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Skin Tone and the Health Returns to Higher Status

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ABSTRACT This study addresses two questions. First, why do Black Americans exhibit worse health outcomes than White Americans even at higher levels of socioeconomic status (SES)? Second, are diminished health returns to higher status concentrated among Black Americans with darker skin color? Novel hypotheses are tested with biosocial panel data from Add Health, a nationally representative cohort of Black and White adolescents who have transitioned to adulthood. We find that White and light-skin Black respondents report improved health after achieving higher SES, on average, while their darker-skin Black peers report declining health. These patterns persist regardless of controls for adolescent health status and unmeasured between-person heterogeneity. Moreover, increased inflammation tied to unfair treatment and perceptions of lower status helps to account for patterns of diminished health returns for dark-skin Black groups. Our study is the first to document skin tone heterogeneity in diminished health returns and one of few studies to identify life course stress processes underlying such disparities. We consider additional processes that could be examined in future studies, as well as the broader health and policy implications of our findings.

KEYWORDS Black–White disparities • Biosocial • Colorism • Diminished returns • Skin tone

Introduction

> The real cost of oppressive structures is not measured in dollars or occupational status. It is measured in the tears that flow behind closed doors, the blood that is spilled without understanding, the anguish that suffuses experience and comes to be who one is . . . . Damaged human relationships, not money or status—*that* is the cost of oppression, to oppressor and oppressed alike.
> —Samuel Lucas, *Theorizing Discrimination in an Era of Contested Prejudice* (2009:x)

Despite notable declines in racialized health disparities over the twentieth century, Black Americans continue to live sicker and shorter lives than their White peers. Black Americans are more likely to suffer from functional limitations and other chronic illnesses over the life course, such as hypertension, diabetes, and kidney disease (Hummer and Hamilton 2019). Black Americans could also expect to live four years less, on average, than White Americans in 2018 (Arias

and Xu 2018). This gap has since widened to six years in the wake of COVID-19 (Arias et al. 2021).

Black–White health disparities are often thought to reflect interracial inequalities in socioeconomic status (SES). According to fundamental cause theory, groups with higher levels of education, income, and wealth can access more health-promoting resources than their less privileged peers, and thus live longer and healthier lives (Hajat et al. 2011; Hummer and Hernandez 2013; Link and Phelan 1995). Given that Black Americans remain disadvantaged across multiple socioeconomic strata relative to White Americans, it stands to reason that unequal access to SES resources could underlie Black–White health disparities. For example, only 24% of Black American adults have a college degree, compared to 38% for White Americans (U.S. Census Bureau 2020). Black families also own virtually no liquid assets, and a meager five to ten cents in intergenerational wealth for every dollar of wealth owned by the average White family (Hamilton et al. 2015).

While SES is no doubt important for health and longevity, a growing body of research has demonstrated that race and SES *interact*, resulting in persistent and sometimes even widening Black–White health disparities across SES levels—what has been termed "diminished health returns" (DHRs) for Black Americans (Assari 2018; Assari and Caldwell 2021; Boen 2016; DeAngelis 2021; Esposito 2019; Farmer and Ferraro 2005; Gaydosh et al. 2018). The purpose of our study is to help advance the DHRs literature and challenge the assumption that socioeconomic inequalities are driving Black–White health disparities in the United States. The perspective advanced here points to the ongoing specter of *anti-Black stigma* as an inextricable component of SES attainment for Black Americans, one that can create gratuitous stress burdens that ultimately suppress the otherwise beneficial health effects of higher status (DeAngelis 2021).

Our study addresses three current limitations of the DHRs literature. First, almost all studies in this area have utilized cross-sectional data, making it difficult or impossible to decompose the unique effects of SES and racism on health (for rare exceptions, see Boen 2016; Colen et al. 2018; Esposito 2019). Second, few studies have identified the biopsychosocial processes contributing to DHRs (Goosby et al. 2018). One commonly proposed mechanism is chronic unfair treatment, but we still know very little about whether or to what extent discrimination accounts for DHRs among higher status Black Americans (DeAngelis 2021). Finally, no study in this area has tested for skin tone heterogeneity in DHRs. This oversight is noteworthy given legacies of colorism in the United States, or the systematic preferential treatment of persons with White (European) phenotypes. What began centuries ago as an ideological legitimation of chattel slavery, colorism has persisted into the modern era, allowing lighter-skin Black Americans to integrate into mainstream society and achieve higher levels of SES and health relative to their dark-skin peers (Dixon and Telles 2017; Hunter 2007; Keith and Herring 1991; Monk 2014, 2015). Ongoing legacies of colorism suggest that light skin may help to buffer discrimination for Black groups striving for higher status in a White supremacist society like the United States (Monk 2015; Pearson 2008).

Leveraging multiple waves of biosocial data from a nationally representative cohort of Black and White adolescents who have transitioned to adulthood, we

develop and test a model that accounts for life course stress processes contributing to DHRs for Black Americans. We also test for skin tone heterogeneity in DHRs. Our two primary research questions are: (1) What biopsychosocial mechanisms explain DHRs among higher SES Black Americans? (2) Are DHRs concentrated among darker-skin Black Americans?

We first develop two hypotheses for the adverse health effects of discrimination among higher status Black Americans. We then explain why darker-skin Black Americans may be exposed to more discrimination than their lighter-skin peers, especially in high-status contexts. Next, we present our conceptual model, research methods, and findings. We close by considering some broader implications of our findings, limitations of the current study design, and avenues for future research into additional mechanisms of DHRs.

Background

Status Incongruence Hypothesis

One commonly proposed mechanism of diminished health returns is chronic unfair treatment or interpersonal discrimination. Given the well-established links between discrimination and poor health among Black Americans, this mechanism appears highly plausible (Goosby et al. 2018; Williams et al. 2019). However, as stated earlier, few studies in this area have explicitly tested discrimination-related stressors as mechanisms of DHRs.

To be sure, some studies have found that higher SES Black Americans tend to report more discrimination than their White peers, and sometimes even relative to their lower status Black peers (Assari 2020; Assari et al. 2021; Assari and Lankarani 2018). One recent longitudinal study by Colen and colleagues found that Black Americans who achieved higher income levels over time tended to self-report worse health than their White counterparts, and that these health gaps were reduced after accounting for group differences in perceived discrimination (Colen et al. 2018). Another cross-sectional study based in Nashville, Tennessee, found that Black Americans who lived in Whiter and higher status block groups reported more chronic unfair treatment relative to their Black peers living in disadvantaged Black areas, which appeared to suppress the health benefits of living in these high-status communities (DeAngelis 2021; see also Assari et al. 2018).

Although the aforementioned studies have expanded our knowledge of DHRs, we still lack a clear understanding of how discrimination operates on the mind and body to suppress the health benefits of higher status. One promising starting point for this type of inquiry is found in the closing discussion of Farmer and Ferraro's (2005) classic study, in which the authors proposed the *status incongruence hypothesis*. The basic idea is that ongoing exposures to discrimination in high-status contexts can create perceptions of relative deprivation and lowered status, or a sense of being blocked from reaching full status equality (see also Dressler 1996). Indeed, studies have found that discrimination is associated with higher goal-striving stress among high-SES Black Americans—namely, the perception of an achievement–aspiration

gap with barriers to success—which then predicts worse mental and physical health (DeAngelis 2021; Sellers and Neighbors 2008).

While the diminished returns hypothesis contends that discrimination suppresses the health benefits of higher status for Black Americans, the status incongruence hypothesis further stipulates that perceived social exclusion and low status mediate the health impacts of discrimination. This premise is further supported by decades of research on subjective social status and health. Survey researchers measure subjective status by showing respondents an image of a ten-rung ladder. Respondents are instructed to imagine that the top and bottom rungs represent some type of "best" and "worst" status, respectively, and are then asked to rank themselves on the ladder. Akin to other common measures of subjective well-being (e.g., Diener et al. 1985), ladder scales have been shown to gauge a respondent's cognitive averaging of their achievements and aspirations relative to salient social reference groups (Andersson 2015). Moreover, people who rank themselves lower on the ladder tend to exhibit worse health outcomes cross-sectionally and over time, regardless of their education, occupation, or income levels (Hoebel and Lampert 2020).

