

Associations between darker skin tone and lower educational attainment, occupational status, residential stability, income, and wealth have long been observed even after controlling for factors like parental socioeconomic status (SES) and ethnoracial affiliation (Dixon and Telles 2017; Keith and Herring 1991; Monk 2014; Painter, Holmes, and Bateman 2016; South, Crowder, and Chavez 2005). Much of the work on social inequality patterned by skin color focuses on Latin America, because it has historically exhibited greater fluidity between racial groups compared to places like the U.S. For instance, Telles and Paschel (2014) use a unique dataset that includes self-reported racial affiliation as well as interviewers' ratings of respondent complexion to examine how skin tone predicts self-identification in Brazil, Colombia, Panama, and the Dominican Republic. They find that complexion is largely orthogonal to racial identification among Dominicans, but highly predictive among Panamanians, with Brazil and Colombia exhibiting intermediate color-race elasticity, and argue that each country's unique historical and cultural context explains this divergence.

Within the literature on skin color in Latin America, Brazil is often a focus, as it has long allowed for an intermediate 'mixed' designation between black and white in government surveys (with this gradation acting as a proxy for complexion in most work). Telles and Lim (1998) use nationally representative Brazilian survey data and find a socioeconomic hierarchy based on racial classification in the expected direction, with whites making more than browns, who in turn earn more than blacks. They also find that income inequality among subgroups is higher when using interviewer- as opposed to self-reported racial affiliation, which suggests the importance of socially ascribed categorization. Meanwhile, in their work on assortative mating in Brazil, Gullickson and Torche (2014) find a negative association between higher educational attainment and marrying spouses with darker skin color, suggesting a 'market exchange' predicated on complexion. Using an analytic approach similar to this paper, Marteleto and Dondero (2016) use Brazilian birth register

data and find that differential skin color designations predict educational disparities, even among twins. Similarly, Schwartzman (2007) finds that highly educated nonwhite parents in Brazil are more likely to categorize their children as white than their less-educated counterparts. These findings suggest that skin color, racial identification, and social status are often interconnected in Latin America.

Research in the U.S. documents similar phenomena, whereby skin color and social position are closely linked. Using late 19th and early 20th century U.S. Census data—which allowed for a 'Mulatto' designation—Saperstein and Gullickson (2013) find that occupational gains over time resulted in a greater likelihood of transitioning from the black to 'mulatto' category as judged by census takers. In their work on recent immigrants, Painter, Holmes, and Bateman (2016) find that darker skinned immigrants are less wealthy than their lighter-complected counterparts, and that this association is independent of racial affiliation (i.e. operates both *within* and *across* discrete race categories). Vargas et al. (2016), however, find that skin tone is not associated with perceived discrimination among Latinx populations in the U.S. (both native and immigrant), though, importantly, skin complexion is self-rated in their dataset.

Since the seminal work of Harburg et al. (1978), research has examined the links between complexion and biophysical outcomes like blood pressure. Analysts have found associations between darker skin tone and higher blood pressure even among high-income African Americans—consistent with research that finds weaker health returns to higher SES among blacks compared to whites (Boen 2016; Colen et al. 2017; Riddell, Harper, and Kaufman 2017; but see Do, Frank, and Finch 2012)—along with evidence that perceived discrimination positively predicts hypertension, further suggesting its potential salience as a mechanism (Sims et al. 2012). Recent work similarly finds associations between darker skin tone, higher levels of perceived discrimination, and measures such as depression and self-rated mental health among African Americans; moreover, intraracial

health differences predicted by gradations in complexion were often as large, or larger, than those found between blacks and whites more generally (Monk 2015). Other work finds that multiracial respondents who select white as the category that 'best describes' them in addition to other backgrounds report worse self-rated health profiles than those who self describe as white alone, though this result is largely driven by the tendency for Native Americans—who have measurably worse health profiles—to 'best describe' as white but still multiracial (Bratter and Gorman 2011).

