# The "Good Times" Cohort in Later-Life: Black–White Differences in Pathways to Functional Limitations

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## Abstract

Race differences in midlife circumstances explain much of the disability gap in older adulthood, but questions remain about whether early life selection processes are race invariant. To address this, we (1) isolate the 1930s cohort to explore potential race-specific life courses and (2) utilize a two-stage estimation procedure to examine the role of early-to-midlife selection in shaping later-life functional limitations. Using data on Black and White adults born 1931–1941 from the *Health and Retirement Study* (W2–W9), we estimate trajectories of later-life functional limitations after modeling midlife income and comorbidity as a function of early life factors. Fair/poor childhood health similarly impacts midlife morbidity for both races. Childhood disadvantage (poor family, father unemployed, and no father/deceased) had an adverse effect on midlife income for White but not for Black adults. An education gradient in functional limitations exists only for White adults. We interpret these findings in the sociohistorical context of this birth cohort.

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A significant advance in the study of racial health disparities in later-life has been the incorporation of the life-course perspective, particularly for outcomes such as functional limitations that are shaped by life-course selection and accumulation processes (Verbrugge & Jette, 1994). To that end, a number of studies have sought to identify whether race differences in early life conditions lead to disparities in later-life functioning. Observations of the higher proportion of Black Americans who have experienced disadvantaged childhood circumstances and subsequently greater functional limitations in older adulthood have led several to conclude that differential exposure to adverse conditions in early life is a fundamental cause of Black–White health disparities in later-life (Bowen, 2009; Haas & Rohlfsen, 2010; Johnson, Schoeni, & Rogowski, 2012). While important information, this line of inquiry is typically limited to identifying the degree and kind of early exposures that may have a lasting imprint on later-life health.

At the same time, epidemiologic work shows that the two predominant predictors of later-life functional limitations are midlife comorbidity (Ferrucci et al., 1996; Stenholm et al., 2015) and midlife socioeconomic status (Thorpe, Clay, Szanton, Allaire, & Whitfield, 2011; Wahrendorf, Reinhardt, & Siegrist, 2013). Given the accumulative character of both chronic morbidity and socioeconomic well-being, a stream of life-course studies examine early-to-midlife selection as a key pathway to later-life functional limitations (e.g., Freedman, Martin, Schoeni, & Cornman, 2008; Haas, 2008; Luo & Waite, 2005).

While consensus exists on the role of childhood socioeconomic status, family structure, and health in shaping later-life functional limitations, either directly or indirectly through midlife circumstances, evidence on whether these life-course selection processes differ for Black and White Americans is mixed. Some have found that pathways from early conditions to later-life functional limitations are invariant by race (Haas & Rohlfsen, 2010), while others find differences in the key factors that drive midlife and later-life health between Black and White adults (Hargrove & Brown, 2015; Shuey & Willson, 2008). For example, the health return on advantages such as educational attainment is substantially muted for Black adults relative to White adults (Shuey & Willson, 2008).

Successful interventions are dependent on understanding which risk factors account for the excess burden of functional limitations among Black

older adults relative to White older adults. One possible reason research on the drivers of racial health disparities has not reached consensus is because the extant literature is often treated as cohort invariant. In other words, there seems to be an implicit assumption that the mechanisms linking childhood and midlife conditions to later-life functional limitations are transferrable across birth cohorts and can therefore serve as one cohesive body of evidence rather than being sensitive to sociohistorical context of a particular cohort's collective life course. This has clinical relevance because neglecting the changes in opportunity structures, policy, and forms of institutional discrimination across cohorts may obscure important explanatory factors for understanding the patterns of health and survival among both minority and majority older adults today compared to future populations of these older adults (Colen, 2011; Rooks & Whitfield, 2004). This amounts to a missed opportunity to distinguish between risk factors that may be cohort-centric and those that may be more universal.

Calls for attention to cohort differences as a way of disentangling the mechanisms of health disparities have long been part of the social science canon (cf. Kuh & Wadsworth, 1993). Yet, some theoretical literature has emphasized that there are *multiple* life courses within a specific cohort (Dannefer, 2003; Moen, 2001). Based on the institutional arrangements of social life (e.g., military service, job-related opportunity structures, and health care) to which groups may have differential access, life paths for particular groups may be more "locked-in" relative to others born at the same time. To this end, the explanatory power for later-life health outcomes lies not in the initial conditions of one's early life as the differential exposure hypothesis articulates, but in navigation of social institutions across the life course, which is a dynamic and structural view of the problem. In this vein, Moen (2001) has suggested conceptual consideration of gendered life courses based on the structural and economic constraints placed on women relative to their male counterparts. And to our purpose here, Jackson and colleagues (Jackson, Govia, & Sellers, 2011; Jackson, Newton, Ostfield, Savage, & Schneider, 1988) similarly argue for consideration of racially distinct life courses within a given cohort, yet this line of inquiry remains underdeveloped.