Social Pain Hypothesis

The status incongruence hypothesis suggests that DHRs among higher SES Black Americans are at least partially explained by *perceptions of lowered status* resulting from chronic unfair treatment. What this hypothesis does not account for is how perceived low status, in turn, harms physiological systems and eventually undermines health. To address this open question, we turn to recent advances in social neuroscience that have revealed links between discrimination, perceived low status, and regions of the brain and nervous system associated with chronic inflammatory stress responses (Eisenberger 2015; Goosby et al. 2018; Muscatell et al. 2016).

Human beings, like many other mammals, have evolved to be highly sensitive to cues of social rejection (Eisenberger 2013; MacDonald et al. 2005; Snyder-Mackler et al. 2020). This is because exclusion from a group was equivalent to a death sentence throughout most of our evolutionary past, when our ancestors traveled in small and highly interdependent hunter-gatherer groups. These small-group dynamics are thought to have created selection pressures for humans to develop greater capacities for accurately inferring the mental states of others, as well as one's ranking in group hierarchies (Massey 2001). According to *social pain theory*, these same evolutionary processes also resulted in overlapping brain regions that encode physical and socioemotional pain—namely, rejection—in similar manners (Eisenberger 2013, 2015; MacDonald et al. 2005).

Neuroscientists have identified several brain regions that encode social evaluative threats, each of which connects to branches of the nervous system that trigger the release of hormones such as epinephrine and cortisol in response to such threats (Muscatell and Eisenberger 2012). In acute stress responses, cortisol serves the adaptive role of suppressing long-term immune and growth functions and quickly redirecting energy stores, such as fat and glucose, to help our bodies mount defenses to stressors (Spencer and Deak 2017). Whenever stressors persist for days or months,

however, our bodies can become desensitized to the anti-inflammatory effects of cortisol, leading to a rapid reproduction of pro-inflammatory cytokines and, ultimately, chronic inflammation (Dhabhar 2009; Miller et al. 2002). Chronic inflammation, in turn, has been linked with accelerated aging and health decline (Chung et al. 2009; Franceschi and Campisi 2014; Pawelec et al. 2014).

The social pain hypothesis further qualifies the diminished returns hypothesis. Accordingly, unfair treatment undermines the health of Black Americans by triggering *chronic inflammation* tied to perceptions of low status. In support of this hypothesis, numerous studies in the United States and abroad have uncovered links between inflammation biomarkers, strained social relationships, and low subjective status (Dressler et al. 2016; McDade 2002, 2005; Muscatell et al. 2020; Yang et al. 2014; Yong et al. 2021). One study found that older-age Black Americans who reported more discrimination exhibited higher levels of inflammatory biomarkers (Lewis et al. 2010). Another previously mentioned study found that Black residents of high-status areas in Nashville reported more discrimination than their peers in disadvantaged Black areas, which then predicted higher levels of goal-striving stress, neuroendocrine hormones, and self-reported bodily pain, a common symptom of chronic inflammation (DeAngelis 2021).

Colorism Hypothesis

We have argued that chronic inflammation tied to unfair treatment and perceived low status could be contributing to DHRs among higher SES Black Americans. The perspective advanced thus far assumes, however, that all Black Americans will be equally exposed to unfair treatment in their striving for higher status. Parallel literatures in the social neuroscience of intergroup prejudice, as well as in the sociology of colorism, challenge this assumption and point, instead, to an increased risk of exposure for darker-skin Black Americans.

Intergroup prejudice appears to comprise distinct "bottom-up" and "top-down" cognitive processes (Kawakami et al. 2017). Bottom-up processes refer to the implicit or split-second perceptions individuals form of other people, which are usually predicated on phenotypical cues such as skin color. It appears many of the same brain structures that have evolved to help us interpret others' mental states, as referenced earlier, also allow individuals to quickly discriminate between perceived "in-group" and "out-group" members. Our capacities for categorizing people in this way are thought to have served a vital need in prehistoric environments to swiftly identify unfamiliar and thus potentially dangerous outsiders (Amodio and Cikara 2021:176).

Importantly, experimental work also suggests that our brains process skin tone cues more readily than other bodily features, leading us to classify people on the basis of racial group memberships even quicker than other social identities like gender (Amodio and Cikara 2021:173). As we draw upon skin tone distinctions, moreover, we determine not only *whether* a given person falls into a certain racial group, but also the *extent to which* that person matches a prototypical member of the target group (Maddox 2004; Monk 2022). For instance, experimental studies show that participants are more inclined to label darker-skin faces "African American," even when

facial structures are manipulated to be more or less prototypically African, thereby indicating that people primarily associate "African American" with "dark skin" (Stepanova and Strube 2009, 2012).

Interpersonal racial categorizations are also influenced by top-down cognitive processes, whereby a person's prior assumptions and broader social-environmental contexts can modulate their implicit cognitions (Kawakami et al. 2017; Payne et al. 2017). One critical contextual factor that is liable to skew judgments of dark-skin persons is *colorism*, a hegemonic ideological system that has served to advantage persons with lighter skin for centuries. As a global system of stratification, colorism originated with European colonialism and slavery in the Americas as a tool to subjugate Black and Brown people, specifically by igniting within-group divisions to preclude a sense of collective identity and linked fate. Colorism also established and perpetuated White dominance by linking dark skin with danger, savagery, and incompetence—deeming dark-skin persons uncivil and unworthy of freedom—while linking light skin with moral virtue, civility, beauty, and intelligence—deeming light-skin persons as innately entitled to socioeconomic resources and privileges (Dixon and Telles 2017; Harris 1993; Russell et al. 1992).

Colorism has ultimately become a complementary derivative of racism in the United States, solidifying hegemonic beliefs in the superiority of White (Northern European) culture and aesthetics (Hunter 2007; Kang 1997). Preferences for Whiteness have translated into darker-skin Black Americans receiving systematically harsher treatment and restricted access to opportunities compared to their light-skin Black counterparts (Hunter 2007; Keith and Herring 1991; Monk 2014; Reece 2018). Relative to their light-skin peers, dark-skin Black Americans are attributed more negative stereotypes by individual raters in experiments (Maddox 2004; Stepanova and Strube 2012); are misrepresented or portrayed negatively in the news and in textbooks (Dixon and Maddox 2005; Louie and Wilkes 2018); and have a higher probability of being arrested and serving longer prison sentences (Monk 2019; Viglione et al. 2011). Studies also find that dark-skin Black Americans exhibit worse health over the life course (Cobb et al. 2016; Hargrove 2018a, 2018b), owing at least partially to increased exposure to colorism-related stressors (Monk 2015).

Further evidence indicates that colorism biases will be particularly salient in high-status and historically White spaces, such as prestigious universities and occupational settings. According to social psychological theories of "prejudiced places" and "biased crowds," implicit anti-Black biases reflect context-specific states of individuals rather than stable personality traits, and thus tend to become amplified in spaces with legacies of excluding or oppressing Black people (Murphy et al. 2018; Payne et al. 2017). For example, counties and states that were more dependent on slavery before the Civil War still exhibit higher aggregate levels of implicit pro-White bias among White citizens, "suggesting that intergroup stereotypes and attitudes are more likely to be automatically triggered in those areas" (Payne et al. 2019:11697).

