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Publication Date

2022-09-01

DOI

10.1016/j.healthplace.2022.102904

Peer reviewed

HHS Public Access

Author manuscript

Health Place. Author manuscript; available in PMC 2023 September 02.

Published in final edited form as:

Health Place. 2022 September ; 77: 102904. doi:10.1016/j.healthplace.2022.102904.

The long shadow of residential racial segregation: Associations between childhood residential segregation trajectories and young adult health among Black US Americans

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Abstract

Residential racial segregation is a key manifestation of anti-Black structural racism, thought to be a fundamental cause of poor health; evidence has shown that it yields neighborhood disinvestment, institutional discrimination, and targeting of unhealthy products like tobacco and alcohol. Yet research on the long-term impacts of childhood exposure to residential racial segregation is limited. Here, we analyzed data on 1,823 Black participants in the Panel Study of Income Dynamics, estimating associations between childhood segregation trajectories and young adult health. Black young adults who consistently lived in high-segregation neighborhoods throughout childhood experienced unhealthier smoking and drinking behaviors and higher odds of obesity compared to other trajectory groups, including children who moved into or out of high-segregation neighborhoods. Results were robust to controls for neighborhood and family poverty. Findings underscore that for Black children who grow up in segregated neighborhoods, the roots of structurally-determined health inequities are established early in life.

Keywords

segregation; neighborhoods; racism; life course; health inequities

INTRODUCTION

Across an array of chronic diseases, Black people in the US suffer a disproportionate burden of morbidity and mortality relative to non-Hispanic White people. These health disparities arise as early as birth[1,2] and are apparent for many of the US' leading causes of morbidity and premature mortality, including cardiovascular disease,[3,4] many types of

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cancer,[5] dementia,[6] kidney disease,[7] asthma,[8] and diabetes,[9,10] among others. The ubiquity of these disparities across so many physiologic systems throughout life, along with the fundamentally social basis of race,[11] indicates that these disparities are in fact inequities with structural, social origins, not a collection of genetic coincidences that happen to map onto gradients of racialized socioeconomic power. That is, these inequities are driven by manifestations of structural and interpersonal racism, affecting many pathogenic exposures and access to diverse health resources.[12]

One such manifestation of structural racism may be residential racial segregation.[2,10,13,14] US neighborhoods remain highly racially segregated: despite weak trends towards integration, the average neighborhood of a White American in 2018 was 71% White and 8% Black.[15] The average neighborhood of a Black American, in contrast, was 45% Black and only 31% White. This segregation is the result of historical, explicitly racist government action; current policies and financial practices that disproportionately impoverish and exclude people of color; and group preferences arising from structural racism ("social-structural sorting").[14,16–19] Because residential racial segregation can lead to discrimination and stigma (including over-policing[20–22] and housing financing discrimination[23–25]), a more pathogenic[26–32] and resource-scarce[33–37] built environment, fewer economic opportunities,[13,38] and concentrated disadvantage within racially isolated social networks,[39] it has been called a "fundamental determinant" of health—a structural factor shaping more proximate social and environmental determinants of health and of racial health inequities.[14,20,21,38] Previous research has linked residential racial segregation to many worsened health outcomes, including cardiovascular disease, birth outcomes, cognitive function, and metabolic health.[40–45]

Epidemiologists' understanding of when in the life course exposure to residential segregation matters, however, is still developing. Few studies have linked exposure to segregation at critical developmental periods (e.g., early childhood, adolescence) to later health.[40,46–48] Fewer have examined lifecourse *trajectories* of exposure to residential racial segregation, incorporating changes in segregation exposure throughout life as well as the duration and developmental timing of that exposure. These factors may be critical. For example, very early exposure to neighborhoods whose conditions induce high stress and offer few healthy food options may cause lasting epigenetic changes that alter stress responses and metabolic function, regardless of later neighborhood exposures.[49,50] Poorer educational opportunities at a young age may set children on a lower educational trajectory even if they move into a better-funded school district in adolescence.[51–53] On the other hand, poor or disrupted access to neighborhoods and schools with sufficient resources during adolescence (a decisive time for degree completion) may serve as a powerful educational stumbling block, with long-term health implications.[54,55] Studies that examine segregation exposure histories, then—not just point-in-time exposures—are needed.

In this paper, we provide some of the first evidence linking trajectories of residential segregation during childhood with young adult health. We use data from a nationally representative longitudinal study of American households, examining a set of health outcomes and chronic disease risk factors likely to be influenced by residential segregation.

METHODS

Data & Sample

We analyzed data from the Panel Study of Income Dynamics (PSID), the longest-running study of individual and family circumstances in the United States. A nationally representative sample of largely Black and non-Hispanic White families were recruited in 1968, drawn from two nationally representative sampling frames: 1,872 low-income households from the sampling frame of the Survey of Economic Opportunity (1966–1967), with an additional 2,930 more economically diverse households from the sampling frame of the University of Michigan's Survey Research Center, yielding 4,802 households living in 40 states. Members of these original households, as well as their descendants, were then surveyed annually through 1997 and biennially thereafter. While the core PSID only collects health information on household heads and their spouses, PSID began a biennial Transition to Adulthood Study (TAS) in 2005 to collect rich data on young adults aged 18–28 years who grew up in PSID households. We linked all waves of the TAS, available through 2017, with core PSID data collected during TAS participants' childhoods, including their residential neighborhoods at the time of each household interview. We then linked TAS participants' childhood residential neighborhoods (Census tracts) to race-specific population counts from the US Census to measure residential racial segregation exposures.