Drawing from this framework of multiple life courses within a cohort, it stands to reason that the particular resources needed to "launch" one into an economically successful and healthy midlife—which are the key predictors of later-life functional health—may differ for Black and White Americans within the same birth cohort. To advance our understanding of potentially distinctive Black and White life courses and the impact on later-life health,

two innovations are necessary. First, we must isolate a single cohort to examine race differences in life-course influences on health. We focus on the cohort born 1931–1941, an era when de facto and de jure race segregation likely bifurcated the Black life course and White life course. Second, we explicitly model the selection process from childhood to midlife separately for Black and White adults. We first document how early life conditions select Black and White adults into midlife income and comorbidity and then we account for that selection in trajectory models of later-life functional limitations. This study is the first to utilize this type of modeling procedure to study health disparities and will help identify race differences in life-course selection processes that manifest in later-life functional limitations. First, however, we discuss the sociohistorical context of our selected cohort and the distinctive Black and White life courses within it.

## The "Good Times" Cohort and Their Life Course(s)

Demographic research has long focused on the distinguishing features of birth cohorts that serve to shape social, economic, and health profiles over the life course (cf. Pampel & Peters, 1995). For those born in the 1930s, Harter (1987) labeled them the Good Times cohort because of their unique opportunity structure. The birthrate of the 1930s cohort was about 25% smaller than the previous cohort from the 1920s, and this contraction in size disrupted a trend of ever-increasing cohort sizes. Their relatively small size led to lower competition for scarce resources in the population overall (Pampel & Peters, 1995). As a result, this cohort advanced more quickly into leadership positions in politics, education, and industry (relative to their larger adjacent cohorts). They were also first to be trained in the post–World War II era with heightened emphasis on science and technology, putting them at an even greater advantage in the labor market relative to previous cohorts (Easterlin, 1978; Harter, 1987).

Despite these common features for everyone born between 1931 and 1941, social institutions affecting health care, education, work, and residence were racially segregated. The call to consider racially distinct life courses (Jackson et al., 2011; Jackson et al., 1988) is to situate life-course selection processes within the structural and economic realities that make Black lives and White lives distinct. Do the early life factors that influence midlife morbidity and income differ by race group in this cohort? Below, we suggest possible hypotheses.

Childhood socioeconomic status may be a key differentiator for midlife well-being among White Americans in this cohort, while its effect on Black

Americans may be less salient. Elder's (1999) study of the children of the Depression, specifically White men who were born in the 1920s, found evidence that family economic deprivation dampened the likelihood of advanced educational attainment. Given the substantial compression of economic resources during the 1930s, being relatively *more poor* than other families may have been detrimental to potential achievement and mobility. Historical research notes that the unprecedented economic stratification led to the rise of a new class: poor Whites (Mell, 1938). Being part of this low resource, low-prestige group of Whites likely limited potential for advancement, manifesting in lower socioeconomic status, and poorer health by midlife.

Yet, the ubiquitous social disadvantage of Black Americans in this cohort led to very little differentiation in economic status for Black families. This challenges us to consider other factors that selected some into a healthy and financially secure midlife and not others. The social forces putting pressures on Black families exceeded the general conditions of the Great Depression: lack of citizenry, crippling unemployment among Black men, and mass migration. Some have argued that the unequal distribution of family structure by race during this era, due heavily to high mortality among Black fathers, served as a key driver of social and economic inequality (McLanahan & Percheski, 2008). Precursors to Social Security, such as widow's pensions, were channeled toward White families with few economic supports provided for Black widows (Poole, 2006). For these reasons, family structure may be a key indicator of additional social disadvantage in early life in this cohort, driving the economic and health profiles of the Black adults by midlife.

An interesting aspect of the American narrative about the 20th century has been the belief that policy interventions such as the G.I. Bill and Social Security acted as a "great equalizer" by providing race-blind opportunities to advance economically and socially. Yet, these policies discriminated against Black Americans through both explicit design (Poole, 2006) and application of the benefits (Katznelson & Mettler, 2008). Black men who qualified for the education benefit from the G.I. Bill, for instance, were disproportionately steered into technical training while their White counterparts were more likely to be steered into college. Thus, "post-high school" training looked markedly different for Black and White Americans at this time. Further, Social Security, passed in 1935, contributed to the increasing inequality in economic security between Black and White families, mostly by excluding the occupational domains of Black workers such as agriculture and domestic labor (Poole, 2006).

