



# Can Early Disadvantage Be Overcome? A Life Course Approach to Understanding How Disadvantage, Education, and Social Integration Impact Mortality into Middle Adulthood Among a Black American Cohort

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## Abstract

Health equity research has identified fundamental social causes of health, many of which disproportionately affect Black Americans, such as early life socioeconomic conditions, neighborhood disadvantage, and racial discrimination. However, the role of life course factors in premature mortality among Black Americans has not been tested extensively in prospective samples into later adulthood. To better understand how social factors at various life stages impact mortality, this study examines the effect of life course poverty, neighborhood disadvantage, and discrimination on mortality and factors that may buffer their effect (i.e., education, social integration) among the Woodlawn cohort ( $N=1242$ ), a community cohort of urban Black Americans followed since 1966. Taking a life course perspective, we analyze mortality data for deaths through age 58 years old, as well as data collected at ages 6, 16, 32, and 42. At age 58, 204 (16.4%) of the original cohort have died, with ages of death ranging from 9 to 58.98 (mean = 42.9). Cox proportional hazard models adjusting for confounders show statistically significant differences in mortality risk based on timing and persistence of poverty; those who were never poor or poor only in early life had lower mortality risk at ages 43–58 than those who were persistently poor from childhood to adulthood. Education beyond high school and high social integration were shown to reduce the risk of mortality more for those who did not experience poverty early in their life course. Findings have implications for the timing and content of mortality prevention efforts that span the full life course.

**Keywords** African Americans · Educational attainment · Health disparities · Life course perspective · Premature mortality · Social roles · Urban health

## Introduction

While the gap in mortality statistics between Black and White Americans has shrunk since 1999 (Cunningham et al., 2017; Kochanek et al., 2015), Black Americans still have a life expectancy that is 3.9 years shorter than their White peers (Arias & Xu, 2020), and experience 1.5 times the premature death as White Americans (Shiels et al., 2017). These higher death rates among Black Americans warrant particular attention to the predictors of mortality in order to identify malleable risk and protective factors across the full

life course as the cause of death shifts dramatically with age for Black Americans. Injuries and homicide contribute the most deaths among Black children, and suicide peaks as the third leading cause of death between ages 15 and 24 (Heron, 2021); however, as Black Americans move into adulthood, physical health conditions outpace unintentional deaths and other harm. HIV appears as a top ten cause of death between ages 20 and 54 (Heron, 2021), matching national statistics that show the incidence of HIV is highest among Black adults (Centers for Disease Control and Prevention, 2021). By mid-adulthood (ages 45–64), the leading causes of death for Black Americans are heart disease and cancer, together representing approximately half of all deaths among that age group, followed by unintentional injuries/accidents, diabetes, stroke, kidney and liver disease, and chronic lower respiratory disease (Heron, 2021). Cancer and heart disease

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remain the leading causes of death through later adulthood, and hypertension and acute conditions (influenza, pneumonia, septicemia) also enter the top ten causes for Black Americans. As types of death vary developmentally, it is critical for research to take a life course perspective to inform the timing and content of preventive efforts.

## Social Determinants of Mortality Risk

Socioeconomic status (SES) is purported to play a central role in these mortality-related disparities. The importance of SES as a key driver in health disparities is grounded in the fundamental cause theory (Link & Phelan, 1995; Phelan et al., 2004). This perspective posits that macro-level processes shape social conditions that in turn shape access to economic, social, and political resources that fundamentally impact health risk across populations. Higher-SES individuals have access to knowledge, money, power, and social connections that create a health advantage over lower-SES individuals (Link & Phelan, 1995). While economic factors lie at the foundation of fundamental cause theory, these fundamental causes are more complex for minority populations whose social structural position is also determined by race/ethnicity. Recent extensions of the fundamental cause theory propose that structural racism is a direct fundamental cause of health outcomes through its influence on discrimination, residential segregation, and neighborhood disadvantage, which all produce differential health and mortality risks (Clouston & Link, 2021; Gee & Ford, 2011; Phelan & Link, 2015).

There is considerable overlap between SES and race—Black Americans are twice as likely than White Americans to be poor (Pew Research Center, 2016) and more often live in neighborhoods characterized by high rates of poverty, segregation, and single-mother households and low rates of college education and home ownership (Firebaugh & Acciai, 2016; Ross & Mirowsky, 2001). White households are about 13 times as wealthy as Black households (Pew Research Center, 2016). Due to racial segregation, higher-income and college-educated Black Americans have historically lived in neighborhoods more similar to those of their lower-income, poorly educated White American counterparts (Erbe, 1975; Perkins & Sampson, 2015). Since the 1970s, close to 40% of Black Americans have been in the poorest 20% of the income distribution in the USA, with less than 10% in the highest quintile; the vast majority of Black Americans (62%) compared with White Americans (4%) are raised in neighborhoods with at least 20% of its residents living in poverty (Sharkey, 2013). Moreover, upward mobility is less common among Black Americans with only one-third moving out of the poorest neighborhoods in the USA within one generation, compared to the majority (60%) of White Americans (Sharkey, 2013).