Sociologists have made similar observations. In his seminal article "The White Space," Elijah Anderson noted that dark-skin persons in historically White spaces often must go to great lengths to prove themselves to White people, who "stigmatize anonymous Black persons by associating them with the putative danger, crime,

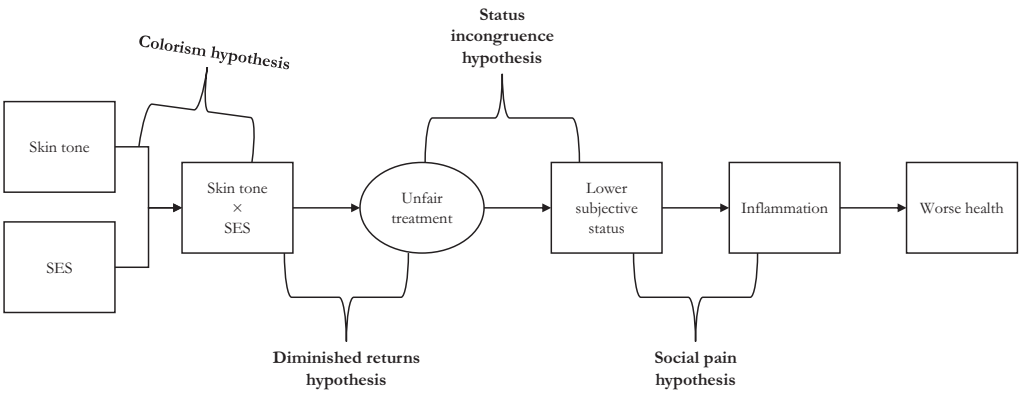

Fig. 1 Conceptual model of study hypotheses

and poverty of the iconic ghetto" (Anderson 2015:13; see also Anderson 2021). Ray (2019) likewise argued that Whiteness operates as a valued social credential in prestigious organizations, permitting expanded agency for White and White-passing persons (see also Harris 1993). Similarly, Monk (2015:415) suggested that the "salience and consequentiality of [skin tone] depends on the particular demographic composition of the various fields or settings individuals find themselves in." According to Monk, light skin tone can be thought of as a form of "bodily capital" within predominantly White social spaces, or a buffer against systemic anti-Black stigma and exclusion.

The colorism hypothesis suggests that *darker-skin* Black Americans will experience greater racism-related distress in their striving for higher status. This is because higher status spaces have been and continue to be dominated by White and White-passing persons, who are likely influenced by implicit pro-ingroup/anti-outgroup biases rooted in skin tone distinctions. When aggregated at higher levels of social organization, even subtle manifestations of pro-White/anti-Black bias can have dire implications for dark-skin persons. This can be evidenced, for instance, by studies showing that cities with higher aggregate scores of implicit anti-Black biases also tend to exhibit larger Black–White disparities in police shootings (Hehman et al. 2018).

Conceptual Model

Drawing on the literatures reviewed, we present the conceptual model depicted in Figure 1. This model represents a serially mediated moderation stress process. First, the colorism hypothesis suggests that *dark skin tone* will moderate the health returns to higher status, such that darker-skin Black Americans will derive fewer health benefits than their White or light-skin Black peers. Second, the diminished returns hypothesis entails that *unfair treatment* will mediate DHRs, particularly among dark-skin Black Americans. Third, the status incongruence hypothesis suggests that *low subjective status* will mediate the health effects of unfair treatment. Finally, the social pain

hypothesis predicts that *chronic inflammation* will mediate the health effects of low subjective status. Unfair treatment is enclosed within a circle to reflect that this variable will be measured as a latent construct with multiple indicators.

Methods

Data

Data come from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a nationally representative cohort of adolescents who were enrolled in grades 7–12 during the 1994–1995 school year, and they have been followed for five waves into adulthood (Harris et al. 2019). Our analyses include data from Wave I (1994–1995; ages 12–19), Wave III (2001–2002; ages 18–26), Wave IV (2008; ages 24–32), and Wave V (2016–2019; ages 33–43). Our analytic sample includes respondents who self-identify as Black or White and who have valid longitudinal weights and measures of skin tone. We exclude 238 White respondents who are documented as having darker than white skin to prevent ambiguity in our between-group comparisons. Our main findings are comparable when these respondents are included. Our final analytic sample consists of Black respondents with varied skin tones and White respondents with only white skin tone, all of whom participated in Waves I, III, IV, and V of the study ($N = 7{,}371$).

Measures

Health Status

Our key health outcome is self-rated health across all waves. Respondents are asked at each wave, "In general, how is your health?" Response options range from poor (1) to excellent (5) and are coded such that higher scores reflect better health. While not a clinical measure, self-rated health is generally accepted by population scientists as a holistic appraisal of health, "shaped by numerous health mechanisms and biological processes" (Gutin 2018:265). Self-rated health also appears to be a reliable predictor of subsequent morbidity and mortality and is commonly employed in the DHRs literature (Colen et al. 2018; Farmer and Ferraro 2005; Idler and Benyamini 1997). Our use of the measure is unique, however, in that we also test for biopsychosocial processes that help to account for disparities in self-reported health between different race/skin tone and SES groups.

Socioeconomic Status

We measure SES with multiple indicators of education, personal earnings, and occupational prestige. Education and earnings are measured at Waves III, IV, and V, while occupational prestige is measured only at Wave IV. *Education* is an ordinal measure with five categories: less than high school (0), high school/GED (1), some college (2),

college (3), and postgraduate (4). To measure *personal income*, respondents are asked at Waves III and IV to report their annual earnings in dollars, or to select their "best guess" from a list of ordinal categories. At Wave V, they are asked only to select their income from a list of ordinal categories. We utilize these measures to create ordinal indicators of income at each wave, ranging from "less than $10,000" (0) to "$200,000 or more" (10). *Occupational prestige* is measured at Wave IV with a single scale reflecting the average of two Hauser and Warren Occupational Income and Occupational Education scores, with higher scores indicating a larger weighted average of earnings and education levels associated with respondents' reported occupations. The analytic sample mean is 98.70, with a range of 21.38 to 179.51. To facilitate structural equation model convergence (see later), scores are rescaled to have a range of 0 to 1. More information on the rationale and construction of prestige scores in Add Health can be found in Belsky et al. (2020).

Race and Skin Tone

Respondents are asked at Wave V, "What is your race or ethnic origin?" They are then given the option to choose multiple racial-ethnic identifications from a list that includes Black/African American, Hispanic, Asian, Pacific Islander, American Indian/Alaska Native, "other" race, and White. Respondents who choose more than one race are asked, "Of the race/ethnicity categories you selected, please pick the one with which you most strongly identify." From these questions, we create categories for non-Hispanic Black and White.[1] Skin tone is interviewer-reported and measured once at Wave III. Categories include "black," "dark brown," "medium brown," "light brown," and "white." We collapse black/dark brown and light brown/white skin tone groups, resulting in three groups of Black respondents with dark, medium, and light skin, who are compared to White respondents with white skin.[2]

Mechanisms

We measure unfair treatment, subjective social status, and inflammation with a combination of survey and biomarker indicators recorded at Waves IV and V. First, we measure *unfair treatment* with six indicators recorded on two separate occasions in adulthood. At Wave IV, respondents are asked, "In your day-to-day life, how often do you feel you have been treated with less respect or courtesy than other people?" At Wave V, respondents are asked to report how often in their day-to-day life: (1) they are treated with less courtesy or respect than other people; (2) they receive poorer service than other people at restaurants or stores; (3) people act as if they are not smart; (4) people act afraid of them; and (5) they are threatened or harassed. Response options for all items range from "never" (0) to "often" (3). Because these items do not set temporal bounds on unfair treatment (e.g., within the past 12 months), we rely

[1] Respondents with missing data on Wave V race-ethnicity were assigned their Wave I racial-ethnic identity ($n = 79$).