For our primary analysis, our sample consisted of Black TAS participants (starting n=1,830). All of these reported at least one health outcome of interest during their young adulthood, and 97% reported every health outcome at least once, with only 7 individuals missing any exposure or covariate information. Of young adult health records, across all participants, 93% included complete data for all outcomes. Given such low missing-ness rates, selection bias from missing data is unlikely, so we opted for a complete case analyses (final n=1,823). All participants in our analytic sample were observed at least twice during childhood, allowing the calculation of at least some kind of segregation trajectory; more than 90% of participants were observed 10 or more times, providing rich data for describing childhood segregation exposures. Outcome models were run using all TAS waves (when health outcomes were reported), so individuals could have multiple records during young adulthood (n=4,925 total young adult records; mean 2.7 records, median 3, range 1–6).

Comparing associations among Black and White participants could have been instructive, allowing us to examine whether living in racially segregated neighborhoods affects residents differentially depending on their race (and thus their access to social and economic power regardless of where they live). However, the proportion of White participants who lived in predominantly Black neighborhoods was too small to make meaningful comparisons. For example, only about two dozen White participants—of thousands—lived their entire childhoods in neighborhoods with high concentrations of Black residents. White TAS participants were thus excluded.

Segregation measures were calculated (see below) using data from the closest (in time) race-specific population counts collected by the US Census for a given wave of measurement in childhood; in particular, either the decennial Census (1990, 2000, 2010) or American Community Survey's 5-year estimates (2010–2014, 2014–2018).

Residential racial segregation

We measured residential racial segregation in each wave that a member of our sample was observed during childhood. We did so by calculating Local Getis-Ord G* statistics at the Census tract level (normalized to 2010 boundaries), a widely used measure in social epidemiology.[40,41,44,45,47,56,57] These G* statistics are Z-scores. They compare the proportion of the population in a focal tract and its neighboring tracts that is Black to the average proportion of a larger surrounding geographic area that is Black.[58] These "larger areas" were Core-Based Statistical Areas (CBSA) for the vast majority of tracts, or counties for tracts that were not in CBSAs. Spatial connectivity matrices assumed "queen" spatial contiguity (all tracts whose borders touched the focal tract were considered neighbors). We considered a neighborhood to be "highly segregated" if its G* statistic was above 1.96, indicating that a tract and its neighboring tracts had a high proportion of residents who were Black compared to the average within their CBSA or county (i.e., two standard deviations above the mean).

Note that it is not only neighborhoods with high concentrations of Black residents that are "highly segregated" – neighborhoods with extremely few Black residents, for example, are as well (i.e., $G^* < -1.96$). Of these two highly segregated categories, however, only isolation of Black residents engenders underinvestment and resource deprivation; these are accordingly our focus.

PSID participants rarely shared neighborhoods. Focusing on the first observed tract for each PSID participant, 67% of tracts had only 1 PSID participant who lived in them, 90% had 2 or fewer, and 95% had 3 or fewer.

Exposure: segregation trajectories

For our primary analysis, we transformed these repeated segregation measures into segregation trajectories, allowing us to assess (A) whether the *duration* of exposure to high vs. low segregation neighborhoods was associated with young adult health/chronic disease risk and (B) whether *trajectories* of segregation exposure (i.e., increasing or decreasing exposure to highly segregated neighborhoods) were associated with those outcomes. To do so, we categorized childhood segregation exposures into five trajectories: (1) always lived in highly segregated neighborhoods, (2) moved from a lower- to a high-segregation neighborhood, (3) moved from a high- to a lower-segregation neighborhood, (4) always lived in lower-segregation neighborhoods, or (5) moved back and forth between high- and lower-segregation neighborhoods. (Because this fifth category represents a diverse array of segregation trajectories, we present model estimates for this group only in the appendix.)

Here, "moved" could refer to a literal move between neighborhoods or a situation where neighborhood characteristics changed over time despite a stable address. The former was much more common: 92% of tract segregation changes were due to a literal move in the high-to-low group, as well as 77% of tract segregation changes among the low-to-high group. For simplicity, we refer to these changes as "moves" throughout.

Outcomes

We included two health outcomes and three risk factors that indicate increased risk for future chronic disease. Health outcomes included self-rated health (a 5-point Likert scale ranging from poor to excellent, which we dichotomized into poor/fair vs. good/very good/excellent) and the Kessler 6 psychological distress inventory (or K-6, a 6-item scale that measures depression and anxiety symptomatology; range: 0–24).[59] Self-rated health is a robust predictor of mortality, physical function, outcomes of healthcare treatment, and an array of biomarkers, providing a global assessment of physical health status.[60–62] The K-6, for its part, provides a reliable screening tool for mental distress of varying severities and has been validated across many social and demographic groups.[63]

Chronic disease risk factors included alcohol consumption, cigarette smoking, and obesity. Alcohol consumption was measured with three variables: whether participants had ever drank alcohol (binary) or had drunk more than once per week in the past year (binary), as well as the average number of days they binged on alcohol in the past year (with "binging" defined as per the National Institute on Alcohol Abuse and Alcoholism as ≥5 drinks in one sitting for male people or ≥4 for female people[64]). Smoking was similarly measured with three variables: whether participants had ever smoked, currently smoked, or currently smoked more than 10 cigarettes per day. Obesity was measured as a body mass index (BMI) of 30 kg/m^2 or more. Though obesity represents a range of body types and adiposity levels, and individuals with the same BMI may have dramatically different mortality risks and fitness levels, it is a useful population-level predictor of chronic disease and mortality risk.[65,66]

Covariates

Model covariates included household socioeconomic status indicators, including average inflation-adjusted family income across childhood, parental education (<12 grades completed, 12 grades, 13–15 grades, or 16+ grades), and whether participants' parents were ever married, as household resources powerfully determine which neighborhoods families are sorted into as well as their health outcomes.[67] We also included average Census tract population and poverty level across childhood, since both could determine both neighborhood access to health-promoting services and degree of residential segregation. Finally, we included fixed effects for the state in which each participant lived for the longest during childhood, accounting for time-invariant state-level confounders (e.g., different regions of the US experience different levels of segregation[68] and different investments in population health[69,70]).