Fundamental cause theory posits this social structural position of Black Americans shapes access to resources and influences proximal health and mortality risk. However, a sole focus on comparing White and Black Americans neglects important questions as to whether and how within-race social structural factors influence mortality risk.

## Mortality Risk Across the Life Course

While medicine historically has focused on proximal factors that impact health (Vineis et al., 2016), the life course perspective emphasizes the importance of early factors and life course trajectories (Elder, 1985). Extensive evidence shows that early social structural position of low SES and belonging to a minoritized population have a dramatic, negative effect on health into midlife (Haas, 2008; Pavalko & Caputo, 2013). Yet the life course perspective does not consider childhood factors as solely deterministic of adult outcomes. Instead, childhood factors interact with later life experiences to promote continuity and change across the life course (Elder, 1985).

With respect to continuity, early life experiences set individuals on a path of accumulating advantages or disadvantages (Dannefer, 2003; DiPrete & Eirich, 2006; Merton, 1968; Pavalko & Caputo, 2013). The health disparities that begin in childhood among disadvantaged individuals are reproduced and compounded throughout the life course (Levine & Crimmins, 2014; Link et al., 2017; O'Rand & Hamil-Luker, 2005; Szanton et al., 2010), creating a life course trajectory of disadvantage that results in persistent deleterious risks to health and well-being (Hatch, 2005). Jackson et al. (2010) suggest that although Black Americans may be resilient throughout the early years, they begin to succumb to accumulated pressures and lifelong experiences (e.g., poverty, discrimination) in midlife. Geronimus et al. (2006) describe this among women as “weathering”—the impact of cumulative adverse social and economic experiences, such as repeated financial strain, excessive kin obligations, or discrimination. Taken together, the life course perspective puts forth the notion that persistent compared to intermittent disadvantage matters for mortality. While previous work focuses on the impact of proximal factors on mortality, the life course perspective requires consideration of various patterns of disadvantage including early-only, persistent, and late-only patterns.

With respect to change, the life course perspective highlights life course experiences, such as education (Hayward & Gorman, 2004; Link et al., 2017) and assumption of social roles (e.g., marriage, Umberson et al., 2010), that can disrupt trajectories of disadvantage and impact health and mortality. Educational attainment is a key coping resource that increases human capital and can reverse the

negative trajectory initiated with early adversity (Hatch, 2005; Mirowsky & Ross, 2003; Olshansky et al., 2012). Ross and Mirowsky (1999) have proposed that education gives people skills and knowledge that increase personal control, which in turn empowers them to engage in healthy behaviors (Seeman & Lewis, 1995). Social roles and community involvement can buffer the impact of early and cumulative adverse circumstances by encouraging compliance with social norms, regulating behavior, and building social capital (Umberson et al., 2010). For Black Americans, family and church are prominent, culturally unique sources of support (Chatters et al., 2009; Krause, 2006; Utsey et al., 2007) that can redirect the impact of adversities, such as discrimination and poverty (Brondolo et al., 2009; Paradies, 2006; Williams et al., 2003).

## Current Study

This study examines the effect of several social causes of premature mortality into middle and later adulthood (to the late 50 s) among the Woodlawn cohort, a community cohort of urban Black Americans followed since 1966. Drawing on the life course perspective and fundamental cause theory, we examine how poverty, neighborhood disadvantage, and racial discrimination across the life course impact mortality among a cohort who experienced similar childhood beginnings. We address the following research aims: (1) identify the age-related patterns of mortality by cause of death into the late 50 s, (2) estimate the impact of poverty at various points in the life course on mortality in the 40 s–50 s, (3) estimate the impact of neighborhood disadvantage during the life course on mortality in the 40 s–50 s, (4) estimate the impact of personal racial discrimination on mortality in the 40 s and 50 s, and (5) identify if education and social integration buffer the impact of early disadvantage on mortality in the 40 s–50 s. We expect life course poverty, neighborhood disadvantage, and discrimination to increase the risk of mortality, while education and social roles are expected to reduce the impact of early disadvantage. We expect persistent disadvantage to have a greater impact on mortality than intermittent (childhood only, adult only) or no disadvantage due to the accumulation of risk.

## Methods

The Woodlawn Study is a community cohort study that began in 1966 as a first-grade intervention program. All first graders attending the public and parochial schools in the Woodlawn neighborhood of Chicago were invited to participate. Only 13 families declined (approximately 1%), thus preserving the representativeness of the community

cohort ( $N = 1242$  children). At the time of the initial study, Woodlawn was one of the most socially disadvantaged communities in Chicago. According to the 1970 census, 96% of the Woodlawn neighborhood was Black, compared with 33% of the Chicago population overall. Households with children under 18 headed by women were also more common in Woodlawn (46%) than in Chicago overall (21%). However, due to residential segregation, some blocks in the Woodlawn neighborhood had high rates of home ownership and educational attainment.

First grade assessments in 1966 included interviews with each child's primary caregiver (primarily mothers) and teacher reports on classroom adaptation; follow-up assessments occurred in adolescence (age 16, 1976–1977), young adulthood (age 32, 1992–1993), and midlife (age 42, 2002–2003). The age 16 assessment included interviews with mothers ( $n = 937$ ) and a group-based survey with teenagers ( $n = 705$ ) in which questions were read aloud to the teenager.<sup>1</sup> In young adulthood, 952 cohort members completed a primarily in-person individual interview that asked about physical and mental health, substance use, crime, family life, social relations, and financial situation, among other topics. In midlife, 832 cohort members again completed a primarily in-person interview that asked about topics similar to those asked in young adulthood. In total, 1053 completed at least one of the two adult interviews, representing 85% of the original cohort.