[2] Findings were similar for Black respondents with black and dark brown skin. Also, less than 1% of Black respondents were rated as having white skin.

on respondents' multiple retrospective accounts to calculate a latent variable of unfair treatment in adulthood, which is purged of idiosyncratic errors associated with different questions and interview periods (Bollen 1989).

The second mechanism is *subjective social status* measured at Wave IV. Respondents are presented with an image of a ten-rung ladder. They are then read the following prompt:

> Think of this ladder as representing where people stand in the United States. At the top of the ladder (step 10) are the people who have the most money and education, and the most respected jobs. At the bottom of the ladder (step 1) are the people who have the least money and education, and the least respected jobs or no job.

Respondents are then asked, "Where would you place yourself on this ladder? Pick the number of the step that shows where you think you stand at this time in your life, relative to other people in the United States." Response options range from 1 to 10 and are coded such that higher scores reflect higher subjective social status.

The third and final mechanism is a biomarker of high-sensitivity *C-reactive protein* (CRP) collected at Wave IV. CRP is a stable protein produced by the liver during an inflammatory response and can be measured precisely through standardized laboratory protocol (Whitsel et al. 2020). CRP is a commonly used biomarker of chronic inflammation that has also proven useful for predicting cardiovascular risk and mortality (Pepys and Hirschfield 2003). CRP was collected via dried blood spots and is recorded continuously in milligrams per liter (mg/L) of blood. We take the natural log of scores to adjust for extreme skewness and kurtosis. For more information on the collection and processing of CRP data in Add Health, see Whitsel et al. (2020).

Covariates

Analyses include covariates of *age* (in years) recorded across Waves III, IV, and V, as well as *sex assigned at birth* recorded at Wave I (1 = female, 0 = male). A recent study of the Add Health cohort also found that respondents who were healthier in adolescence not only tended to stay healthier as adults, but also attained higher levels of SES (Kane et al. 2018). To account for potential confounding by adolescent health status, longitudinal estimates of self-rated health during the transition to adulthood adjust for *adolescent self-rated health* at Wave I (ranging from 1 = poor to 5 = excellent).

Models that include CRP as a mechanism also adjust for a constructed scale of Wave IV *preexisting conditions* that are often associated with chronic inflammation. These include subclinical conditions such as cold or flu-like symptoms, fever, night sweats, nausea/vomiting/diarrhea, blood in stool, frequent urination, and skin rash or abscess. The scale also includes common infectious or inflammatory conditions such as asthma/chronic bronchitis/emphysema, hepatitis C, gum disease, active infection, injury, acute illness, and active seasonal allergies. Scores are top-coded at three or more conditions (see Whitsel et al. 2020).[3]

[3] Preliminary analyses included additional covariates for immigrant status, fast food consumption, gym attendance, depressive symptoms, and waist circumference (see Goosby et al. 2016). These variables were dropped because they did not alter the main findings and degraded or did not enhance the fit of our models.

Analytic Strategies

Our analyses proceed in two steps. To address whether diminished health returns are concentrated among darker-skin Black respondents, we first test for between-group differences in average within-person changes in SES and self-rated health during the transition to adulthood (Waves III to V). Using a hierarchical within-between regression estimator, with multiple observations nested within respondents, we test multilevel interactions between time-invariant (race/skin tone) and time-varying (SES) predictors of self-rated health. One major advantage of the within-between estimator is the ability to calculate coefficients for predictors that are conventionally treated as time-invariant, such as race/skin tone, and would otherwise be subtracted out of a pure fixed-effects equation. Thus, this hybrid model combines the unique strengths of random and fixed-effects estimators (see Bell and Jones 2015; Schunck 2013). The following equation summarizes our general model:

$$\begin{aligned} Y_{it} = {} & \beta_0 + \beta_{1\text{–}3}(\text{Skin tone}_i) + \beta_4(\overline{\text{SES}_i}) + \beta_5(\text{SES}_{it} - \overline{\text{SES}_i}) \\ & + \beta_{6\text{–}8}\left[\text{Skin tone}_i \times (\text{SES}_{it} - \overline{\text{SES}_i})\right] + \beta'(\text{Covariates}_{it}) \\ & + \beta''(\text{Covariates}_i) + u_i + e_{it}. \end{aligned} \quad (1)$$

This equation states that we are modeling a respondent's self-rated health at each wave as a linear function of the following eight components: (1) the sample mean of self-rated health averaged across Waves III to V (β_0); (2) the respondent's between-person skin tone classification, relative to the omitted White group ($\beta_{1\text{–}3}$); (3) the respondent's between-person SES averaged across Waves III to V (β_4); (4) the respondent's within-person deviation in SES at each wave (β_5); (5) a cross-level interaction of between-person skin tone and within-person deviations in SES at each wave ($\beta_{6\text{–}8}$); (6) a collection of time-varying (β') and time-invariant (β'') covariates; (7) a respondent-specific intercept or average self-rated health across Waves III to V (u_i); and (8) a respondent-specific residual at each wave (e_{it}).

Our theoretical focus is on the cross-level interactions between skin tone and within-person change in SES ($\beta_{6\text{–}8}$). These interactions test the degree to which average changes in SES and self-rated health vary for Black respondents of different skin tones relative to Whites. Given that Whites are the omitted group, however, the β_5 coefficient is also relevant as this reflects average within-person changes in SES and self-rated health for White respondents. The lower order skin tone coefficients ($\beta_{1\text{–}3}$) reflect average skin tone disparities in self-rated health across Waves III through V when change in SES is held constant (=0).

The between-person SES coefficient (β_4) is also important to note, as this operates as a control for unmeasured heterogeneity in the SES–health association. For example, respondents who achieve higher SES early in adulthood and maintain these levels over the study period, thereby scoring higher on between-person SES, could have come from more privileged families and developed certain dispositions or skill sets, all of which contributed to enhanced adult achievements and health. In short, the goal of this first model is to quantify the degree to which *individual changes* in SES and health during the transition to adulthood vary across skin tone groups, on average, regardless of other stable and unmeasured personal characteristics. This strategy will

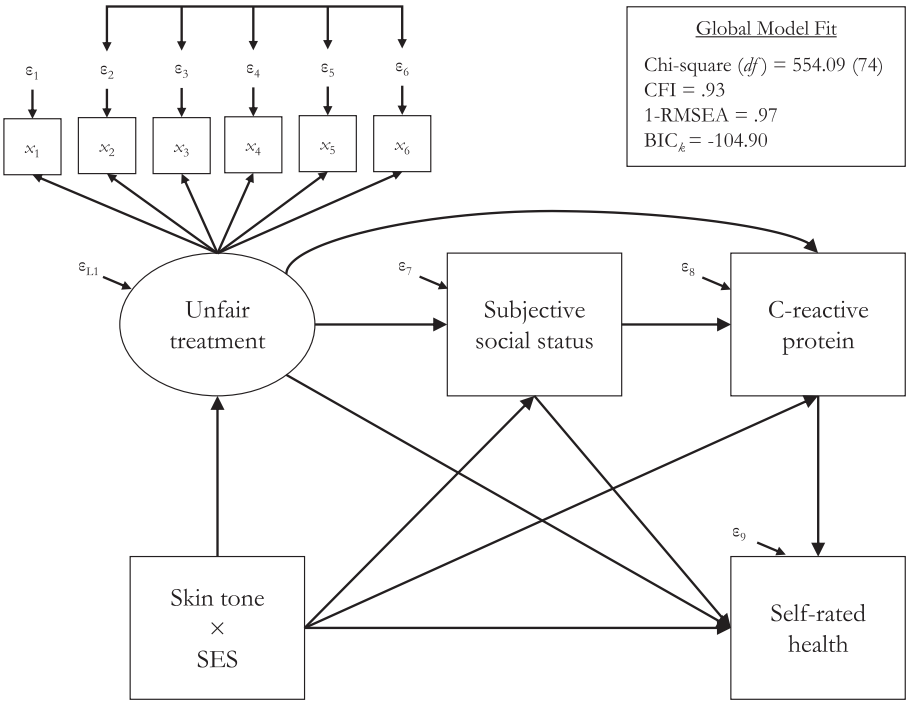

Fig. 2 Structural equation model: Add Health, Waves III–V. Skin tone, SES, and covariates have direct paths to all endogenous variables (not shown). Epsilons represent error terms. N=7,371. SES = socioeconomic status.

provide clear evidence of disparities emerging specifically during the SES attainment process.