Analysis

We estimated associations between residential racial segregation trajectories in childhood and each outcome of interest using multivariable regression. We used linear regression for continuous variables and logistic regression for binary variables. We adjusted for the covariates above and clustered standard errors at the individual level to account for auto-correlation between participants' (repeatedly measured) outcomes. In sensitivity analyses, despite few PSID participants sharing Census tracts, we also ran models accounting for tract-level clustering.

In sensitivity analyses, we excluded Census tract poverty and population density as covariates, because population and poverty distributions across neighborhoods are inextricably linked to racial segregation.[71] On the one hand, neighborhood poverty likely serves as a mediator between residential segregation and health because residential racial segregation can lower economic opportunities for area residents (with subsequent health consequences even independent of household poverty[72,73]). On the other hand, neighborhood poverty likely also serves as a confounder, since housing costs are lower in poorer neighborhoods and thus Black families who experience employment and education discrimination may be sorted into disinvested, segregated neighborhoods because those are disproportionately the neighborhoods they can afford to live in.[67] In other words, neighborhood poverty may shape the racial make-up of in-movers as well as cause poor health. This makes neighborhood poverty and residential racial segregation co-constitutive, forming a constantly mutually reinforcing, dynamic relationship.[13,38,71] By presenting results including and excluding neighborhood poverty and population density from our models, we provide multiple reasonable analytic alternatives that are both scientifically justifiable, and which help offer insight into whether neighborhood poverty is the only path through which residential segregation impacts health. In another sensitivity analysis, we changed the segregation trajectory group serving as the reference in regression models, allowing us to compare all trajectories groups either to those who consistently lived in high segregation neighborhoods (our main results) or to those who consistently lived in low segregation neighborhoods (sensitivity).

Secondary analysis: developmental timing of segregation changes

Our secondary analysis assessed whether the *developmental timing* of children's moves into or out of highly segregated neighborhoods mattered. For example, those who moved into highly segregated neighborhoods in early childhood (and thus spent the majority of their childhood there) may have experienced few health benefits relative to the always-highlysegregated, while those who moved into highly segregated neighborhoods in adolescence (after living their whole early childhoods in more integrated neighborhoods) may have experienced relative health advantages. To test this, we restricted the sample either to those whose segregation levels were stable in adolescence (Sample A) or those whose segregation levels were stable in early childhood (Sample B). We then redefined our five-category exposure using only observations in early childhood (for Sample A) or only observations in adolescence (for Sample B). For example, for Sample A, the second exposure category was defined as "moved from a low segregation to a high segregation neighborhood in early childhood, then levels were stable during adolescence," the third as "moved from a high segregation to a low segregation neighborhood in early childhood, then levels were stable during adolescence," etc.

As we restricted to those whose segregation levels were stable during early childhood or adolescence, our secondary analysis samples were smaller, containing 1,767 Black TAS participants who had at least two observations for each period (ages 0 and 10 and ages 11 and 17). For examining early childhood moves between neighborhoods with different segregation levels, we retained 1,377 Black TAS participants contributing 3,769 observations during young adulthood. For examining adolescent moves between neighborhoods with

different segregation levels, we retained 1,157 Black TAS participants contributing 3,064 observations.

All analyses were run in StataMP 16.[74]

RESULTS

Sample characteristics

Demographics and average health varied across segregation trajectory groups (Table 1). Mean average family incomes across childhood ranged from about $33,000 to $43,000, between 8 and 17% of each group had a parent without a high school education, and 28–41% of each groups' parents had never married. Mean birth years ranged between 1991 and 1992. The proportion of childhood spent in a high-segregation neighborhood followed a rough gradient of social marginalization, with those living in high-segregation neighborhoods their entire childhoods also experiencing lower parental educational attainment, higher rates of being raised by a single parent, higher rates of neighborhood poverty, and among the lowest average household incomes across childhood, on average.

Across segregation groups, 8–12% were in poor/fair health, 18–32% were classified as obese, and 36–40% had ever drunk alcohol (with 12–17% drinking more than once per week over the past year, and the average young adult binging 3–7 days in the past year). Between 14 and 19% had ever smoked cigarettes, 17–22% currently smoked, and 4–9% smoked more than 10 cigarettes a day.

Living in a high-segregation neighborhood was common. Seventeen percent (N=302) lived in a high-segregation neighborhood for their entire observed childhoods, an additional 8% (N = 141) moved into a high-segregation neighborhood from a low-segregation neighborhood, and 12% (N=219) lived in a high-segregation neighborhood before moving into a low-segregation neighborhood. About 37% (N = 675) lived in a low-segregation neighborhood for their entire observed childhoods.

Additionally, 27% of participants (N = 486) moved back and forth between high-segregation and low-segregation neighborhoods. Because this group represents many different trajectories, we include regression results for this group only in appendices.

Primary analysis: measuring associations with the duration and trajectory of segregation exposure

In models estimating associations between childhood residential segregation trajectories and young adult health (Figure 1; Appendix 1), all groups exhibited some health advantage over the always-highly segregated, particularly those who lived consistently in low segregation neighborhoods. Compared with Black young adults who lived in high-segregation neighborhoods for their entire observed childhoods (the reference group), Black young adults who lived in lower-segregation neighborhoods their entire childhoods exhibited healthier smoking and drinking behaviors, including lower odds of drinking more than once a week (OR=0.65; 95% CI=−0.48, 0.88; p=0.005), ever smoking cigarettes (OR=0.71; 95% CI=0.53, 0.96; p=0.028), currently smoking (OR=0.67; CI=0.47, 0.96; p=0.028), and

smoking more than 10 cigarettes per day (OR=0.51; CI=0.30, 0.87; p=0.014). Young Black adults who started out in a low-segregation neighborhood before moving into a high-segregation neighborhood also exhibited lower odds of ever smoking (OR=0.56; CI=0.35, 0.88; p=.012), as well as lower odds of obesity (OR=0.40; CI=0.25, 0.65; p<0.001). Those who started out in high-segregation neighborhoods before moving into a low-segregation neighborhood had lower odds of obesity (OR=0.64; CI=0.42, 0.98; p=0.038) than the always-highly-segregated; marginally significant results also offered weak evidence that high-to-low movers had lower odds of smoking more than 10 cigarettes per day (OR=0.50; CI=0.23, 1.07; p=0.074).