Record retrieval, including accessing mortality records, has occurred over the course of the study. In 2020, we searched mortality records from the National Death Index. Names and other identifying information were submitted for all participants who were not previously confirmed to be deceased, including those who had been reported by family members to have died ( $n = 14$ ). At the time of the search, complete death data from the National Death Index were available for 1979 to 2018, which equates to approximately ages 19 to 58, as the vast majority of the cohort was born in 1960 (85%).<sup>2</sup> The National Death Index returned date of death and cause of death for potential matches. These results were reviewed by the research team to determine whether each potential match was a cohort member. If additional information was needed to make the determination, we conducted internet searches for obituaries and requested death certificates from the state where the death occurred to confirm matches.

<sup>1</sup> The smaller sample size in adolescence is related to funding limitations that did not allow participants who moved out of the Chicago area to be included in the in-person assessment.

<sup>2</sup> At that time, children needed to be 6 years of age by December 1, 1966, to begin first grade in public school.

## Measures

### Mortality

We created an indicator of whether or not the participant died by 2018 and age of death based primarily on official records from National Death Index. For those who did not die by the end of 2018, we censored the data at age 58. We ascertained cause of death from National Death Index records and categorized these causes into physical illness, HIV/AIDS-related, violent deaths (i.e., homicides, suicides, and trauma), and drug or alcohol-related deaths. Alcohol and drug-related deaths are primarily overdoses due to cocaine or heroin, but also include deaths classified as substance use being the primary cause (e.g., alcohol dependence syndrome). For the eight deaths before 1979, we based age and cause of death on parental reports since the National Death Index was not established until 1979 (see Juon et al., 2003, 2014 for more details on coding).

### Life Course Poverty

Respondents were categorized as poor or not poor for each of the four timepoints using federal poverty lines at the time of each assessment to determine the cutoff. To compute, we used household income from all sources for the previous year and household size as reported by the mothers in childhood and adolescence and by the participants in young adulthood and midlife and supplemented with welfare status for those missing income. We combined childhood and adolescence poverty to create a dichotomous variable of early poverty indicating if the respondent was below the federal poverty line at either timepoint. We also combined young adult and midlife poverty to create a dichotomous variable of late poverty indicating if the respondent was below the federal poverty line at either timepoint. To calculate life course poverty, we created four categories: never poor, early poverty (childhood and/or adolescence only), late poverty (adulthood only), and persistent poverty (both childhood/adolescence and adulthood).

### Neighborhood Disadvantage

Using the residential addresses provided by mothers in adolescence and self-reported in young adulthood and midlife, we used census data to compute the Neighborhood Disadvantage Index (NDI, Ross & Mirowsky, 2001). NDI is computed by adding the percentage of poverty and mother-only households and subtracting the percentage of home ownership and college educated residents for each respondent's census tract. Positive values indicate relative neighborhood disadvantage while negative values indicate relative advantage. The adolescent NDI was based on the 1980 census,

the young adult NDI on the 1990 census, and the midlife NDI on the 2000 census. For analyses, we dichotomized neighborhood disadvantage into high ( $\geq 0$ ) and low ( $< 0$ ) and created four categories: persistent disadvantage (all 3 timepoints  $\geq 0$ ), persistent advantage (all 3 timepoints  $< 0$ ), adolescent advantage only (only adolescence  $< 0$ ), and adult advantage only (either adult measure  $< 0$  but adolescence  $\geq 0$ ). We also calculated a mean of the NDI at the three timepoints, which resulted in a continuous indicator that we termed cumulative disadvantage.

### Life Course Discrimination

In young adulthood and midlife, participants reported on experiences of discrimination in various settings. The question stem was "Because of being black, have you ever...?" We created a count of the items endorsed at each timepoint. In young adulthood, we captured discrimination in employment, housing, school, entertainment venues, as well as police hassling and while walking around a neighborhood (range 0–6). In midlife, we captured discrimination related to these six items as well as being called insulting names, while shopping, while dining, racial violence, obtaining a loan, and related to medical care (range 0–12). For life course discrimination, we dichotomized discrimination as none ( $= 0$ ) or any ( $> 1$ ) due to low frequency of endorsement, and then created four categories: neither, young adulthood only, midlife only, and both young adulthood and midlife.

### Educational Attainment

Educational attainment was based on reports in adulthood about the highest level of schooling participants had completed. Categories included (1) no high school diploma, (2) GED or high school graduate, (3) some college or associate degree, and (4) college degree and beyond. For ease of interpretation of interaction effects in the moderation testing, we created a dichotomous measure of higher education in which 0 = no high school diploma, GED, or high school graduate and 1 = some college or associate degree and above.