## Black Life Course, White Life Course, and Later-Life Functional Limitations

Exemplar work has laid the foundation for understanding the role of socio-historical context in health disparities. Krieger et al. (2014) integrated the racially segregated economic and social context of the 20th century to explain trends in the Black–White mortality gap. Other research has studied cohort shifts in predictors of later-life health and mortality, finding that some early life influences on later-life health, like educational attainment, vary in impact based on the particular cohort (Lynch, 2003). One noteworthy study (Herd, 2006) isolated the 1930s cohort to examine the question of whether educational disparities in functional health diminished at later ages. In so doing, Herd was able to conclude that the relatively modest education gradient in functional limitations—relative to other cohorts—could be attributed to its unique sociohistorical context. Although some of this work accounts for race differences, very little existing research has considered explicitly the hypothesis of a separate Black life course and a White life course within a cohort. In this study, we seek to integrate these elements of sociohistorical context and cohort-centric effects into the examination of life-course selection mechanisms that manifest in later-life functional limitations.

## Method

### Data

Data for this project are drawn from the *Health and Retirement Study* (HRS), which is a nationally representative panel study of adults aged 50 years and above interviewed every 2 years from 1992 (W1) to 2008 (W9; RAND Corporation HRS data file, Version j). The HRS utilizes a multistage area probability sampling design and has oversamples of Black and Hispanic adults. We isolate analyses to only the HRS cohort (born 1931–1941) so that all analyses of interindividual variability can be generalized to intracohort variability. The HRS cohort is ideal because the prospective panel design allows us to observe respondents in midlife (ages 50–59 years) who then transition into older adulthood (age 60+ years). Our key outcome, functional limitations, was measured differently in 1992 (W1) so we only use W1 to document nonrandom attrition. Measures of childhood conditions were first included for the total sample in 1998 (W4). We adjust all models to handle nonrandom panel selection. Proxy interviews were excluded since the outcome is self-reported functional limitations. After listwise exclusion of missing values and proxy interviews, our final analytic sample is composed of

5,706 White and 1,272 Black adults aged 53–63 years at baseline
(1994; W2).

## Measures

Our outcome is *functional limitations*, a wave-specific summated scale of
nine activity limitations. For each item, respondents were asked: "How
difficult is it for you to … " for a battery of activities. We use nine activity
limitations that capture the critical intermediate stage of functional loss
between impairment and disability (Nagi, 1976; Verbrugge & Jette, 1994),
consistent with other studies (e.g., Haas, 2008). These included ability to
walk several blocks, sit for 2 hr, reach arms above head, and lift/carry 10
pounds. We sum the difficulty performing each domain: *no difficulty* (0),
*some difficulty* (1), *cannot do* (2), with the final scale ranging from 0 to 18.
This approach is recommended because it captures variation in physical
function and is more sensitive for subgroup comparisons (Long & Pavalko,
2004). Average score on functional limitations at W2 is 2.49 ($SD = 3.66$) for
White adults and 3.75 ($SD = 4.78$) for Black adults, and this is significantly
different.

   To capture early life circumstances, we use respondents' retrospective
reports of childhood conditions. Respondents were asked: "Before age 16,
was there a time of several months or more when your father had no job?"
Father ever unemployed is measured with affirmative responses ($= 1$ and
*else* $= 0$). An alternative response category is "never lived with father/father
was not alive," which we use to identify having no father in the home/
deceased ($= 1$ and *else* $= 0$). It is particularly important for the present study
because of the high mortality rate of Black men (Rooks & Whitfield, 2004).
For childhood socioeconomic circumstances, we utilize the question: "Now
think about your family when you were growing up, from birth to age 16.
Would you say that your family during that time was pretty well-off finan-
cially, about average, or poor?" We create a binary indicator whether family
was poor relative to other families ($= 1$ and *else* $= 0$). In preliminary anal-
yses, we also included whether family was well-off financially, but it was
confounded with education attainment, particularly among White adults, so
dropped from further analysis.

   To capture childhood health status, respondents were asked to "consider
your health while you were growing up, from birth to age 16. Would you say
that your health during that time was excellent, very good, good, fair, or
poor?" Due to its bimodal distribution, we create a dichotomized variable
indicating whether the respondent's health during childhood was fair or poor

(= 1 and *else* = 0). Educational attainment is measured with three binary indicators (= 1 and *else* = 0): less than high school, high school graduate or equivalent, and more than high school. High school graduate is the reference category in the analyses.

We utilize two midlife indicators, income and comorbidity, due to their robust association with functional limitations and downstream impact from childhood conditions. Midlife income is measured as the sum of all wages and salaries at W2 (ln-transformed). Midlife morbidity is a self-report count of seven self-reported chronic conditions (e.g., cancer and diabetes) at baseline (age 53–63 years).

Later-life indicators include nonhousing wealth, which is the sum of all household assets excluding primary and secondary residence minus any debts. Since wealth is measured at the household level in the HRS, we created individual-level equivalencies by dividing each measure by the square root of household size, to be consistent with other empirical work (Willson, 2003). We logarithmically transform wealth measures to adjust for left skewness. We also identify whether the respondent currently smokes (time varying; *smokes* =1 and *else* = 0), *female* (= 1 and *men* = 0), and married (time varying; *married* = 1 and *else* = 0).