Secondary analysis: the developmental timing of moves between neighborhoods with different segregation levels

In secondary models, we restricted our sample to those whose segregation levels were stable during adolescence (Sample A) or stable during early childhood (Sample B). All moves made by Sample A occurred in early childhood, while all moves made by Sample B occurred during adolescence. This allowed us to assess whether the timing of moves between neighborhoods with different levels of segregation mattered (Figure 2, Appendices 3 & 5).

Differences in the coefficients produced were often sizable, excepting nearly identical associations for obesity. However, statistical power for formally comparing coefficients across these stratified models was low, given the smaller samples on which our secondary models were run. Qualitatively, movers appeared to experience larger health advantages in terms of smoking behaviors (compared to the always highly segregated) if they spent more of their childhoods in low segregation neighborhoods, but confidence intervals were consistent with equivalent effects across move timing. For example, low-to-high movers who moved in adolescence (spending most of their early years in a low-segregation neighborhood) were estimated to have 0.36 times the odds of ever smoking (CI=0.13–0.96; p=0.040) and 0.37 times the odds of currently smoking (0.13–1.05; p=0.062) compared to the always-highly-segregated; while equivalent estimates among low-to-high movers who moved in early childhood were 0.61 (CI=0.35–1.06, p=0.081) and 0.97 (CI=0.49–1.89, p=0.922), respectively.

Of note, the only trajectory that showed even weak evidence of an association with psychological distress was high-to-low segregation moves during adolescence. People in this group scored 0.78 higher points on the K6 scale (CI=−0.12–1.68; p=0.089) than those who were always highly segregated, on average. In comparison, the equivalent difference for high-to-low movers who moved during early childhood was effectively 0, at 0.03 points (CI=−0.67–0.72; p=0.941). Again, however, evidence of a unique effect for psychological stress among any group was inconclusive.

Sensitivity analyses

Results excluding neighborhood poverty and population size from our models yielded similar findings across all models (Appendices 2, 4, 6). Models where we clustered at the tract level instead of the individual level yielded more precise estimates but similar

conclusions. Because individuals and tracts were colinear for two thirds of the sample (1 participant per tract), models attempting to account for tract- and individual-level clustering simultaneously failed to converge.

To further assess differences between segregation trajectory groups, we also re-ran all models while changing the reference group to those who consistently lived in lower segregation neighborhoods (selected results in Appendix 7). Compared to this group, the always-highly-segregated exhibited unhealthier smoking and drinking behaviors, including higher odds of drinking more than once per week (OR=1.54; CI=1.14–2.08; p=0.005), ever smoking (OR=1.41, CI=1.04–1.91, p=0.028), currently smoking (OR=1.50, CI=1.05–2.15; p=0.028), and smoking more than 10 cigarettes per day (OR=1.97, CI=1.15–3.39; p=0.014). Those who moved from low- to high- or high- to low-segregation neighborhoods appeared to have similar average health to the always-low-segregation group, except that both groups of movers had lower odds of obesity.

DISCUSSION

In this study, we examined how a key structural determinant of health, childhood residential racial segregation, was associated with health in young adulthood among Black US Americans. To our knowledge, we are among the first to do so—especially, to account for the duration, trajectory, and developmental timing of segregation exposure. Black young adults who lived in lower segregation neighborhoods for their entire observed childhoods experienced healthier smoking and drinking behaviors than those who always lived in high-segregation neighborhoods as children. Children who started out in low-segregation neighborhoods and then moved into high-segregation neighborhoods experienced healthier smoking behaviors in young adulthood, too, as well as lower odds of obesity. Results weakly suggested that children who started out in high-segregation neighborhoods and moved into low-segregation neighborhoods similarly exhibited healthier average smoking behaviors if they moved into low-segregation neighborhoods earlier in childhood; this group also had lower odds of obesity. All groups, in other words, experienced health benefits relative to the always-highly-segregated.

Potential mediating paths

As per our conceptual diagram (Figure 3), residential racial segregation is a structural threat to the health of Black populations that becomes embodied through more proximate social, economic, and biologic processes. Past work suggests multiple such pathways that could explain associations between residential racial segregation and young adult health. First, racial isolation may increase experiences of institutional discrimination, causing psychosocial stress and poor mental health.[75] Such institutional discrimination, for example, could include increased discriminatory policing[20,22,76] or neighborhood stigma.[21,77] This chronic stress may then lead to unhealthy coping behaviors, or prioritizing behaviors that enable survival in the face of stress over behaviors that theoretically minimize health risks but are untenably burdensome on people's time and mental energy.[78–81] Chronic stress can also cause disease directly via dysregulated endocrine, immune, and nervous system function.[82–89] Second, the marginalization of segregated, predominantly Black

communities may lead to an unhealthy built environment, such as a dearth of nearby, high-quality healthcare providers,[33–37] targeted marketing of unhealthy products with fewer regulations (including cigarettes and alcohol),[26–28,90,91] lower geographic healthy food access,[92] increased exposure to air pollution,[29–32] or less access to green space[93,94] and opportunities for exercise.[95,96] Third, segregation may constrain social and economic opportunities, including lower quality schools.[38] This can worsen educational and economic outcomes, with broad health consequences.[97] Fourth, segregation may alter social networks and concentrate disadvantage within those networks, resulting in differing social norms (e.g., around tobacco initiation)[78] and inequitable access to social capital.[39] These mediating pathways are not well-captured in PSID; which of these paths (if any) explain the associations we observe is a question for future research.