### Social Integration

To capture overall social integration in adulthood, we created a summative measure of social roles for young adulthood and for midlife. This operationalization drew on the life course notion of cumulative advantage (Dannefer, 1987; Merton, 1968), which suggests that those with more social roles will be more socially integrated and thus have better outcomes in general than those with fewer social roles (Durkheim, 1951; House et al., 1988). We created a count of the number of social roles an individual occupied (currently married, currently employed full or part-time, a parent

currently living with his/her children, a member of a social organization, and a member of a church), which ranged from 0 to 5.

### Control Variables

Control variables included sex (0 = female, 1 = male), birth-weight (10 categories ranging from 0 = 8 pounds or more to 9 = less than 3.5 pounds), whether or not the child was ever hospitalized overnight (0 = no, 1 = yes), whether the child had a chronic health condition (0 = no, 1 = yes), and whether either parent had a chronic health condition (0 = no, 1 = yes). These health-related variables were reported by mothers in first grade. We also controlled for childhood aggressive behavior as rated by first grade teachers (0 = adapting to the classroom environment, 3 = severely maladapting). Finally, we controlled for parental mortality as reported in young adulthood (age 32) by participants (0 = both parents alive, 1 = one or both parents deceased).

### Analytic Plan

First, we describe the overall cohort by examining the timing and causes of death across the life course. Next, we limit our analytic sample to the  $n = 1159$  participants who survived past age 42 (i.e., the midlife assessment) to examine predictors of mortality in those who died between the ages of 43 and 58.<sup>3</sup> In Table 1, we compare those who died between ages 43 and 58 and those who remained alive on study variables calculating means and percentages with  $p$  values based on binary logistic regression due to the imputed data. While death records were considered complete, missingness on predictors due to wave nonresponse and a minimal amount of item nonresponse (< 5%) was accounted for by using multiple imputations; 40 datasets were imputed to maximize power and decrease bias (Graham et al., 2007). After examining descriptive characteristics of this analytic sample, we next ran bivariate Cox proportional hazard models and computed hazard ratios, 95% confidence intervals, and  $p$  values for each predictor and moderator. We tested for violations of the proportional hazard assumption using the Schoenfeld residual-based test. Next, we ran adjusted Cox proportional hazard models to provide greater insight on potential causal effects. Adjusted models included theoretically important control variables regardless of statistical significance and was run for any predictor value in which the statistical significance was less than  $p = 0.20$  in unadjusted models, as suggested by Hosmer and Lemeshow (1989). In all Cox

proportional hazard models, the persistently disadvantaged group served as the reference group, according to theory and the hypothesized direction of effect. Finally, we tested the ability of higher education and social roles to reduce the risk of early disadvantage by testing the significance of interaction terms within adjusted Cox proportional hazard models (e.g., early disadvantage  $\times$  higher education). Sensitivity analyses tested the mean of the three NDI scores in addition to the categorical specification. All analyses were conducted using the statistical package STATA (StataCorp, 2019).

### Results

By the end of 2018, 204 of the 1242 original Woodlawn children had died, representing 16.4% of the original cohort. Overall, 76 of the 636 females (11.9%) and 128 of the 606 males (21.1%) in the cohort had died by age 58; 62.7% of the 204 deaths were among males, showing a significant sex difference in mortality ( $p < 0.001$ ). Ages of death range from 9 to 58.98 (mean = 42.9, std dev = 12.0); 17.2% of deaths occurred before age 30, 18.1% between ages 30 and 39, 29.9% between ages 40 and 49, and 34.8% between ages 50 and 58. Figure 1, which addresses aim 1, shows that the proportion of deaths classified as violent (homicides, suicides, and trauma) steadily decreased as participants aged, with these deaths accounting for 51.4% of deaths before age 30, 16.2% of deaths between ages 30 and 39, 12.9% of deaths between ages 40 and 49, and 2.9% of deaths from ages 50 to 58. This contrasted with deaths related to physical illness, which steadily increased from 25.7% of deaths before age 30 to 85.7% of deaths between ages 50 and 58. Physical illnesses became the primary cause of death as the participants entered their 40 s. Deaths related to alcohol and drugs ranged from 5.7% of deaths among those younger than 30 to 16.2% of deaths among those who died between ages 30 and 39.

Among those who remained alive through age 42 ( $n = 1159$ ) but died between ages 43 and 58 ( $n = 121$ ), 57.0% were males and 43.0% were females (see Table 1). On average, a higher percentage of those who died between ages 43 and 58 had childhood health risk factors than those who remained alive, though these factors did not statistically significantly vary by mortality status ( $ps > 0.05$ ). Loss of a parent by young adulthood was more common among those who died versus those who remained alive at 58 ( $p = 0.06$ ). Those who died also experienced greater social structural disadvantage in childhood and across the life course compared to their alive counterparts, most notably persistent poverty ( $p < 0.001$ ) and adolescent neighborhood disadvantage ( $p = 0.03$ ). Educational attainment was lower for those who died; 27% had not completed high school, compared to 18% of those who remained alive. Over 43% of those who

<sup>3</sup> Previously published work on the Woodlawn study examined childhood factors that predict mortality through age 33 (Juon et al., 2003) and age 44 (Juon et al., 2014).