## Statistical Model

We employ a two-stage estimation procedure that allows us to document the impact of early life circumstances on midlife income and morbidity and then estimate trajectories of functional limitations accounting for the early life selection into midlife income and morbidity. To do so, we generate instrumented versions of the midlife indicators that are estimated as a function of the early life circumstances. The logic of the instrumented variable approach is to regress the midlife indicator on the early life circumstances and then extract the predicted scores of the midlife indicator to use in the substantive model predicting later-life health.

To integrate the elements of both latent growth curves and instrumented midlife indicators, we use the generalized multilevel latent variable model, described in Skrondal and Rabe-Hesketh (2004). The fully instrumented model is the following:

$$y_{ti} = \beta_0 + \beta_1 \text{Age}_{ti} + \beta_2 \lambda_i^{\text{mortality}} + \beta_3 \lambda_i^{\text{midlife morbidity}} + \beta_4 \lambda_i^{\text{midlife earnings}}$$
$$+ \zeta_{1i} + \zeta_{2i} \text{Age}_{ti} + \epsilon_{ti}.$$

Consistent with the traditional growth curve model, $y_{ti}$ is the number of functional limitations for individuals ($i$) at each wave ($t$). The initial level of functional limitations is represented by $\beta_0$, while the term $\beta_1 Age_{ti}$ accounts for the linear change with age. We capture measurement error across repeated measurement occasions within each individual (Level 1; intraindividual) with the term $[Errorstraightepsilon]_{ti}$. The other model components represent the incorporation of the instrumented variables represented with $\lambda$ to distinguish them from their directly measured counterparts. The base model already includes one instrumented variable: late life mortality risk ($\beta_2 \lambda_i^{mortality}$). This adjusts our growth curve model estimates for potential selection bias over the panel by incorporating the hazard of nonselection (inverse Mills ratio) due to mortality based from a probit selection equation (Heckman, 1979; Stolzenberg & Relles, 1997). The terms $\beta_3 \lambda_i^{midlife\ morbidity}$ and $\beta_4 \lambda_i^{midlife\ earnings}$ represent the slope coefficients for the instrumented versions of midlife morbidity and midlife earnings.

## Analysis Plan

We stratify all analyses between Black and White adults and test for significant differences where appropriate with an equality of coefficients test. The analysis proceeds in two steps. First, we instrument midlife morbidity (using a Tobit estimator) and midlife earnings (using an ordinary least squares [OLS] estimator). Specifically, we estimate regression equations predicting each with a series of early life circumstances: fair/poor childhood health and family socioeconomic status, no father in the home, father ever unemployed, and education level. We also include the unique indicators for each outcome to reduce the potential multicollinearity in the instrumented variables. For morbidity, we identify whether the respondent has been hospitalized in the past 2 years and indicate whether the respondent is currently obese (body mass index $\geq 30$). For income, we use the indicators of whether the respondent currently receives a pension and whether the respondent has private health insurance. To complete instrumentation, we extract the predicted values of each outcome to use as the new measures of midlife morbidity and midlife income.

Second, we estimate a series of latent growth curve models of late life functional limitations. In Model 1, we enter early life indicators and directly measured midlife morbidity and income, with controls for later-life functional status. Then in Model 2, we replace the direct measures of midlife morbidity and midlife income with their instrumented versions to account for

the influence of early life circumstances on midlife health and socioeconomic status.

## Results

Table 1 presents the means and standard deviations for baseline functional limitations and all independent variables separately for Black and White older adults. Significant differences between groups were tested with $t$-tests or $\chi^2$ as appropriate. In this cohort, Black older adults have significantly greater functional limitations than White older adults on average. Consistent with differential exposure hypotheses, we find a higher proportion of the Black adults with economically disadvantaged childhoods. Forty-two percent of Black adults recall that their family was poor relative to other families, as did 28% of White adults. While, 20% of Black older adults report that no father present in the home/deceased versus 7% for White adults, a higher proportion of White adults reported at least one period of time when his or her father was unemployed relative to Black adults (.19 vs. .14).

Slightly more Black older adults recall their health in childhood as being fair or poor (8%) than White older adults (6%). With regard to education, 43% of Black adults and 20% of White adults have less than a high school education. For both groups, a high school degree is most common: 60% of White adults and 42% of Black adults. A greater amount of White adults achieved training beyond high school than Black adults (20% and 15%, respectively). Midlife income and household wealth are significantly higher for White older adults than for the Black older adults. As for health, morbidity is significantly higher for Black older adults (1.45 [$SD = 1.22$]) compared to White older adults (1.03 [$SD = 1.02$]).

The first step of the analysis is to estimate the impact of early life circumstances on midlife morbidity and income and test for any potential race differences in those. Table 2 presents the tobit models for the morbidity count, separately for Black and White older adults. No race differences exist in the impact of early life social and health indicators on midlife morbidity, meaning that the pathway to greater morbidity in midlife is relatively similar for Black and White adults in this cohort. For both groups, fair/poor childhood health and recalling one's family as poorer relative to other families are associated with a higher number of chronic conditions for both groups. Having no father in the home/deceased during childhood had a small, positive impact on morbidity for White adults.