Implications for segregation and health research

This study is novel in its findings that childhood segregation exposure *durations* and *trajectories* may matter for adult obesity, alcohol consumption, and smoking, providing a potential path towards explaining the diversity of past results using more limited segregation measures. Specifically, present results help clarify a muddled literature on segregation and obesity and flesh out a surprisingly weak literature on segregation and either smoking or alcohol consumption. For obesity, past results have been mixed and overwhelmingly cross-sectional.[42,57,98–100] Of those analyzing longitudinal data, the *duration* and *trajectory* of exposure have been critical: women followed longitudinally as part of the Black Women's Health Study, for example, showed increased chances of incident obesity only if they lived consistently in highly segregated neighborhoods throughout follow-up.[57,101] We similarly find that, for children, spending even a portion of one's childhood in a lower-segregation neighborhood may be protective against health risks associated with obesity—i.e., that *consistent* exposure to highly segregated neighborhoods stands out. Whether obesity is associated with poor health because more adipose tissue increases future chronic disease risk *per se*—e.g., as opposed to representing the effects of weight bias and fatphobia in healthcare, employment, and everyday life[102–106]—is beyond the scope of this paper. We find only that residential segregation is associated with obesity as a *predictor* of higher chronic disease risk, and that residential segregation, through obesity, may compound exposure to multiple forms of discrimination.

For smoking, few studies have been published; most examine adults, finding that higher adult segregation is associated with higher smoking prevalence,[107,108] while some that examine self-reported child and adult segregation levels find that only childhood exposure matters for adult smoking.[109] Our results indicate that Black children who live in highly segregated neighborhoods consistently throughout childhood are at unique risk of unhealthier smoking behaviors in young adulthood, including higher risk of ever smoking, currently smoking, and smoking more than 10 cigarettes per day. Potentially core to this finding is research documenting that predominantly Black, segregated neighborhoods are specifically targeted by tobacco companies, including a higher density of tobacco retailers in these neighborhoods and predatory marketing of menthol cigarettes, which are both more addictive and more harmful to health.[110,111]

The link between alcohol consumption and residential segregation remains understudied; our evidence is among the first to directly examine this question through a life course lens. Though numerous studies agree that residential segregation is associated with increased alcohol advertising and higher alcohol retailer density,[14,90,91,112–114] few have examined whether this translates into higher alcohol consumption, with inconclusive results.[115,116] Currently, Black US Americans tend to begin drinking later in life than their White counterparts and to drink less on average, although Black US Americans also suffer higher rates of alcohol-related morbidity and mortality.[117] Thus, living in a neighborhood of predominantly Black residents, even in the presence of excess alcohol retailers and marketing, could plausibly be protective. Our study clarifies that, irrespective of neighborhood and family poverty, consistent exposure to residential segregation in early life is associated with substantially higher odds of regular drinking as an adult.

Lastly, we find no evidence of a statistically significant association between any residential racial segregation trajectory and mental health. This is consistent with a body of research suggesting that residential racial segregation may be protective for mental well-being or even mortality among Black US Americans,[14,118–120] given that segregated Black neighborhoods may offer higher social cohesion, a greater sense of belonging, and lower exposure to interpersonal racism.[121–125] Our null results for mental health could thus reflect that residential racial segregation may have both deleterious (e.g., being targeted for disinvestment and unhealthy product marketing) and protective (e.g., more social support) impacts on residents' health. These countervailing forces may also help explain our null findings on self-rated health despite a worsened health profile in terms of drinking, smoking, and markers of cardiometabolic risk like obesity. Further, lack of evidence for an association with mental health could indicate that stress-driven coping may not serve as the major driver of unhealthier smoking or drinking behaviors for the always-highly-segregated (as opposed to, e.g., targeted marketing to Black neighborhoods by tobacco and alcohol retailers).

Our findings also have important implications for measuring residential racial segregation as an exposure, at least for Black US Americans' health. First, both the directionality and timing of exposure to childhood residential racial segregation appear to matter for predicting later health risks. A single summary score (such as an average across childhood), or a single point-in-time measurement, is likely to miss important heterogeneity between children's segregation exposures. For instance, a single point-in-time measurement would likely conflate children who lived in high-segregation neighborhoods for their entire childhoods and children who only lived in a high-segregation neighborhood for part of their childhoods, groups whose health risks diverge as they age. Second, these findings add to the body of evidence indicating that studies of segregation and health later in life are likely to be confounded by childhood segregation.[46,48,126]

Limitations and strengths

This study has limitations. First, outcomes and covariates were self-reported and may suffer from reporting biases. Second, this is an observational study; associations cannot be assumed to be causal. Specifically, it likely suffers from bias due to confounding by unmeasured characteristics of individuals or neighborhoods (e.g., unmeasured aspects

of socioeconomic position) and selection (people with different segregation trajectories, particularly movers, may be more likely to attrit from PSID, as might people in poor health). Third, we have little information on what precipitated childhood moves between neighborhoods with different levels of segregation (e.g., whether a move was the result of an eviction or the result of a family purchasing a new, more spacious home), and so cannot examine heterogeneity by move type. Fourth, we do not have complete residential histories for every child, meaning trajectories may be misclassified. We nonetheless benefit from observing the vast majority of our sample 10 or more times across childhood, such that our PSID data is among the most complete records of child residential history in a cohort study. Fifth, our measures of segregation for years after 2010 used ACS data (a sample of the population), which may not be perfectly comparable to measures of segregation in earlier years using decennial data (a full count). However, estimates from both the decennial Census and 5-year estimates are comparably robust and are both considered reliable.[56] Additionally, we operationalize neighborhoods as Census tracts, the geographic level at which most Black-non-Black segregation occurs in US cities;[127] but these may not perfectly align with neighborhood boundaries as they are understood by local area residents. Finally, PSID represents Black US Americans whose families have been living in the US since the 1960s. While this reflects families exposed to anti-Black US public policy for generations, their experience may not be generalizable to more recent Black immigrants.