**Table 1** Characteristics of the Woodlawn cohort who remained alive by age 43 by mortality

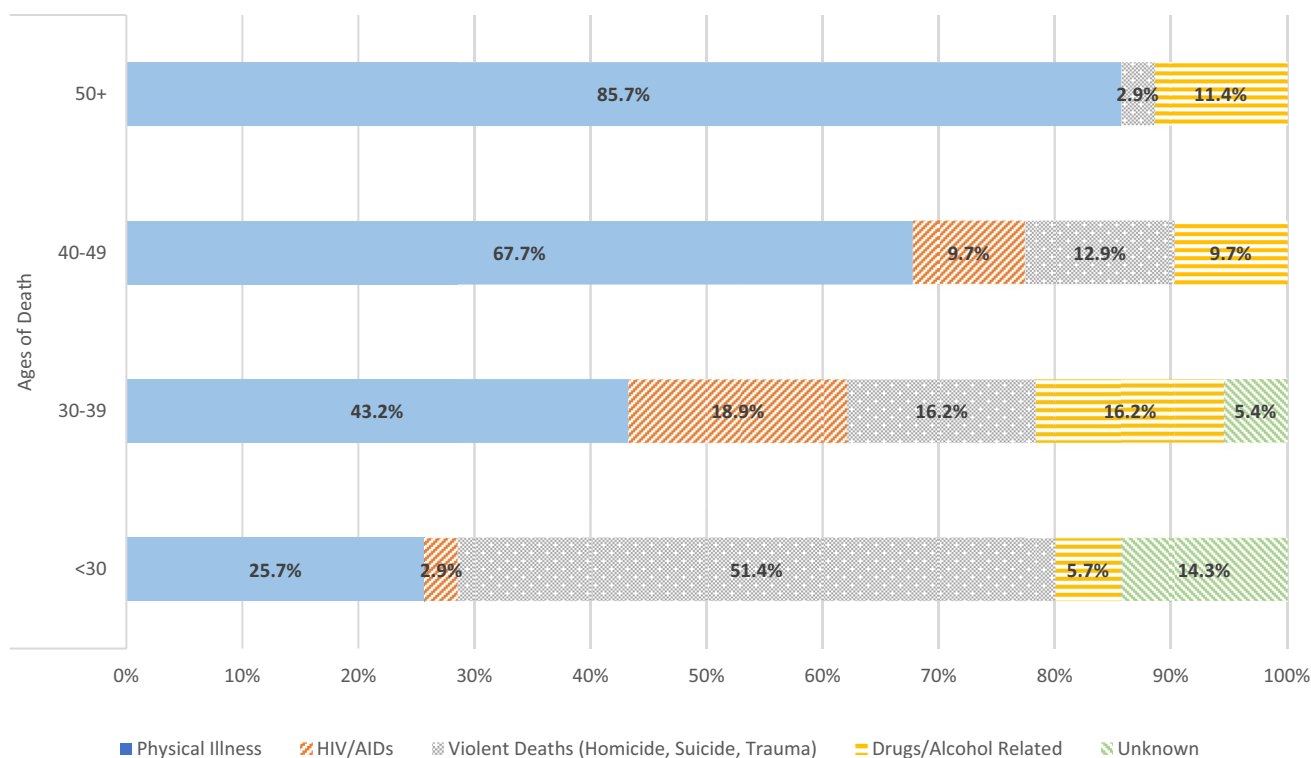
	Alive <i>n</i> = 1038	Died <i>n</i> = 121	Total <i>n</i> = 1159	<i>p</i> value
Sex (%)				
Male	46.1%	57.0%	47.2%	.023
Female	53.9%	43.0%	52.8%	
Lower birthweight, mean (se), range 1–10	2.34 (0.07)	2.60 (0.24)	2.37 (0.07)	.238
Childhood overnight hospitalization (%)	26.2%	29.8%	26.6%	.404
Childhood chronic condition (%)	3.7%	5.0%	3.8%	.481
Parental chronic condition (%)	12.4%	14.9%	12.7%	.444
Aggressive childhood behavior, mean (se), range 0–3	0.52 (0.03)	0.64 (0.09)	0.54 (0.03)	.180
Parental death by child age 32 (%)	51.6%	41.4%	50.5%	.060
Childhood or adolescent poverty (%)	62.0%	74.8%	63.3%	.007
Young adult or midlife poverty (%)	40.3%	58.2%	42.2%	.001
Life course poverty (%)				
Never poor	27.9%	13.2%	26.4%	(ref)
Early poverty only	37.9%	35.5%	37.6%	.024
Late poverty only	11.0%	12.4%	11.1%	.021
Persistent poverty	23.2%	38.8%	24.8%	< .001
Adolescent Neighborhood Disadvantage Index (NDI), mean (se), range – 27.9–39.0	1.66 (0.03)	5.03 (1.49)	2.02 (0.50)	.030
Young adulthood NDI, mean (se), range – 34.6–46.2	2.77 (0.56)	5.54 (1.67)	3.06 (0.54)	.098
Midlife NDI, mean (se), range – 37.9–44.7	– 2.60 (0.51)	0.05 (1.61)	– 2.32 (0.49)	.110
Life course neighborhood disadvantage (%)				
Adolescent and adult disadvantage	22.8%	26.6%	23.2%	(ref)
Adolescent and adult advantage	34.5%	28.8%	33.9%	.223
Adolescent advantage/adult disadvantage	8.1%	6.4%	7.9%	.426
Adolescence disadvantage/adult advantage	34.6%	37.8%	35.0%	.791
Young adult perceived discrimination, mean (se), range 0–6	1.66 (0.05)	1.89 (0.16)	1.68 (0.05)	.152
Midlife perceived discrimination, mean (se), range 0–12	3.03 (0.10)	3.08 (0.28)	3.03 (0.09)	.884
Life course perceived discrimination (%)				
Neither young adulthood nor midlife	12.9%	12.0%	12.8%	(ref)
Young adulthood only	8.9%	9.4%	8.9%	.799
Midlife only	17.1%	15.5%	16.9%	.948
Both young adulthood and midlife	61.2%	63.1%	61.4%	.748
Educational attainment (%)				
No high school diploma	17.6%	26.6%	18.6%	(ref)
GED/high school graduate	39.2%	42.5%	39.5%	.186
Some college/associates degree	30.0%	23.5%	29.4%	.022
College degree or more education	13.2%	7.3%	12.5%	.014
Young adulthood social roles, mean (se), range 0–5	2.50 (0.05)	2.10 (0.13)	2.46 (0.04)	.005
Midlife social roles, mean (se), range 0–5	2.71 (0.05)	2.07 (0.15)	2.64 (0.05)	< .001