Education attainment, however, has substantial influence on midlife morbidity for both Black and White adults. Having less than a high school

**Table 1.** Means and Standard Deviations of Baseline (W2) Study Variables Separately for Black and White Adults.[a]

| Variables | White Adults (n = 5,706) | Black Adults (n = 1,272) | |
|---|---|---|---|
| Functional limitations | 2.49 (3.66) | 3.75 (4.78) | *** |
| Age | 57.98 (3.17) | 58.00 (3.16) | |
| Female | 0.52 | 0.60 | |
| Early life | | | |
|   Childhood health fair/poor | 0.06 | 0.08 | ** |
|   Family was poor | 0.28 | 0.42 | *** |
|   No father in home/deceased | 0.07 | 0.20 | *** |
|   Father ever unemployed | 0.19 | 0.14 | *** |
|   Less than high school | 0.20 | 0.43 | *** |
|   Greater than high school | 0.20 | 0.15 | *** |
| Midlife | | | |
|   Obesity | 0.23 | 0.37 | *** |
|   Hospitalized in past 2 years | 0.16 | 0.20 | ** |
|   Private health insurance | 0.76 | 0.60 | *** |
|   Receiving pension | 0.15 | 0.15 | |
|   Income (ln) | 10.15 (0.71) | 9.98 (0.59) | *** |
|   Morbidity count | 1.03 (1.02) | 1.45 (1.22) | *** |
| Later-life | | | |
|   Wealth (ln) | 11.13 (1.83) | 9.48 (2.01) | *** |
|   Currently smoking | 0.24 | 0.25 | |
|   Married | 0.80 | 0.55 | *** |

[a]Mean (standard deviation); boxes indicate significant differences by race. Differences in means tested with $\chi^2$ for binary variables and $t$-test for continuous variables.
*$p < .05$. **$p < .01$. ***$p < .001$.

**Table 2.** Tobit Models Predicting Midlife Morbidity for Black and White Adults Aged 53 to 63 Years: Health and Retirement Study Cohort 1994 (W2).[a]

| Variables | White Adults (n = 5,706) Coefficient[Sig.] (SE) | Black Adults (n = 1,272) Coefficient[Sig.] (SE) |
|---|---|---|
| Childhood health fair/poor | 0.322*** (.080) | 0.453** (.149) |
| Family was poor | 0.110* (.046) | 0.195* (.088) |
| No father in home/deceased | 0.169* (.077) | 0.074 (.106) |
| Father ever unemployed | 0.108* (.052) | 0.151 (.125) |
| Less than high school | 0.285*** (.054) | 0.263** (.097) |
| Greater than high school | −0.185*** (.044) | −0.314** (.111) |
| Obese | 0.629*** (.046) | 0.631*** (.085) |
| Hospitalized in past 2 years | 0.827*** (.052) | 0.603*** (.102) |
| Constant | 0.355*** (.038) | 0.703*** (.093) |
| Sigma | 1.388 | 1.423 |
| LR $\chi^2$ | 600.35*** | 154.43*** |
| Log likelihood | −8,116.294 | −2,012.838 |

*Note.* Sig. = significance; SE = standard error; LR = likelihood ratio.
[a]Unstandardized coefficient (SE); Black–White differences tested with the equality of coefficients test. No significant differences.
*p < .05. **p < .01. ***p < .001.

education is associated with higher average count of comorbid conditions compared with those who have a high school degree. Training past high school is associated with lower average comorbidities for both race groups, but the effect is slightly stronger for Black adults (−.314 [.111] vs. −.185 [.044]). The two instrumental predictors of midlife morbidity were obesity and whether one has been hospitalized in the past year. Both are significant and positively associated with morbidity count, but recent hospitalization has a stronger relationship with morbidity for White adults than Black adults.

Turning to midlife income, Table 3 presents the OLS estimates separately for Black and White adults. We observe some race differences in the early life influences on income. Fair/poor childhood health is associated with less income for White adults but not so for Black adults. Likewise, having a family more poor relative to others is also associated with diminished midlife earnings among White adults. For Black adults, however, not having a father in the home/deceased is significantly associated with lower income in midlife. We interpret these race differences with caution, however, because none achieved significance in the equality of coefficients tests.