While on balance the body of literature on skin tone offers suggestive evidence that color-based discrimination ('colorism') directly affects outcomes independent of racial or ethnic affiliation, the cross-sectional nature of most extant work and the possibility of endogeneity largely preclude causal inference. For example, genetic evidence confirms that African Americans who remained in the South after the Great Migration had less European ancestry—and, presumably, darker skin—than their peers who moved north (Baharian et al. 2016). If residence in the south causes hypertension through diet or another behavioral channel that cannot be realistically captured by customary data sources, darker skin tone—as rough proxy for region—may predict worse health outcomes when it is merely associated with the true cause(s). Region is, of course, but one potential confounder. While other work has utilized within-family fixed-effects methodology (Marteletto and Dondero 2016), included indicators of complexion rather than discrete racial classification (Monk 2014), and sought to examine health outcomes patterned on these distinctions (Monk 2015), we make a novel contribution to the literature on colorism by combining these features. Moreover, we present supplementary evidence that the genetic architecture of skin tone does not likely act as a source of bias (by also directly affecting the health outcome).

Methods

We use a restricted access file within the National Longitudinal Study of Adolescent to Adult Health (Add Health) that identifies genetic links between individual respondents. Add Health is a nationally representative longitudinal study of adolescents who were in grades 7-12 in the fall of 1994 to the spring of 1995 academic year. To minimize the influence of contextual factors that may be implicated in discordant outcomes in wellbeing—i.e. household, neighborhood, and school environments—we restrict our analyses to full siblings and use a family fixed effects (FE) analytic approach to examine associations between our predictors and outcomes of interest within family units. (We produce estimates based on an expanded sample that includes twins in addition to full siblings in the Appendix, Table A1). We use measures of interviewer-reported skin tone (an ordinal scale of 1-5)—only measured in wave III (2001-2002, when respondents were about 18 to 26 years of age)—to predict ever having been diagnosed with hypertension in wave IV (self-reported in 2008, when respondents were about 27-32 years old, with other indicators constructed from supplementary biometric data).

The 'constructed' hypertension indicators are derived from a combination of self-reports and blood pressure measurements collected in Wave IV, whereby readings over relevant thresholds would result in respondents being coded as positive for hypertension, even if they had indicated no formal medical diagnosis. Our main results consist of the total sample of full siblings from any background, and a black/Latinx subgroup, which we might expect to see more pronounced associations between complexion and health outcomes (though research is lacking on whether this may operate similarly in other racial or ethnic categories). In Add Health, respondents are free to check any racial affiliation that applies to them rather than a 'best overall' category, and as a result by using a black/Latinx subsample we include multiracial respondents in this grouping. We present results stratified on more rigid ethnoracial lines (i.e. white only, black only, etc.) in the Appendix (Table A4).

We find within-family discordance in skin tone and self-reported hypertension to be rather common in our data; about 21% of the overall sample and 38% of black/Latinx subsample siblings were recorded as having different skin tones (intraclass correlation coefficients [ICC]=.860 and .764, respectively). For hypertension self-reports, about 19% of sibling groups exhibited Wave IV discordance in high blood pressure diagnosis both in the full and black/Latinx subsamples. We control for sex (some research indicates that, among certain populations, females tend to have lighter skin than men [Jablonski and Chaplin 2000]), age, body mass index (BMI) collected in Wave IV. We also control for self-reported time outdoors in the summer months in Wave IV, as it relates both to variations in complexion but behaviors that may be systematically related to health outcomes, and thus a confounding influence. Because skin tone was assessed years before hypertension, it is impossible that interviewers were primed to report darker skin based on blood pressure readings or medical diagnoses. We detail the variables in the analyses in Table 1, and include standard OLS and logistic regression estimates in the supplemental Appendix (Table A6).

Results

Naive OLS and logistic regression estimates (supplemental Appendix Table A6) show associations between darker skin tone and self-reported hypertension diagnosis at the $p < .1$ level among the black/Latinx subsample ($N = 446$). We find similar results ($p < .05$; $N = 446$) using stage II hypertension as outcome, constructed from both self-reports and biometric measures taken by Add Health at Wave IV, but not for the stage I indicator. Our preferred linear FE estimates (Table 2)—which account for family background and household environment, BMI, age, sex, and time outdoors—show that darker skin color positively and significantly predicts every hypertension outcome among our black/Latinx subsample ($N = 446$). (We stratify results based on discrete racial categories in Appendix Table A4.) Our FE conditional logit results based on the same subsample,

which model hypertension as a binary outcome, offer similar estimates (though the stage II outcome is significant only at the $p < .1$ level [$p = .059$]). Using our preferred linear specifications for self-reported hypertension, estimates suggest that each unit darker complexion (e.g. going from 'medium brown' to 'dark brown') is associated with a nearly 9% increase in the probability of ever having been diagnosed, with similar effect sizes for constructed stage I/II outcomes. In addition to our main analyses which use an ordinal scale of tone as outcome, we also construct a binary measure of skin complexion based on this scale (1=black or dark brown; 0=white, light brown, or medium brown) and find similar results, except for the linear FE specification modeling constructed Hypertension I as the outcome (Appendix Table A2).