After establishing whether we find DHRs, we then use structural equation modeling (SEM) techniques to test our stress process model. Our SEM is depicted in Figure 2. The main predictor is Wave IV SES (education and occupational prestige).[4] The moderator variable is skin tone. The main outcome is self-rated health at Wave V. Mechanisms include unfair treatment, subjective status, and CRP as described earlier. Covariates are treated as exogenous variables in the path model and are allowed to correlate with skin tone and SES, and to predict endogenous variables (not shown). Sobel (1982) statistics are calculated to identify indirect paths via the mechanisms.

The model depicted in Figure 2 exhibits acceptable fit. The 1-RMSEA (root-mean-square error of approximation) and CFI (comparative fit index) score above the minimum accepted threshold of .90 (Weston and Gore 2006). The BIC$_k$ (Bayesian information criterion) is also negative, indicating that the estimated model is superior to its fully saturated counterpart (see Eq. (21) in Raftery 1995). The error terms for the x_2 through x_6 indicators of unfair treatment are correlated to account for them being recorded together at Wave V. The standardized factor loadings for the six indicators are .50, .50, .40, .50, .35, and .33, respectively (not shown). The rho reliability

[4] Income was excluded from these analyses for reasons described later.

coefficient for the unfair treatment latent variable is .403 (not shown), which represents the squared correlation between the latent variable and the unweighted sum of its indicators (Bollen 1980:378).

Within-between models are tested in Stata 16, and our SEM is tested in Mplus 7. We use Stata's *mixed* command with maximum likelihood estimation to test our within-between models, and specify repeated observations nested within individual respondents. Missing observations are replaced with five iterations of multiple imputation by chained equations. We use full information maximum likelihood procedures in Mplus to replace missing observations and generate estimates for our SEM (Enders and Bandalos 2001). All analyses account for complex survey design, including weighting and clustering of standard errors, in accordance with the recommended procedures by Add Health staff (Chen and Chantala 2014).

Results

Descriptive Analyses

Patterns in Table 1 are generally consistent with prior research on skin tone stratification in the United States. With few exceptions, White and light-skin Black respondents report better adult health, higher adult SES, better treatment from others, and higher subjective status relative to medium- and especially dark-skin Black respondents. Intriguingly, all groups report comparable health in adolescence, and dark-skin Black respondents even report significantly fewer preexisting health conditions than their White peers by Wave IV. These patterns indicate that health disparities steadily accumulate during the transition to adulthood.

Do We Find Diminished Health Returns?

Table 2 focuses on education and income as SES indicators, since only these items were measured consistently across all waves.[5] Model 1 tests interactions with education, and Model 2 tests interactions with personal income. We include an additional age-squared term to more accurately model age-related changes in self-rated health.

First, the within-person education coefficient in Model 1 indicates that for each ordinal-unit increase in educational attainment across waves (e.g., from some college to college), self-rated health among *White respondents* is expected to improve modestly by .05 units ($b = .050$; $p < .05$). Second, the BA (Black American) dark × within-person education interaction coefficient is negative and statistically different from zero ($b = -.165$; $p < .001$). This indicates that each unit increase in education across waves predicts an average *decline* in self-rated health of .115 units among *dark-skin Black respondents* ($b = -.165 + .050 = -.115$). No significant differences in within-person associations of educational attainment and self-rated health emerge

[5] Logit models of ordinal and dichotomized self-rated health produce comparable results. Patterns are also similar for females and males in sex-stratified models.

Table 1 Descriptive statistics of study variables for White Americans and light-, medium-, and dark-skin Black Americans: Add Health, Waves III–V (N=7,371)

	White (n=5,468)		BA Light (n=268)			BA Medium (n=602)			BA Dark (n=1,033)		
Self-Rated Health (range, 1–5)											
Wave III	4.02	(0.84)	4.05	(0.85)		3.90	(0.94)	**	4.01	(0.89)	
Wave IV	3.75	(0.88)	3.68	(0.90)		3.61	(0.91)	***	3.56	(0.94)	***
Wave V	3.60	(0.96)	3.47	(0.95)	*	3.40	(0.96)	***	3.36	(0.97)	***
Education (range, 0–4)											
Wave III	1.67	(0.89)	1.67	(0.88)		1.52	(0.89)	***	1.47	(0.85)	***
Wave IV	2.31	(1.05)	2.43	(1.00)		2.18	(1.15)	**	2.02	(1.07)	***
Wave V	2.45	(1.07)	2.62	(1.08)	**	2.35	(1.16)	*	2.24	(1.11)	***
Personal Income (range, 0–10)											
Wave III	1.34	(1.53)	1.14	(1.36)	*	1.08	(1.42)	**	1.04	(1.36)	***
Wave IV	3.53	(2.26)	3.27	(2.04)		3.01	(2.22)	***	2.82	(2.13)	***
Wave V	4.70	(2.74)	4.33	(2.48)	*	3.93	(2.63)	***	3.60	(2.50)	***
Occupational Prestige (Wave IV)	100.33	(37.34)	99.55	(34.60)		95.14	(35.48)	**	89.38	(37.07)	***
Unfair Treatment (range, 0–3)[a]											
Treated with less respect or courtesy than others (x_1)	0.95	(0.80)	0.96	(0.81)		1.03	(0.85)	*	1.03	(0.89)	**
Treated with less respect or courtesy than others (x_2)	2.03	(0.83)	2.08	(0.82)		2.12	(0.84)	*	2.13	(0.88)	**
Receive poorer service at restaurants or stores (x_3)	1.55	(0.67)	1.93	(0.71)	***	1.97	(0.77)	***	2.02	(0.80)	***
People act as if they think you are not smart (x_4)	1.70	(0.78)	1.84	(0.85)	**	1.93	(0.87)	***	1.93	(0.91)	***

Table 1 (continued)

	White (n=5,468)		BA Light (n=268)			BA Medium (n=602)			BA Dark (n=1,033)		
People act as if they are afraid of you (x_5)	1.52	(0.74)	1.72	(0.81)	***	1.78	(0.87)	***	1.83	(0.91)	***
Threatened or harassed (x_6)	1.32	(0.60)	1.33	(0.57)		1.32	(0.61)		1.37	(0.66)	**
Subjective Social Status (Wave IV)	5.15	(1.69)	5.01	(1.69)		4.88	(1.77)	***	4.83	(1.73)	***
C-Reactive Protein (Wave IV)	0.70	(1.33)	0.92	(1.44)	**	0.88	(1.49)	**	0.87	(1.41)	***
Preexisting Conditions (Wave IV)	0.95	(1.11)	0.90	(.96)		0.91	(1.09)		0.79	(.97)	***
Adolescent Self-Rated Health (Wave I)	3.92	(0.88)	3.94	(0.88)		3.90	(0.94)		3.93	(0.94)	

Notes: Unweighted means are reported with standard deviations in parentheses. BA = Black American. Light/medium/dark = skin tone distinctions. C-reactive protein is reported in logged mg/L of blood.

[a] x_1 is measured at Wave IV, and x_2 through x_6 are measured at Wave V.