Due to the use of multiply imputed data, *p* values are based on unadjusted logistic regression and show the statistical significance for the odds ratios predicting mortality

survived attended or graduated college compared to almost 31% of those who died. Finally, those who died had fewer social roles in both young adulthood and in midlife compared to those who remained alive (*ps* < 0.01).

To examine the impact of life course poverty (aim 2), neighborhood disadvantage (aim 3), and personal racial discrimination (aim 4) on mortality, we estimated unadjusted

and adjusted Cox regression analyses. As shown in Table 2, after adjusting for sex, childhood and parental health, and childhood behavior, those who were never poor were significantly less likely to die between ages 43 and 58 compared to those who were persistently poor; this translated to the never poor group being about 3 times more likely to be alive (*p* < 0.001). Those who experienced early poverty only also



**Fig. 1** Causes of death in the Woodlawn cohort over the life course ( $n=204$ ). Note: There were 35 deaths before age 30, 37 deaths between 30 and 39, 62 deaths between 40 and 49, and 70 deaths from ages 50 to

58. Cause of death was not systematically collected for early familial reports of death, hence the larger number of missing on cause of death for those who died before age 30

had a lower risk of mortality between ages 43 and 58 compared to those who were persistently poor ( $p=0.019$ ). In varying the reference group, we found that in adjusted analyses both those who were poor in adulthood only (aHR = 2.22, 95% CI = 1.09–4.53,  $p=0.028$ ) and those who were poor in early life only (aHR = 1.80, 95% CI = 1.01–3.22,  $p=0.046$ ) had about double the risk of mortality between ages 43 and 58 compared to those who were never poor. Early poverty only and late poverty only were not significantly different from one another in terms of mortality risk (aHR = 0.82, 95% CI = 0.45–1.48,  $p=0.509$ ).

In unadjusted models testing neighborhood disadvantage (aim 3) and discrimination (aim 4), we found no statistically significant associations with mortality (all  $ps > 0.20$ ); however, in sensitivity analyses, we found that cumulative neighborhood disadvantage (the mean of adolescent, young adult, and midlife NDI) increased mortality risk (aHR = 1.02, 95% CI = 1.00–1.04,  $p=0.039$ ), with greater mean disadvantage across time significantly predicting an increased risk of mortality. This adjusted model controlled for sex, birthweight, childhood hospitalization, childhood chronic health problems, parental chronic health problems, parental mortality, and childhood aggressive behavior.

When we analyzed adjusted models of higher education, young adult, and midlife social roles on mortality, we found

direct effects on mortality for all three variables. Those who had schooling past high school had 0.66 the risk of mortality between ages 43 and 58 compared to those who earned a high school diploma or had less education ( $p=0.049$ ). Those with more social roles in young adulthood ( $p=0.020$ ) and in midlife ( $p < 0.001$ ) also had a decreased risk of mortality.

To address our final aim (aim 5), we tested the potential for higher education and adult social roles to buffer the impact of early poverty on mortality between ages 43 and 58. In adjusted Cox regression analyses, we found a statistically significant interaction term for early poverty by higher education ( $p=0.040$ ). Specifically, those with higher education but grew up poor, those without higher education but did not grow up poor, and those without higher education and grew up poor all had over triple the risk of mortality compared to those who had higher education and did not grow up poor (HR = 3.31, 95% CI = 1.37–7.97,  $p=0.008$ ; HR = 3.43, 95% CI = 1.39–8.47,  $p=0.008$ ; HR = 3.54, 95% CI = 1.54–8.14,  $p=0.003$ , respectively). None of these three groups (i.e., those with higher education but grew up poor, those without higher education but did not grow up poor, and those without higher education and grew up poor) differed significantly on mortality risk from one another (HRs ranging from 0.94 to 1.03, all  $ps > 0.780$ ).