**Table 3.** Ordinary Least Squares Regression Models Predicting Midlife Income for Black and White Adults Aged 53 to 63 Years: Health and Retirement Study Cohort 1994.[a]

| Variables | White Adults ($n = 5,706$) Coefficient[Sig.] (SE) | Black Adults ($n = 1,272$) Coefficient[Sig.] (SE) |
|---|---|---|
| Childhood health fair/poor | −0.117** (.035) | −0.049 (.050) |
| Family was poor | −0.074** (.020) | −0.041 (.029) |
| No father in home/deceased | −0.054 (.034) | −0.081* (.035) |
| Father ever unemployed | −0.002 (.023) | 0.028 (.041) |
| Less than high school | −0.097*** (.024) | −0.091** (.032) |
| Greater than high school | 0.288*** (.019) | 0.220*** (.036) |
| Receives pension | −0.324*** (.024) | −0.384*** (.039) |
| Private health insurance | 0.501*** (.020) | 0.569*** (.029) |
| Constant | 9.757*** (.022) | 9.715*** (.034) |
| $F_{(df1,df2)}$[Sig.] | 170.73$_{(8,5697)}$*** | 79.45$_{(8,1263)}$*** |
| Adjusted $R^2$ | .19 | .33 |
| Root MSE | .64 | .48 |

*Note.* Sig. = significance; SE = standard error; MSE = mean square error.
[a]Unstandardized coefficient (SE); Black–White differences tested with the equality of coefficients test. No significant differences.
*$p < .05$. **$p < .01$. ***$p < .001$.

The effect of education attainment on midlife income is robust and similar for Black and White adults: Relative to high school graduates, those with less than high school have significantly lower incomes and those with more than high school tend to have greater income. On average, those who receive pensions have lower income and those with private health insurance have higher income.

The second stage of the analysis is to estimate latent growth curve models of later-life functional limitations with and without adjusting for early-to-midlife selection. Model 1 includes early life circumstances, directly measured midlife morbidity and income, and later-life controls. Model 2 replaces the directly measured morbidity and income with their instrumented counterparts. Table 4 presents the findings from two models, stratified by race.

We first discuss Models 1 and 2 for White adults. On average, functional limitations increase, .077 ($SD = .003$), with each year of age. Women are significantly higher on average. Among the early life indicators, having fair/poor health in childhood is associated with greater functional limitations. Those whose family was poorer relative to others have greater functional

**Table 4.** Maximum Likelihood Estimates of Mixed-Effects Models Predicting Functional Limitation Trajectories With Direct and Indirect Effects of Early Life on Midlife Morbidity and Income for Black and White Adults Aged 60 Years and Above: Health and Retirement Study Cohort 1994–2010.[a]

| | White Adults | | | Black Adults | | |
| | Model 1 | Model 2 | | Model 1 | Model 2 | |
| Variables | Uninstrumented | Instrumented | | Uninstrumented | Instrumented | |
|---|---|---|---|---|---|---|
| Fixed effects | | | | | | |
| Age | 0.077*** (.003) | 0.077*** (.003) | | 0.067*** (0.008) | 0.066*** (0.008) | |
| Female | 0.893*** (.060) | 1.012*** (.064) | | 0.894*** (0.157) | 1.146*** (0.163) | |
| Childhood health fair/poor | 0.656*** (.125) | 0.237 (.136) | * | 0.422 (0.272) | 0.014 (0.293) | |
| Family was poor | 0.197** (.070) | −0.007 (.076) | * | −0.096 (0.159) | −0.262 (0.169) | |
| No father in home/ deceased | 0.198 (.118) | −0.037 (.127) | | 0.416* (0.193) | 0.043 (0.205) | |
| Father ever unemployed | 0.078 (.079) | −0.035 (.085) | *** | 0.377 (0.222) | 0.402 (0.233) | |
| Less than high school | 0.708*** (.085) | 0.278** (.095) | *** | 0.415* (0.178) | −0.102 (0.195) | * |
| Greater than high school | −0.100 (.076) | 0.233** (.083) | *** | 0.023 (0.215) | 0.366 (0.230) | |
| Midlife morbidity | 1.100*** (.030) | 1.750*** (.078) | *** | 1.045*** (0.064) | 1.906*** (0.170) | *** |
| Midlife income | −1.435*** (.124) | −1.610*** (.133) | | −2.254*** (0.293) | −2.747*** (0.306) | |
| Wealth | −0.855*** (.178) | −0.941*** (.179) | | −2.737* (1.321) | −2.708* (1.330) | |
| Currently smoking | −0.135** (.044) | −0.137** (.045) | | −0.351** (0.114) | −0.381** (0.115) | |

*(continued)*

Table 4. (continued)

