not fully explain race differences in mortality by age.

Neighborhood SDI is added in Model 7. We see that living in a neighborhood with greater disadvantage is associated with a greater risk of dying (e**0.086 = 1.09), net of demographic, and individual SES variables, and it further reduces the association between race and mortality by age. Again, there remains a significant racial crossover effect on mortality due to the positive coefficient of race and negative coefficient of the interaction of race and age. The turning point of the crossover is about 74 after controlling for SES at multiple levels.

Finally, all variances of neighborhood tracts are significant in Models 1b to 4b and Models 5 to 7. The standard error presents how much an individual neighborhood varies in its mortality rate compared to the norm [53, 54]. For example, the variance is 0.0051 in Model 4b. Its standard error is 0.07. This suggests that an individual neighborhood has an average 7 percent higher or lower mortality level compared to the norm.
The second debate is whether individual SES and neighborhood SES help explain race differences in mortality for older adults. Although only very few studies have explicitly examined this issue, the results have been inconsistent. Some studies showed that individual SES fully mediates the relationship between race and mortality, while other studies found that individual SES helped explain the difference with neighborhood context further explaining the difference. However, none of these prior studies considered or modeled the race crossover effect of mortality between black and white older adults.

In this study, we examined the contribution of individual SES and neighborhood SDI to mortality through two sets of analysis—with and without modeling a racial crossover effect. The analysis without modeling a potential crossover effect showed that individual SES fully mediated the relationship between race and mortality. However, the analysis with the interaction term between age and race demonstrated that there is a race crossover effect and further indicated that race differences in mortality persist after both individual SES and neighborhood SDI were controlled. Our analysis suggests that the crossover effect happens around ages 76–80, which is consistent with previous studies [27, 34].

There are a number of implications of this study. First, our study suggests that age reporting bias is probably not the main reason for the racial crossover in mortality. This is because, even if more young black older adults were falsely categorized into the oldest old groups, social factors still exert a large effect on race difference in mortality since significant race differences in mortality exist between blacks and whites at early older age. At the oldest ages, the aging process itself, especially biological factors, may have more weight on mortality and health [33]. Given our data and findings, our results are consistent with a selective mortality explanation for the racial crossover in mortality at later old age for black and white older adults. The selective survival of robust black older adults likely explains the racial crossover.

Another important finding is that our analyses reveal that individual SES explains more of the race differences in mortality than does neighborhood SDI. This is consistent with previous studies [13]. However, we must keep in mind that some of the neighborhood effects on mortality may work through their effects on individual-level income
over the life course. Actual neighborhood effects are likely underestimated when individual-level income is controlled. There are a number of limitations to our study. First, we only include white and black older adults in our analysis due to sample limitations of racial/ethnic distribution in the ACL data. Future studies should examine how individual and neighborhood socioeconomic context contribute to racial/ethnic disparities in mortality between other racial/ethnic groups. Second, we only include baseline individual SES and neighborhood SDI variables in the analysis. Using these static measures may underestimate their effects on mortality and in explaining race disparities in mortality [55]. Examining how dynamic individual SES and neighborhood SDI measures affect mortality over time for older adults is an interesting and challenging research direction for the future.

Third, we limited our analysis to the cohort of older adults aged 65 and above. It may be interesting to compare the results with other age cutoffs and cohorts. Cohort is an important concept in life course theory [56], which expects that people born at a particular time (cohort effect) may experience similar life events that will affect their life path, SES, health, and mortality [57]. For example, the type and degree of racial discrimination experienced may differ by cohort, affecting race differences in the accumulation of individual-level SES and neighborhood segregation. Testing cohort differences in future research could enrich theory and evidence about how multilevel SES contributes to race difference in mortality.

5. Conclusion

Our study extends previous research and contributes to the literature in two major ways. First, we use appropriate statistical methods to estimate the association between multilevel socioeconomic status (at individual and neighborhood levels) and mortality and confirm that older adults living in a disadvantaged neighborhood context experience higher risk of dying at earlier old ages, beyond the impact of individual SES. Second, we demonstrate that there are race crossover effects in mortality at later old age, with black older adults having a mortality advantage at later old age. Moreover, neighborhood SDI helps explain race differences in mortality at older ages. Finally, race differences in mortality did not disappear even after controlling for both individual SES and neighborhood SDI measures once we modeled the racial crossover effect on mortality.

These results help resolve debates in previous studies and help us better understand the association among race, individual SES, and neighborhood context. The socioeconomic contexts that affect black and white Americans into old age affect their mortality risk, leading to selective survival among the most robust black older adults at later older ages. Addressing the individual and neighborhood socioeconomic disadvantage of black people over the life course is necessary to reduce mortality disparities that culminate in early older adulthood.

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