**Table 2** Unadjusted and adjusted regression results predicting mortality between ages 43 and 58 ( $n = 1159$ )

	Unadjusted models			Adjusted models		
	Unadjusted hazard ratio*	95% confidence interval	<i>p</i> value	Adjusted hazard ratio*	95% confidence interval	<i>p</i> value
Life course poverty (reference: persistent poverty)						
Never poor	<b>0.30</b>	<b>0.17–0.53</b>	<b>&lt;.001</b>	<b>0.36</b>	<b>0.19–0.60</b>	<b>&lt;.001</b>
Early poverty only	<b>0.59</b>	<b>0.39–0.88</b>	<b>.012</b>	<b>0.61</b>	<b>0.40–0.92</b>	<b>.019</b>
Late poverty only	0.70	0.39–1.25	.227	0.75	0.42–1.34	.325
Life course neighborhood disadvantage (reference: persistent disadvantage)						
Adolescent and adult advantage (persistent advantage)	0.73	0.43–1.23	.234	–	–	–
Adolescent advantage/adult disadvantage	0.68	0.26–1.78	.430			
Adolescence disadvantage/adult advantage	0.93	0.54–1.60	.795			
Life course perceived discrimination (reference: persistent discrimination—both adult timepoints)						
No discrimination in adulthood	0.90	0.49–1.68	.747	–	–	–
Young adulthood discrimination only	1.02	0.48–2.14	.966			
Midlife discrimination only	0.88	0.51–1.52	.658			
Buffers						
Higher education vs. high school diploma or less	<b>0.61</b>	<b>0.40–0.91</b>	<b>.015</b>	<b>0.66</b>	<b>0.43–1.00</b>	<b>.049</b>
Young adult social roles (range 0–5)	<b>0.82</b>	<b>0.71–0.94</b>	<b>.005</b>	<b>0.85</b>	<b>0.74–0.97</b>	<b>.020</b>
Midlife social roles (range 0–5)	<b>0.73</b>	<b>0.63–0.85</b>	<b>&lt;.001</b>	<b>0.76</b>	<b>0.65–0.88</b>	<b>&lt;.001</b>

Results are bolded when  $p < .05$ . Adjusted models were not tested for predictors where  $p > .20$

\*Four separate adjusted models were run (one for poverty and one for each buffer). Adjusted models controlled for sex, birthweight, childhood hospitalization, childhood chronic health problems, parental chronic health problems, parental mortality, and childhood aggressive behavior

We also observed a significant interaction between early poverty and midlife social roles, with midlife social roles reducing the risk of mortality more for those who were not poor than for those who were poor. The reduction in mortality was 0.54 for those who did not experience early poverty (95% CI=0.39–0.73  $p < 0.001$ ) and 0.84 for those who did experience early poverty (95% CI=0.72–0.99  $p = 0.038$ ), and these two hazard ratios were significantly different from one another ( $p = 0.024$ ). The interaction between young adult social roles and early poverty was not statistically significant ( $p = 0.183$ ).

## Discussion

This study provides a rare opportunity to characterize mortality among those who began their schooling in one crowded, segregated neighborhood of a large US city and identify prospectively gathered life course risk and protective factors related to mortality through midlife (ages 6–58). We found high rates of death in the cohort, with most deaths occurring in the 40 s and 50 s (29.9% and 34.8% of deaths, respectively), largely as a result of physical illnesses. Over 20% of the men and almost 12% of the women died by age 58, which

is significantly higher than the general US population at 13% of men and 7% of women (Bell & Miller, 2002).

Not surprisingly, lifelong poverty increased the risk of premature mortality, but more importantly, our findings show that timing matters most. We found that the effects of early poverty (without adult poverty) affected mortality decades later—those who were poor only as children had almost twice the risk of premature mortality than those who were never poor. This highlights the necessity of mitigating early poverty in order to reduce premature mortality, since the impact on mortality persists even when poverty does not. This finding extends those of Juon et al. (2003, 2014), who found evidence that early disadvantage (e.g., living in foster care, single-mother households) was related to mortality by age 32 and 42 for Woodlawn cohort members. However, those who were able to escape early poverty did live longer than those who remained poor throughout their lives. We further observed a proximal effect of adult poverty on death on its own and in concert with early poverty; those who experienced persistent poverty had the highest rate of mortality, highlighting the importance of the life course concept of cumulative disadvantage and its compounding impact on mortality risk.



Despite this increased risk of poverty across the life course, findings suggest some promise. Previous research among this cohort found that educational attainment and social integration are protective against substance use disorders (Crum et al., 1998, 2006; Fothergill et al., 2008; Green et al., 2010), and our findings show they also have a direct effect on mortality. Theoretically, education improves personal control and provides access to knowledge and resources (e.g., preventative health care), which are critical in longevity. Likewise, social integration builds social capital and offers social support, which can improve health outcomes, hence our expectation for a protective effect.