| Variables | White Adults | | Black Adults | |
|---|---|---|---|---|
| | Model 1 | Model 2 | Model 1 | Model 2 |
| | Uninstrumented | Instrumented | Uninstrumented | Instrumented |
| Married | −0.021 (.042) | −0.027 (.042) | −0.291** (0.105) | −0.280** (0.106) |
| Selective mortality (λ) | −4.217*** (.472) | −2.782*** (.501) * | −3.669** (1.181) | −1.705 (1.236) |
| Intercept | 1.652*** (.190) | 0.948*** (.208) | 1.900*** (0.475) | 0.838 (0.518) |
| Random effects | | | | |
| Var(intercept) | 7.740*** (.205) | 8.612*** (.222) | 12.771*** (0.736) | 13.124*** (0.755) |
| Var(age) | 0.024*** (.001) | 0.024*** (.001) | 0.042*** (0.003) | 0.043*** (0.003) |
| Cov(intercept, age) | −0.282*** (.011) | −0.293*** (.012) | −0.544*** (0.044) | −0.532*** (0.044) |
| Level 1 residual | 2.542*** (.021) | 2.539*** (.021) | 4.184*** (0.075) | 4.183*** (0.075) |
| Log likelihood (df) | −89,410.6 (19) | −89,789.6 (19) | −20,860.9 (19) | −20,924.4 (19) |
| AIC/BIC | 178,859.2/179,023.5 | 179,617.2/179,781.6 | 41,759.77/41,894.37 | 41,886.8/42,021.4 |

Note. Var = variance; cov = covariance; AIC = Akaike information criterion; BIC = Bayesian information criterion.
[a]Unstandardized coefficient (standard error); differences between instrumented and noninstrumented models tested with equality of coefficients test. Boxed estimates are significantly different.
*p < .05. **p < .01. ***p < .001.

limitations. Higher education is associated with fewer functional limitations. Midlife morbidity ($b = 1.10$, $SE = .030$) is positively associated and midlife income ($b = -1.44$, $SE = .124$) is negatively associated with functional limitations. As for the later-life indicators, wealth and current smoker status are negatively associated with functional limitations. The Heckman adjustment for selective mortality indicates that those at highest risk of mortality tend to have lower levels of functional limitations. Post hoc analyses indicate this negative association is likely due to the high mortality rates among men and the high prevalence of smoking in this cohort.

In Model 2 for White adults, when we utilize the instrumented midlife morbidity and income, both fair/poor childhood health and having a poor family cease to significantly predict functional limitations. This means that accounting for the early-to-midlife selection eliminates any residual direct effect of childhood circumstances on later-life functional limitations. Likewise, the education gradient is significantly attenuated, although still significant, indicating some additional impact education has on later-life not yet captured. All other covariates significant in Model 1 remained so in Model 2.

Turning to Models 1 and 2 for Black adults, functional limitations increase, on average, .067 ($SE = .008$), for each year of age. Women are significantly more likely to have functional limitations. Having no father in the home/deceased during childhood is associated with greater functional limitations but no other childhood conditions are significant. Relative to high school graduates, those who have less than high school education have significantly higher scores on functional limitations. However, there is no significant difference between high school graduates and those who achieve higher degrees. Greater midlife morbidity and lower midlife income are both associated with greater functional limitations. Wealth and being a current smoker are associated with fewer functional limitations. The Heckman adjustment for selective mortality indicates that those at high risk of mortality have fewer functional limitations.

Turning to Model 2, with the instrumented midlife morbidity and income, two covariates became nonsignificant. Having less than a high school education becomes nonsignificant in Model 2. Functional limitations among those with education beyond high school were already not significantly different from high school graduates. Thus, when we account for the early-to-midlife selection process, we observe no education differences in functional limitations for Black adults. Further, having no father in the home/deceased becomes nonsignificant. No other covariates change from Model 1 to Model 2, except that the adjustment for selective mortality becomes nonsignificant.

## Discussion

It has been suggested that race differences in midlife circumstances explain much of the disability gap in older adulthood (Bowen, 2009; Haas & Rohlfsen, 2010; Johnson et al., 2012; Thorpe et al., 2011). Thus, a number of studies examine how early and midlife circumstances may lead to later-life functioning among Black and White adults (e.g., Haas & Rohlfsen, 2010; Hargrove & Brown, 2015; Shuey & Willson, 2008), but questions remain about the degree to which these mechanisms are race invariant. One possible reason for this lack of consensus is the tendency to treat the extant evidence as cohort invariant, leaving no way to adjudicate mixed findings. To advance our understanding of later-life racial health disparities, we present two innovations in this study relative to the previous literature. First, we isolate the 1930s cohort to contextualize potential race-specific life courses for Black and White Americans born during this time. Second, we utilize a two-stage estimation procedure allowing us to examine whether the selection mechanisms from early life circumstances to midlife health and socioeconomic status differ by race. Our findings indicate points of difference and similarity for Black and White adults in early-to-midlife selection effects on later-life functional limitations, providing support for both cohort-specific factors and racially distinct life courses within the cohort.

The first finding of interest is the robust association of fair/poor childhood health with midlife morbidity for both race groups in this cohort. Fair/poor childhood health, reported by only 6% of White adults and 8% of Black adults, likely represented very serious illnesses relative to later cohorts (Kelley-Moore, 2010). Penicillin was not widely available until the mid-1940s and immunizations for measles, polio, and other infections that could affect long-term functional capacity only became available much later. This represents a cohort-centric effect that would have affected all children regardless of race, thus the robust association—and its race invariance—between fair/poor childhood health and midlife morbidity is not surprising. It is possible, however, that we would observe a growing race gap in the impact of childhood health on midlife morbidity in subsequent cohorts, perhaps reflecting unequal access to the resources that became available to protect childhood health.