Using local Getis-Ord G* statistics to measure segregation allowed us to test whether anti-Black residential segregation was associated with health disadvantages on top of those that accrue via neighborhood poverty—i.e., we are able to enter racial segregation and neighborhood poverty as separate variables in our models. While not a limitation, we note that other studies using different metrics—such as the Index of Concentration at the Extremes, often used to jointly measure racial and economic segregation—may offer distinct insights.[128]

Our study also has important strengths. It is among the first to examine the influence of residential segregation exposure in childhood on later-life health and chronic disease risk,[46,48] and in particular to focus on segregation trajectories and their developmental timing. It does so using uniquely rich data and careful exposure conceptualization: we used a national cohort followed longitudinally for up to 27 years, using neighborhood-level segregation measures derived from repeated, (semi)annual observations that captured detailed segregation trajectories. While the present study is observational, experimental studies in this field are largely infeasible and would be unethical; this paper represents a rigorous investigation on an understudied research question. Its findings provide urgent new evidence on the health of Black Americans who live in highly segregated neighborhoods produced by the US' ongoing legacy of anti-Black racism.

Implications for policy and practice

Our results suggest that early exposure to residential segregation has a lasting influence on Black US Americans' chronic disease risk—even now, more than half a century since housing discrimination on the basis of race was declared illegal. This increased risk appears distinct from that of neighborhood or family poverty, implying that race-blind interventions

for children living in low-income neighborhoods would miss the ways that highly segregated communities are specifically targeted for underinvestment[38,71 19,129–133] and experience distinctly heightened risk of poor future health.

The exact nature of the policy interventions required to correct this added risk and protect the well-being of segregated Black communities are beyond the scope of this paper. Still, we urge policymakers to think beyond intervening solely on health behaviors without intervening on their causes. Residential segregation is not inevitable, and is the result of (often deliberate) policy choices that can be undone.[19] Solutions could include adding financial teeth to the 1968 Fair Housing Act's requirement to "affirmatively further fair housing," banning single family zoning or rental discrimination against Section 8 holders, or increasing Low-Income Housing Tax Credits for developers building in integrated neighborhoods, among other reforms.[19] Nor is it inevitable that Black people living in community with other Black people would be at higher risk of chronic illness. Rather, these excess risks are driven by structural and systemic racism against Black communities that manifest in specific policy regimes undertaken by specific federal and state governments—regimes constructed by modifiable choices.[13,14,19]

Finally, we urge caution around misinterpreting our trajectory results as advocating for special housing vouchers to move out of segregated neighborhoods, in the style of the Moving to Opportunity experiment.[54] Such mobility programs can backfire: adolescent boys assigned to the Moving to Opportunity treatment group, for example, suffered poorer mental health and higher levels of substance use.[134] The present paper's results likewise show weak, suggestive evidence of a similar dynamic for adolescent moves, wherein adolescent moves from high- to low-segregation neighborhoods are associated with higher levels of adult mental distress. More to the point, while some individual children's health may benefit from these moves, these kinds of vouchers are not likely to be a lasting solution to what are ultimately community-level exposures affecting everyone who continues to live in segregated neighborhoods. In other words, taking children out of their communities is not a sustainable (or just) answer to the health inequities we observe; rather, policymakers must actively undo the structural oppression under which segregated communities live.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Black children consistently residing in segregated areas have worse adult health
- Even Black children who move into or out of segregated areas fare better as adults
- Long-term childhood segregation linked to adult smoking, drinking, obesity
- The developmental timing of moves to more- or less-segregated areas may matter
- Results are robust to adjustment for neighborhood and family poverty in childhood