The significant interactions we found, though, align more with the power of continuity and cumulative advantage. Both education and social integration decreased the risk of mortality significantly more so among those who were not poor in childhood, supporting the fundamental cause theory notion that those who start life with relative advantage (i.e., higher SES) have access to more resources (e.g., knowledge, money, social connections), which in turn facilitate and amplify the health advantage over lower-SES individuals across the life course (Link & Phelan, 1995). While we expected education to buffer the impact of early poverty, we found no effect for education on mortality among those poor in childhood; this suggests that attainment of higher education should not be the primary target to reduce mortality disparities among those who grow up in poverty. In contrast, social roles in adulthood were able to reduce mortality for both those who experience poverty in childhood and among those who did not; however, the power of social roles was significantly greater for the non-poor, again suggesting limited ability in reducing disparities unless interventions are targeted only at disadvantaged groups. Future study is clearly necessary to identify factors that interrupt trajectories of poverty to inform intervention to eliminate health disparities.

Surprisingly, only cumulative neighborhood disadvantage (a form of structural racism), not personal racism (a form of perceived discrimination), was significantly related to mortality; however, its effect was small. It may be that racial segregation in the USA is so pervasive that even relative neighborhood advantage does not confer meaningful protection against premature mortality for Black Americans, though neighborhood improvements would surely benefit health. Research shows that even relatively advantaged Black Americans live in worse neighborhoods than their White counterparts (Perkins & Sampson, 2015). Personal racism, despite relatively low prevalence of self-reports, may be so ubiquitous among a disadvantaged Black cohort that its impact on mortality risk may only be detectable when compared to White Americans.

## Strengths and Limitations

There are significant strengths of this work. Prospective longitudinal data of this length (over 50 years) are exceedingly uncommon; research on the long-term outcomes of childhood SES often relies on adults' retrospective accounts, which can introduce bias. The Woodlawn Study data allow us to examine the timing and persistence of multiple types of disadvantage, both objective and subjective, to address the dynamic nature of social inequality and health over the life course. Moreover, this study focused on within-group differences, which is critical in uncovering heterogeneity that is masked when comparing between groups. We are able to separate out race from economic/social disadvantage, factors that are usually confounded in between-race studies, and provide insight on strength and resilience in Black communities.

As for limitations, this study examines mortality in a single community cohort. While there is significant value in describing and predicting mortality in a well-defined population, the generalizability of the findings is unclear. Theoretically, we expect the important factors identified in this study to generalize to other disadvantaged populations; however, this must be tested in other urban and non-urban cohorts, as well as cohorts of other races and ethnicities. Moreover, our measures of racial discrimination were somewhat limited and do not capture the intensity of the discrimination. Finally, while 16% of the cohort experienced death by age 58, we had insufficient power to stratify our cohort by sex or cause of death to better understand differential effects.

## Future Research

Findings point to multiple areas of inquiry for future work, in addition to cause-specific and sex-specific analyses to provide more insight into premature mortality for urban Black Americans. Risk and protective factors may vary depending on whether the death was related to, for example, a physical illness or an injury. As some sources of disadvantage in the Black community vary significantly in prevalence by sex (e.g., incarceration) and sex differences are evident in social positions and victimization, future studies should explore potential sex differences in risk and protection. As major racial disparities in mortality have been found during the COVID-19 pandemic, it is critical to determine if these are the result of socioeconomic position (e.g., disproportionate involvement as frontline workers, household overcrowding) or other structural factors rooted in the US's history of racism (e.g., vaccine distrust).

## Implications for Prevention

Health trajectories are not predetermined or static over time, showing the importance of interventions across the full life course, including the under-investigated period of midlife. While early childhood remains a critical time-point for intervention to disrupt early trajectories, one clear conclusion from our study is that it is beneficial to reduce poverty at multiple points in the life course, including midlife, in order to improve life expectancy among those from high-risk communities. As suggested by Jones et al. (2019), interventions need to be tailored to specific developmental stages and account for both positive and negative cumulative social and environmental exposures. In the absence of widespread social policy to improve socioeconomic inequality that contribute to Black/White disparities in mortality, which our results suggests as a critical target, it is necessary to intervene with protective factors that can alter early trajectories of disadvantage. Social integration in adulthood, which we defined as the accumulation of social roles, offers promise for reducing mortality among those who grow up poor (though interestingly not as much as it does for those who grow up not poor). Increasing social integration through broad efforts, such as prison reform and working with religious and social organizations to engage and support Black communities, could have tangible effects on decreasing premature mortality and decrease Black-White health disparities if targeted at high-risk groups, such as Black-Americans who experienced poverty early in the life course.

We also found that accessing higher education has a direct effect on reducing the risk of premature mortality among those from a disadvantaged neighborhood. However, this overall reduction was driven by the ability of accessing higher education to reduce the risk of mortality among cohort members who did not grow up in families experiencing poverty. Higher education did not alter the trajectory for those who grew up in families experiencing poverty despite many who grew up poor being able to access higher education. Clearly, equitable access to early and later intervention to mitigate the impact of early poverty is critical to reduce mortality disparities.

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## Declarations

**Ethics Approval** This study was approved by the University of Maryland Institutional Review Board and was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

**Consent to Participate** Freely given, informed consent to participate in the Woodlawn study was obtained from participants in adolescence, young adulthood, and midlife and their parent (or legal guardian) in childhood.

**Conflict of Interest** The authors declare no competing interests.

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