*p<.05; **p<.01; ***p<.001; significant difference in means relative to White respondents using one-way ANOVA (two-tailed)

Table 2 Multilevel within-between regression estimates of self-rated health: Add Health, Waves III–V

	Model 1			Model 2		
	b	SE	*p*	*b*	SE	*p*
Intercept (β_0)	3.734	(0.042)	***	3.734	(0.042)	***
Between-Person Race/Skin Tone ($\beta_{1–3}$)						
White (ref.)	—			—		
BA light	–0.122	(0.051)	*	–0.122	(0.051)	*
BA medium	–0.156	(0.041)	***	–0.156	(0.041)	***
BA dark	–0.101	(0.029)	**	–0.101	(0.029)	**
Between-Person SES (β_4)						
Education	0.158	(0.014)	***	0.158	(0.014)	***
Personal income	0.061	(0.008)	***	0.061	(0.008)	***
Within-Person SES (β_5)[a]						
Education	0.050	(0.023)	*	0.025	(0.020)	
Personal income	0.023	(0.004)	***	0.028	(0.005)	***
Cross-Level Interactions ($\beta_{6–8}$)						
BA light × within-person education	–0.094	(0.067)		—		
BA medium × within-person education	–0.021	(0.057)		—		
BA dark × within-person education	–0.165	(0.031)	***	—		
BA light × within-person income	—			–0.027	(0.026)	
BA medium × within-person income	—			–0.008	(0.010)	
BA dark × within-person income	—			–0.039	(0.010)	***
Time-Varying Covariates (β')						
Age	–0.054	(0.006)	***	–0.054	(0.006)	***
Age^2	0.001	(0.000)	***	0.001	(0.000)	***
Time-Invariant Covariates (β'')						
Female (vs. male)	–0.004	(0.021)		–0.004	(0.021)	
Adolescent self-rated health	0.247	(0.010)	***	0.247	(0.010)	***
Random Components						
Level-1 residual (e_{it})	0.693	(0.008)		0.693	(0.008)	
Level-2 intercept (u_i)	0.471	(0.011)		0.471	(0.011)	

Notes: N = 7,371. Estimates are based on the model summarized in Eq. (1). Unstandardized linear regression coefficients (*b*) are reported with robust standard errors clustered by Wave I school in parentheses. Coefficients are weighted and derived with maximum likelihood procedures. Age and age^2 are centered on the youngest age-group at Wave III (=18). Adolescent self-rated health is centered on the median (=4). BA = Black American. SES = socioeconomic status. Random components are standard deviation estimates.

[a] Represents the estimated within-person effect of SES for White respondents when included in the interactions.

*$p < .05$; **$p < .01$; ***$p < .001$ (two-tailed)

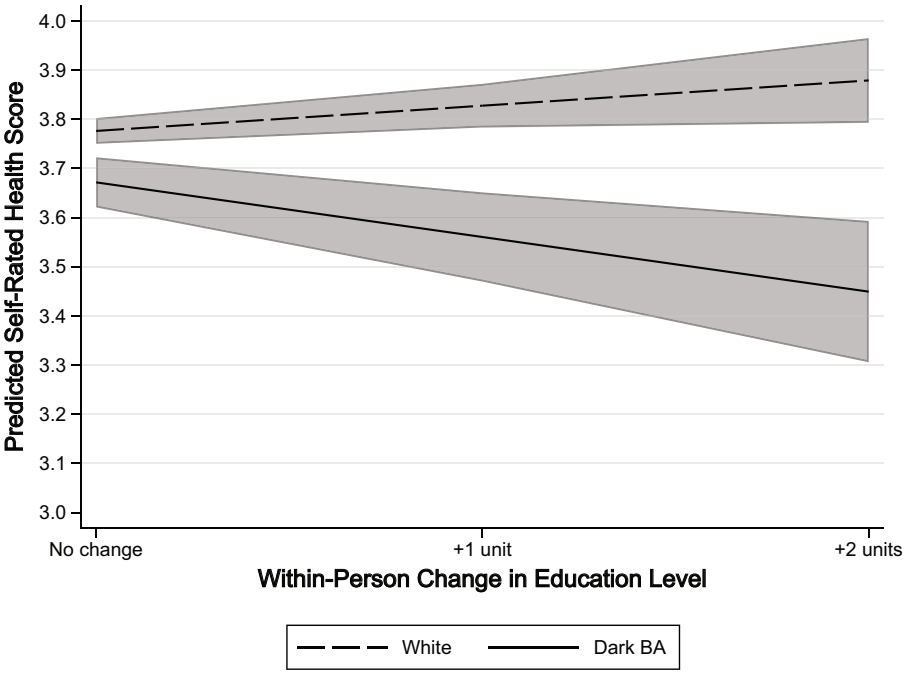

Fig. 3 Self-rated health by skin tone and within-person changes in educational attainment. Education is measured in ordinal units (0 = less than high school, 4 = postgraduate). Gray shading represents 95% confidence intervals. BA = Black American.

between White respondents, light-skin Black respondents, and medium-skin Black respondents.

Similar patterns are replicated for personal income. For each unit increase in income (e.g., from $30,000–39,999 to $40,000–49,999), self-rated health among White respondents is expected to improve modestly by .03 units ($b = .028$; $p < .001$). For dark-skin Black respondents, however, each unit increase in earnings predicts an average decline in self-rated health of .011 units ($b = -.039 + .028 = -.011$). Once again, no consistent differences emerge for light- or medium-skin Black respondents.

An example of these interactions is depicted in Figure 3, with slopes split across White and dark-skin Black groups. This figure reveals modest increases in self-rated health for higher achieving Whites and declines in health for their dark-skin Black counterparts. That is, the slope for the latter group is pulled down toward 3.0 ("good health") as their education levels increase across waves, while the slope is pulled upward toward 4.0 ("very good health") for White respondents. The interaction with personal income is visually comparable to the education interaction (not shown). Thus, we find initial evidence consistent with DHRs among higher achieving, dark-skin Black Americans relative to their White counterparts.

Does Discrimination-Related Stress Explain Diminished Health Returns?

Tables 3 and 4 report the results of our path model depicted in Figure 2. We do not find skin tone disparities in the associations between earnings and discrimination-related mechanisms after accounting for occupational status (not shown), suggesting that our within-between model with personal earnings is gauging distinctions in occupational environments.

The first column of Table 3 reveals significant skin tone × education interactions predicting unfair treatment. First, the education coefficient indicates that for each unit increase in education, reports of unfair treatment are expected to decrease by .083 units for Whites ($b = -.083$; $p < .001$). Second, the BA medium and BA dark × education interaction terms indicate that reports of unfair treatment are expected to increase modestly by .033 and .038 units, respectively, among medium- and dark-skin Black respondents for each unit increase in education. No such disparities emerge between White and light-skin Black respondents. The first column of Table 4 reveals similar patterns with occupational prestige, but only for dark-skin Black versus White respondents.

The remaining columns in Tables 3 and 4 are consistent with our conceptual model. For example, Table 3 shows that unfair treatment predicts lower subjective status ($b = -1.101$; $p < .001$). Higher subjective status then predicts lower CRP levels ($b = -.040$; $p < .01$). Finally, higher levels of CRP predict worse self-rated health at Wave V ($b = -.127$; $p < .001$). However, while findings are consistent with our stress process model, additional stress processes appear to be contributing to DHRs. For instance, unfair treatment, subjective social status, and CRP are all significant predictors of Wave V self-rated health. Even after accounting for unfair treatment, darker-skin Black groups still tend to report lower subjective status than their White peers at higher SES levels. In Table 4, dark-skin Black respondents in more prestigious occupations continue to report worse health than their White counterparts, even after accounting for the three mechanisms.

Table 5 summarizes results from our path decomposition analysis. When considering the column for White respondents, the "direct" coefficient reflects the association between the SES indicator and health, *net of* the three mechanisms. The "total indirect" row reflects the sum of all indirect paths between SES and health *via* the three mechanisms; there are a total of seven indirect paths summarized in this row. The "total" row represents the sum of all direct and indirect paths. When looking at the medium- and dark-skin Black columns, coefficients represent the *difference* in paths *relative to White respondents*. The "percentage mediated" row reflects the percentage of the total association accounted for by the mechanisms.