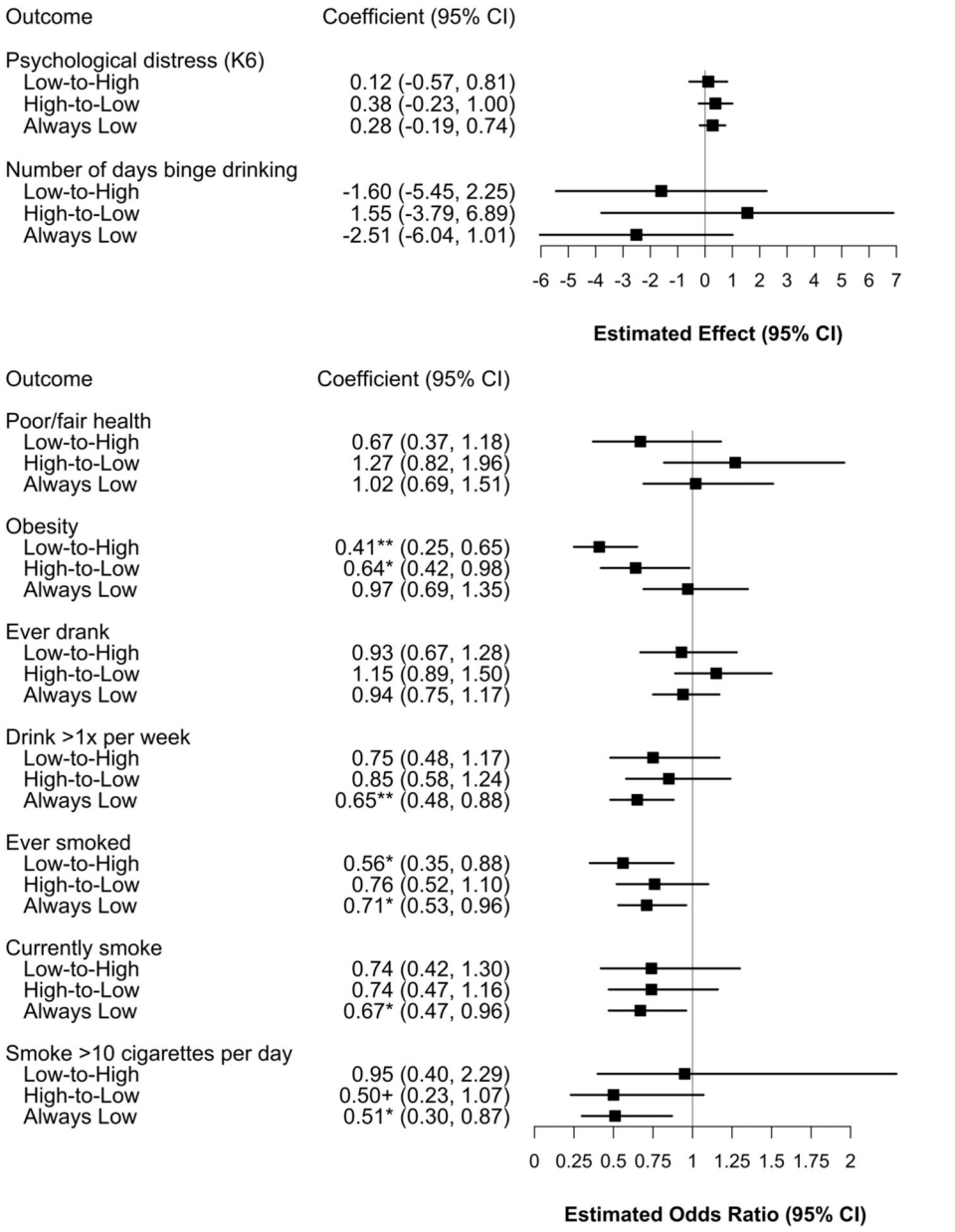

Figure 1.

Estimated associations between childhood segregation trajectories and health, comparing those who always lived in high-segregation neighborhoods during childhood (the reference) to all other segregation trajectory groups

Note: Results for each health outcome are derived from separate multiple regression models, run on 4,925 observations contributed by 1,823 Black TAS participants. SEs were clustered at the individual level to account for repeated outcome observations. Those who always lived in high-segregation neighborhoods are the reference group.

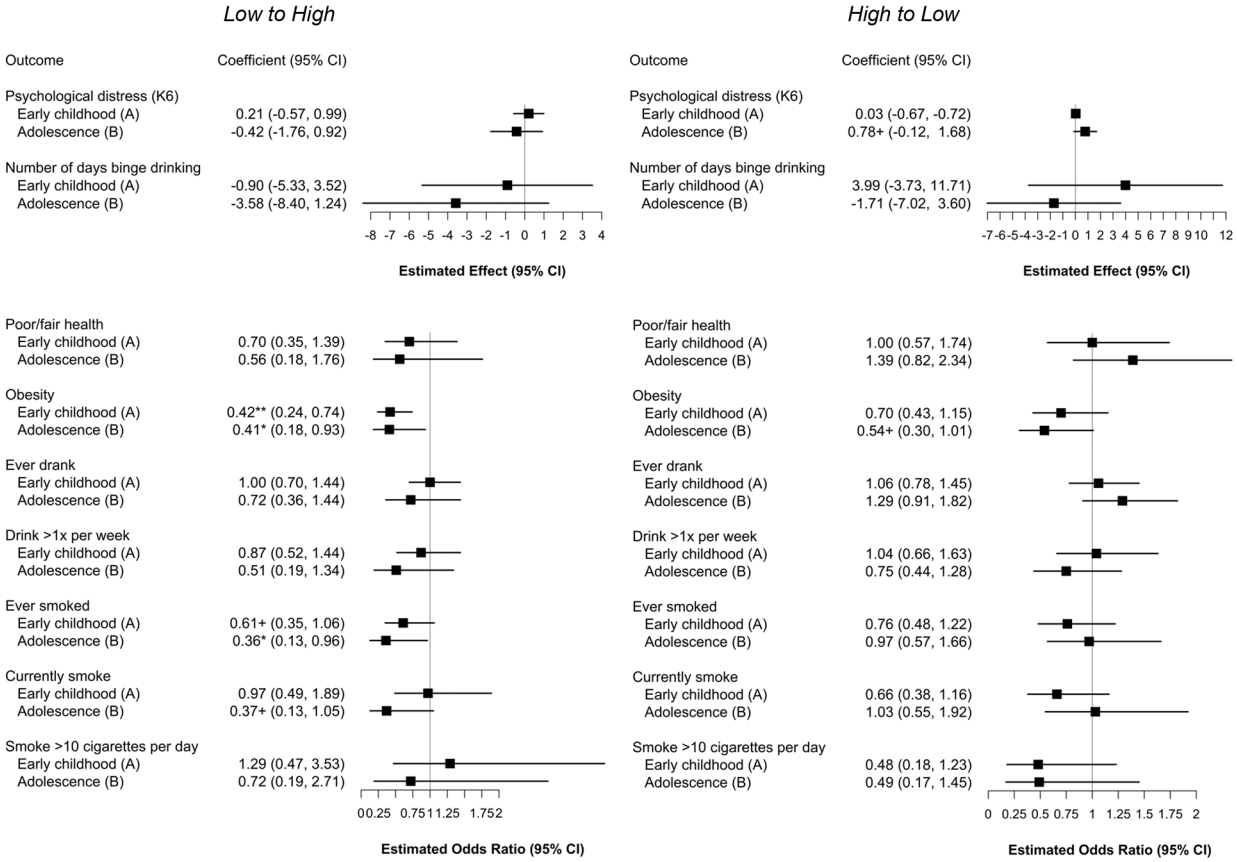

Figure 2.