The second major finding of this study is the mixed impact of education on midlife circumstances versus later-life functional limitations. On the one hand, the effect of education attainment on midlife morbidity and income is relatively invariant by race, exhibiting a clear gradient across the categories of less than high school, high school graduate, and greater than high school.

The fact that education has an equivalently robust gradient for both race groups in its effect on midlife morbidity and income underscores previous literature showing that education attainment has a stronger return in cohorts who come of age in advantageous economic periods like the Good Times cohort (Cutler, Huang, & Lleras-Muney, 2015; Harter, 1987).

On the other hand, we find that education has a stronger effect on later-life functional limitations for White adults than Black adults. This finding is consistent with other research (Luo & Waite, 2005; Shuey & Willson, 2008), yet our work goes a step further using the two-stage estimation procedure to examine whether education attainment continues to have an independent effect on later-life functional limitations once we account for its selection effects on midlife morbidity and income. Among Black adults, we find no residual direct effect of education on later-life functional limitations beyond its impact on midlife circumstances. For White adults, however, after accounting for its selection effects on midlife morbidity and income, education continues to have an *additional* independent impact on later-life functional limitations. The lack of an education gradient in late life functional limitations among Black Americans likely underscores the institutionalized racism operating for this cohort, such as lower quality of education in segregated schools or systematic exclusion from occupational domains, which would have allowed Black Americans to capitalize on their advanced education. It is evidence such as this that supports Williams and Collins's (2001) assertion that the segregation is a fundamental cause of racial health disparities.

Our third major finding is Black–White differences in the specific childhood circumstances that shape midlife morbidity and income. Among White adults, all of the indicators of childhood economic and social disadvantage (family was poor, father ever unemployed, and father not in home/deceased) were associated with higher morbidity in midlife and, additionally, having a poor family predicted lower midlife income. Black adults did not have nearly the degree of differentiation in early-to-midlife selection; having a poor family was the only indicator of elevated midlife morbidity and no such associations among childhood indicators were found for midlife income.

These findings may be the strongest evidence of a separate Black life course and White life course. In a cohort imprinted by its unprecedented opportunities, we observe increasing socioeconomic differentiation among Whites via midlife health and income and then later-life functional limitations. At the same time, greater social disadvantage and fewer opportunities available to Black adults in this cohort suppressed the prospect of such differentiation. However, the civil rights movement and desegregation of

key social institutions in the middle of the century could mean that commensurate economic gradients in later-life health would appear for Black Americans in later cohorts.

Finally, based on previous literature (e.g., Haas, 2008; McLanahan & Percheski, 2008), we had hypothesized that family structure would be a key indicator of midlife health and socioeconomic status for Black adults. However, we found only one modest association between no father in the home/ deceased and lower midlife income. Our focus on the Good Times cohort may have been temporally premature to see the full impact of economic disadvantage and family structure on midlife and later-life well-being among Black adults. It is possible that the family structure hypothesis would be more valid in later cohorts, as the intersecting forces of urbanization and racist social welfare policies drove many predominantly female-headed households into crippling poverty (Moynihan, 1965; Poole, 2006).

This study has several limitations. First, the indicators of early life resources are measured through retrospective self-report and the measures themselves are not particularly specific. Future work should focus on reconstructing both the family and childhood economic histories to identify more precisely the pathways to midlife inequality. Second, the health measures (morbidity; functional limitations) are based on self-report. Black Americans, particularly men, are likely to underreport actual limitations, which could introduce a downward bias to the estimates (Thorpe et al., 2011). Third, the childhood measures in the HRS were not introduced until W4, introducing selection bias into the panel study. While we adjust for nonrandom attrition in the statistical models, selective mortality more generally is a major driver of racial composition for this cohort and may lead to an underestimation of the impact of childhood disadvantage.

Taken together, these findings advance the inquiry on minority aging by emphasizing the importance of cohort context and race-specific life courses within a particular cohort. It challenges the notion that certain well-known markers of childhood disadvantage, such as education attainment, not having a father in the home/deceased, or the family being poor, have a relatively universal impact on later-life health, independent of cohort context. The 1930s, or Good Times, cohort had unprecedented economic and social mobility opportunities. It is possible that subsequent cohorts who came of age in different sociohistorical contexts, particularly with regard to the degree of race segregation and the health of the economy, may exhibit unique risk factors for later-life functional limitations that were previously obscured by treating the extant evidence as cohort invariant. Systematic consideration of cohort-specific context and the multiple institutionalized life courses

within it will help contextualize the variability in health and functioning that we observe among older adults and provide a more nuanced understanding of the mechanisms of social inequality for all race/ethnic groups.

## Declaration of Conflicting Interests

## Funding

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