In the educational attainment panel, for example, the "total" coefficient in the first column indicates that each ordinal-unit increase in education at Wave IV predicts a .210-unit increase in self-rated health at Wave V among White respondents. The remaining rows show that 41% of this association is explained by the fact that more educated Whites tend to enjoy better treatment, higher subjective status, and less inflammation. Among medium- and dark-skin Black respondents, however, the respective total associations between education and health are .120 and .093 units *lower than* those among White respondents. Moreover, 65% to 100% of diminished returns among darker-skin Black respondents are explained by worse treatment, lower subjective status, and higher inflammation.

Table 3 Coefficients from a path model testing mechanisms of skin tone disparities in health returns to educational attainment: Add Health, Waves IV and V

	Unfair Treatment			Subjective Social Status			C-Reactive Protein			Self-rated Health		
	b	SE	*p*	*b*	SE	*p*	*b*	SE	*p*	*b*	SE	*p*
Education[a]	−0.083	(0.013)	***	0.310	(0.033)	***	−.0161	(0.025)	***	0.123	(0.018)	***
Race/Skin Tone												
White (ref.)	—			—			—			—		
BA light	0.084	(0.054)		−0.012	(0.122)		0.307	(0.118)	**	−0.181	(0.082)	*
BA medium	0.151	(0.057)	**	0.011	(0.114)		0.060	(0.118)		−0.060	(0.059)	
BA dark	0.135	(0.045)	**	0.190	(0.091)	*	0.136	(0.070)		−0.027	(0.049)	
Interactions												
BA light × education	0.068	(0.040)		−0.137	(0.100)		0.087	(0.120)		0.062	(0.057)	
BA medium × education	0.116	(0.031)	***	0.065	(0.091)		0.062	(0.101)		−0.042	(0.042)	
BA dark × education	0.121	(0.030)	***	−0.217	(0.092)	*	0.106	(0.062)		0.007	(0.038)	
Mechanisms												
Unfair treatment	—			−1.101	(0.111)	***	−0.028	(0.107)		−0.580	(0.093)	***
Subjective social status	—			—			−0.040	(0.015)	**	0.043	(0.011)	***
C-reactive protein	—			—			—			−0.127	(0.012)	***
Covariates												
Age	0.005	(0.005)		0.023	(0.014)		0.018	(0.012)		−0.022	(0.007)	**
Female	−0.069	(0.023)	**	0.019	(0.052)		0.535	(0.044)	***	0.134	(0.036)	***
Occupational prestige	−0.095	(0.049)		0.644	(0.139)	***	0.102	(0.126)		0.099	(0.079)	
Personal income	−0.025	(0.007)	***	0.182	(0.014)	***	−0.013	(0.012)		0.046	(0.007)	***
Preexisting conditions	0.064	(0.012)	***	−0.037	(0.024)		0.141	(0.020)	***	−0.065	(0.015)	***
Intercept	0.661	(0.158)	***	6.382	(0.402)	***	−0.028	(0.388)		4.800	(0.252)	***
R^2	.149	(.022)	***	.287	(.017)	***	.081	(.007)	***	.234	(.015)	***

Notes: $N = 7{,}371$. Coefficients are based on the model depicted in Figure 2. Unstandardized linear regression coefficients (*b*) are reported with robust standard errors clustered by Wave I school in parentheses. Coefficients are weighted and derived with full information maximum likelihood procedures. All exogenous variables are recorded at Wave IV except skin tone (Wave III) and unfair treatment (Waves IV and V). Education and income are centered on their medians. Occupational prestige is centered on its mean. BA = Black American.

[a] Represents the effect of education for White respondents.

*$p<.05$; **$p<.01$; ***$p<.001$ (two-tailed)

Table 4 Coefficients from a path model testing mechanisms of skin tone disparities in health returns to occupational attainment: Add Health, Waves IV and V

	Unfair Treatment			Subjective Social Status			C-Reactive Protein			Self-rated Health		
	b	SE	*p*	*b*	SE	*p*	*b*	SE	*p*	*b*	SE	*p*
Occupational Prestige[a]	−0.150	(0.057)	**	0.812	(0.142)	***	0.127	(0.143)		0.166	(0.079)	*
Race/Skin Tone												
White (ref.)	—			—			—			—		
BA light	0.100	(0.052)		−0.056	(0.122)		0.315	(0.106)	**	−0.171	(0.073)	*
BA medium	0.154	(0.058)	**	−0.062	(0.125)		0.051	(0.121)		−0.072	(0.060)	
BA dark	0.151	(0.047)	**	0.137	(0.085)		0.116	(0.075)		−0.070	(0.052)	
Interactions												
BA light × occupation	0.135	(0.219)		−0.434	(0.555)		−0.393	(0.552)		−0.192	(0.302)	
BA medium × occupation	0.177	(0.157)		−1.253	(0.443)	**	−.0144	(0.364)		−0.253	(0.271)	
BA dark × occupation	0.393	(0.153)	**	−0.965	(0.372)	**	−0.118	(0.319)		−0.460	(0.182)	*
Mechanisms												
Unfair treatment	—			−1.095	(.108)	***	−0.019	(0.106)		−0.573	(0.092)	***
Subjective social status	—			—			−0.042	(0.015)	**	0.041	(0.011)	***
C-reactive protein	—			—			—			−0.127	(0.012)	***
Intercept	0.674	(0.158)	***	6.402	(0.403)	***	−0.007	(0.386)		4.797	(0.252)	***
R^2	.139	(.022)	***	.288	(.017)	***	.081	(.007)	***	.235	(.015)	***

Notes: $N = 7{,}371$. Coefficients are based on the model depicted in Figure 2. Unstandardized linear regression coefficients (*b*) are reported with robust standard errors clustered by Wave I school in parentheses. Coefficients are weighted and derived with full information maximum likelihood procedures. All exogenous variables are recorded at Wave IV except skin tone (Wave III) and unfair treatment (Waves IV and V). Coefficients for covariates are excluded from this table (see Table 3). Education and income are centered on their medians. Occupational prestige is centered on its mean. BA = Black American.

[a] Represents the effect of occupation for White respondents.

*$p<.05$; **$p<.01$; ***$p<.001$ (two-tailed)

Table 5 Path decomposition analysis: Add Health, Waves IV and V

	White			BA Medium			BA Dark		
Educational Attainment									
Direct	0.123	(0.018)	***	–0.042	(.042)		0.007	(0.038)	
Total indirect	0.087	(0.010)	***	–0.078	(.024)	**	–0.100	(0.019)	***
Total	0.210	(0.017)	***	–0.120	(.035)	**	–0.093	(0.037)	*
% mediated	41			65			100		
Occupational Prestige									
Direct	0.166	(0.079)	*	—			–0.460	(0.182)	*
Total indirect	0.115	(0.042)	**	—			–0.273	(0.101)	**
Total	0.280	(0.082)	**	—			–0.734	(0.191)	***
% mediated	41			—			37		

Notes: N = 7,371. Unstandardized linear coefficients are presented with robust standard errors in parentheses. Coefficients represent the direct, indirect, and total associations between education/occupation and self-rated health as depicted in Figure 2 and reported in Tables 3 and 4. BA = Black American. Medium/dark = skin tone distinctions.

*$p<.05$; **$p<.01$; ***$p<.001$ (two-tailed)

In the occupational prestige panel, the "total" coefficient indicates that White respondents at the highest occupational prestige level (=1) self-report their health to be .280 units higher, on average, than their White peers at the lowest prestige level (=0). Recall that prestige scores are rescaled to range from 0 to 1. Moreover, 41% of this gap is explained by the mechanisms. For dark-skin Black respondents, however, those at the highest prestige level report their health to be not only .734 units lower than that of their high-status White counterparts, but also .454 units lower than that of their dark-skin Black peers at the lowest prestige level (–.734 + .280 = –.454). Nevertheless, our current model accounts for only 37% of these between-group disparities.