Associations between segregation trajectories and health by move timing (comparing movers to the always-highly-segregated)

Note: Results for each health outcome are derived from separate multiple regression models with standard errors clustered at the individual level. For Sample A, models were run on up to 3,769 observations contributed by 1,377 Black TAS participants. For Sample B, models were run on up to 3,064 observations contributed by 1,157 Black TAS participants. Those who always lived in high-segregation neighborhoods are the reference group.

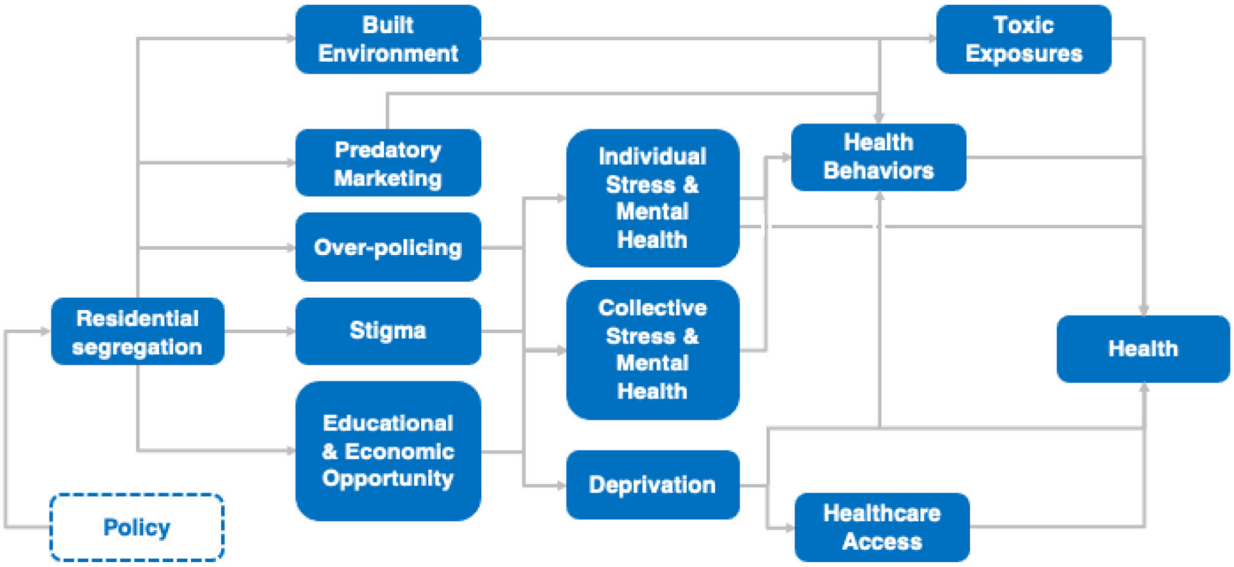

Figure 3.
Hypothesized mediating paths between childhood residential segregation exposure and young adult health

Table 1.

Sample characteristics by childhood residential segregation trajectories (means/prevalences, with SDs in parentheses for continuous variables)

Variable	Always high segregation	Moved: Lower to high segregation	Moved: High to lower segregation	Always lower segregation	Others*
Birthyear	1991.9	1990.9	1991.4	1991.0	1991.9
	(4.2)	(4.4)	(4.3)	(4.5)	(4.7)
Female	48.3%	53.9%	51.6%	49.9%	50.6%
Family income**	$39,231.47	$41,365.94	$38,217.20	$43,261.86	$33,296.99
	($31,930.60)	($34,261.18)	($30,565.28)	($35,241.84)	($28,031.35)
Parental education (grades completed)					
<12	17.2%	7.8%	13.2%	11.3%	11.1%
12	34.1%	35.5%	28.8%	33.9%	31.9%
13–15	28.1%	33.3%	34.2%	35.1%	39.1%
16+	20.5%	23.4%	23.7%	19.7%	17.9%
Parents ever married	57.6%	67.4%	67.1%	72.1%	59.3%
Neighborhood poverty***	26.9%	23.1%	22.5%	21.0%	23.9%
	(10.4%)	(17.3%)	(9.6%)	(9.7%)	(9.2%)
Neighborhood pop.***	4139.6	4206.6	4407.3	4455.1	4436.5
	(1294.1)	(1190.0)	(1147.5)	(1357.0)	(1113.3)
Health Outcomes					
Health(poor/fair)	11.2%	8.0%	12.0%	10.1%	11.2%
Obese	29.8%	17.9%	22.6%	31.9%	25.4%
K6 psychological distress scale (0–24)	4.9	5.0	5.2	5.0	5.1
	(3.6)	(4.1)	(4.0)	(3.9)	(4.1)
Alcohol consumption					
Ever drank	37.9%	35.9%	40.2%	35.8%	35.5%
Drinks > 1X / week	16.8%	15.5%	17.0%	14.2%	12.2%
Number of days binge drinking / year	4.5	5.0	7.1	3.7	3.3
	(28.9)	(29.2)	(39.2)	(18.5)	(21.9)
Smoking					
Ever smoked	18.8%	13.9%	14.8%	15.5%	14.3%
Currently smoke	22.2%	22.1%	18.7%	18.5%	16.9%
Smoke >10 cigarettes / day	6.1%	8.8%	4.3%	4.8%	4.6%
N	*302*	*141*	*219*	*675*	*486*

Note: Sample characteristics represent means or prevalence at baseline (the earliest observed childhood observation, unless otherwise specified) for Black TAS participants, who were aged 18–28 years during at least one TAS wave from 2005 to 2017. For continuous variables, standard deviations are included in parentheses.

* Participants in this category moved back and forth between high-segregation and lower-segregation neighborhoods throughout childhood.

** Family income is calculated as the average family income across childhood observations in inflation-adjusted USD.

*** Neighborhoods are Census tracts, normalized to 2010 tract boundaries