We should also note that the serial mediation pathway depicted in our conceptual model is significant for the medium-skin ($z = -2.32$; $p<.05$) and dark-skin ($z = -2.31$; $p<.05$) Black × education interactions in Table 3 (not shown). However, this same path is only marginally significant for the occupational prestige by dark-skin Black interaction term in Table 4 ($z = -1.92$; $p = .055$). Moreover, our proposed serial mediation pathway explains only about 1% of the total indirect associations in our path model. Most of the indirect association is accounted for solely by our measure of unfair treatment. We consider the implications of these findings in the discussion to follow.

Discussion

Our study addressed two questions. First, why do Black Americans appear to derive fewer health benefits from higher SES than their White peers? Second, are diminished health returns (DHRs) concentrated among darker-skin Black Americans? We then tested a series of hypotheses derived from these questions. According to the original *diminished returns hypothesis*, discrimination-related stressors suppress the health

benefits of higher status for Black Americans. The *status incongruence hypothesis* further stipulates that discrimination undermines the health of high-status Black Americans by creating perceptions of lowered status. The *social pain hypothesis* suggests that low subjective status, in turn, harms health by triggering chronic inflammation. Finally, the *colorism hypothesis* contends that all these stress processes will be amplified for darker-skin Black Americans.

Our first key finding is that we find patterns of DHRs primarily among darker-skin Black groups. To be specific, Black Americans rated as having dark brown or black skin tend to report slightly declining health after achieving higher levels of SES during the transition to adulthood, while their White and lighter-skin Black counterparts report marginally improved health. Our study is the first to uncover skin tone heterogeneity in DHRs among Black Americans, thereby replicating yet qualifying prior research on DHRs. Moving forward, researchers in this area should acknowledge legacies of colorism in the United States and attempt to account for skin tone disparities within Black American groups (Hargrove 2018b; Monk 2015).

The second key finding of our study is that unfair treatment, low subjective status, and inflammation explain some—but not all—of DHRs. For one, the serial mediation pathway developed in our study accounts for a small proportion of DHRs among darker-skin Black groups, with the bulk of these patterns explained solely by reports of unfair treatment. The generally worse health profiles of darker-skin Black respondents also appear to be rooted in other concrete socioeconomic inequities, particularly blocked access to higher education, income, and occupational prestige during the transition to adulthood.

There are several potential explanations for these findings. First, a growing literature on goal-striving stress and racism-related vigilance implies that *anticipatory* stress may be a stronger predictor of inflammation and health (DeAngelis 2020, 2021; Hicken et al. 2014; Monk 2015:412). Vigilance or anticipatory stress refers to a cognitive state in which a person ruminates on *potential* stressors. Studies find that Black Americans who report worrying over future racist encounters or barriers to their aspirations exhibit elevated blood pressure and neuroendocrine hormone levels, regardless of prior or ongoing discrimination (DeAngelis 2020; Hicken et al. 2014). Moreover, past exposures to unfair treatment can bring on racism-related vigilance, which, in turn, appears to mediate much of the physiological effects of past discrimination (DeAngelis 2021). Future studies should incorporate measures of anticipatory stress, which are currently unavailable in Add Health, as these measures may help to account for further variance in DHRs linked to discrimination.

There could also be additional mechanisms of DHRs that are unrelated—or, at most, indirectly related—to discrimination. Consider, for example, that higher status and darker-skin Black respondents still report lower subjective status than their White peers even after accounting for discrimination. A parallel literature on the "impostor phenomenon" suggests that Black Americans often report feeling as if they must constantly work harder than their White peers to establish a sense of validation in predominantly White institutions (Bernard and Neblett 2018). Future work is needed to assess whether impostor feelings account for further variance in DHRs.

Another plausible mechanism of DHRs is neighborhood context (Assari 2018; DeAngelis 2021). For example, Black Americans have also been shown to derive fewer "locational returns" to higher SES than Whites, meaning the same high levels

of education and income often do not buy them similar access to high-status neighborhoods (Logan and Alba 1993; Sharkey 2013). Living in involuntarily segregated Black areas, in turn, has been identified as a critical social determinant of poor health for Black Americans, shaping exposure to opportunities and environmental hazards, as well as limiting one's ability to translate individual SES resources into better health (Williams and Collins 2001). While the segregation literature suggests that neighborhood context could be an additional mechanism of DHRs, research mentioned before finds that discrimination from White neighbors can also suppress the health returns to higher neighborhood status for Black Americans. Thus, future studies must also take care to decompose countervailing mechanisms in health returns to neighborhood status among higher SES Black groups (see DeAngelis 2021).

This study may also have broader health and policy implications. Recent advances in social neuroscience demonstrate that many of the chronic conditions known to disproportionately impact Black Americans stem from similar stress processes rooted in the brain and nervous system (Barrett 2017; Goosby et al. 2018). Chronic inflammation, in particular, appears to be strongly implicated in aging-related diseases (Franceschi and Campisi 2014). Our findings indicate that inflammation tied to unfair treatment and perceived low status contributes to persistent Black–White health disparities across levels of SES, especially among dark-skin Black Americans. Although the impacts appear modest in the current context, these disparities may accumulate or widen as Add Health respondents transition into middle adulthood and old age. Indeed, it is worth reiterating that the oldest Add Health respondents were only 43 at the most recent wave of data collection. The fact that we are already beginning to uncover racialized disparities in inflammation and self-rated health could be a harbinger of more serious aging disparities down the road (e.g., Brown et al. 2016; Goosby et al. 2016).

Our findings can also speak to recent calls for implementing implicit bias training across high-status institutions in the United States (Green and Hagiwara 2020). From a "bias of crowds" (BOC) perspective, such individualized training is fundamentally flawed because it assumes that implicit bias is a stable personality trait rather than context-specific (Payne et al. 2017). Instead, the BOC perspective contends that *social contexts* should be the focus of intervention to reduce or eliminate the accessibility of anti-Black cognitive schemas. For example, one strategy could be to increase the visibility of dark-skin Black Americans within institutional leadership roles, offering counter-stereotypical examples of Black achievement and excellence (Payne and Vuletich 2018). While the current study cannot directly address the efficacy of such structural interventions, our findings at least signal a current need to reduce anti-Black stigma within high-status institutions, as doing so could conceivably mitigate Black–White disparities in stress and health.

This study is not without limitations. For one, we restricted our focus to self-rated health and CRP as health indicators. Left unknown is whether similar patterns would be observed for other indicators of health, aging, and well-being. Additionally, our measure of skin tone was limited to interviewer ratings, which may capture only one aspect of skin tone stratification. Other measures of skin tone, such as respondent self-reports or spectrophotometer readings, may provide insight into additional stress processes not identified here. More work is also needed to test additional mechanisms of DHRs mentioned earlier. It will also be important to document the

extent to which skin tone disparities emerge among other historically marginalized racial groups.

Conclusion

This study advances our understanding of persistent Black–White health disparities in the United States. Findings indicate that dark-skin Black Americans do not derive the same health benefits from higher SES as their White or light-skin Black peers, partially owing to inflammation tied to unfair treatment and perceived low status. Findings also suggest that a critical and vulnerable segment of the population will be entering middle and late life with poor health, which may further exacerbate existing inequities. Researchers, policymakers, health practitioners, and the public can all benefit from recognizing that darker-skin persons across the country are still at higher risk of exposure to unfair treatment even in high-status social settings, and that this systemic form of anti-Black stigma continues to unjustly compromise the health of Black Americans. ■

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