Though the sibling FE analytic approach ostensibly accounts for a common family environment, our inability to restrict our sample to monozygotic twins precludes us from wholly holding genetic background constant, though of course full siblings do share a substantial share of their genetic architecture. (We use an expanded sample that includes twins in Appendix Table A1, and find substantively similar results.) There remains a possibility that genes that predict both complexion and hypertension risk could vary between full siblings and dizygotic twins (i.e. genetic pleiotropy). In this case, our estimates would potentially be biased by not accounting for a possible genetic link between complexion and hypertension. We perform a robustness check using newly available supplementary genetic data in Add Health (McQueen et al. 2015) to determine whether several Single Nucleotide Polymorphisms (SNPs) related to skin and eye coloration predict skin tone and hypertension (see Appendix Table A7 for details on the SNPs, and Appendix Table A8 for full results).

We perform this analysis among non-Hispanic whites, where such alleles are intuitively less likely to cause colorist discrimination—though research on the effect of complexion among whites is lacking—and thus any residual association with hypertension likely works directly through

endogenous biochemical pathways. Neither PC1 (an indicator of African ancestry and darker skin), nor five of the six skin tone-related single nucleotide polymorphisms (SNPs) predict hypertension. Only the 'rs12913832' SNP significantly predicts hypertension; however, this is primarily an eye color allele (secondarily associated with complexion in prior work), does not predict skin tone in our data, and does not display variation in non-white populations, making it an unlikely candidate to explain sibling differences in hypertension within black/Latinx subgroups. Though there may be other pleiotropic alleles we have not tested, we believe the weight of evidence suggests that the effects shown in the main analysis are more likely the result of social pathways than within-family variation in genetic background.

Discussion

In our main results, skin tone significantly predicted hypertension outcomes only in the black/Latinx subsample. Our use of interviewer-coded skin color (which we are limited to due to data availability) is thus a consideration when interpreting our results. One possibility is that skin tone is rated differently in a systematic way that could bias results, such as if white interviewers were more likely to interview siblings in more affluent settings which could relate to their health profiles, while simultaneously coding them as lighter or darker than a black interviewer. Or, it could be possible that white interviewers disproportionately interviewed (and coded) darker skinned siblings, and caused a 'white coat' effect, or systematically higher blood pressure readings. Indeed, Hill (2002) finds that different-raced interviewers coded respondents in systematically different ways, with white interviewers tending to rate blacks as darker than their counterparts. When we stratified by interviewer race discordance (i.e. two siblings being interviewed by white and black field workers), however, we found that our results are concentrated in same-race interviewer dyads or triads, and thus this is not a compelling potential source of bias (Appendix Table A5). It is possible that more

straightforwardly 'objective' measures (i.e. using spectrophotometric instruments) or self-reported coloration that may reflect internalized ideas of social status would offer different results. In one of the only studies we are aware of that employ both interviewer-coded and self-reported skin tone as predictors, Monk (2015) finds that self-reported complexion measures significantly predict self-reported hypertension, while interviewer-coded measures fail to do so. Thus it is possible that our estimates are actually conservative, and that using self-reported skin tone would evince more robust associations. We also stratified on same- and discordant-sex sibling groupings (Appendix Table A3), and find our results are driven by those siblings of opposite sex. We believe this is attributable the greater variation in tone within these families; we find ICC's of .68 and .81 in mixed- and opposite-sex sibships, respectively.

We also found that an ordinal scale commonly used to proxy discrimination in Add Health (0 = never, 3 = often)—'how often do you feel you have been treated with less respect or courtesy than other people?'—did not predict hypertension in our models when added, and did not appreciably alter the results for skin tone. A follow-up question specifically asked participants what this disrespect could be attributable to, and merely 1.6% reported skin color, with an additional 8.9% citing race. When we constructed a dichotomous indicator of whether respondents indicated past discrimination based on either race or color (we could not use the latter on its own because of the rarity of the response) and included it in our models, the results were substantively similar for tone, while the discrimination indicator was significant at $p < .1$, but predicting less hypertension, not more. We feel the direction of the coefficient is likely the artifact of how few observations indicate positive responses; only 31 of 546 total black/Latinx respondents said they were disrespected because of either skin color or racial background. Because of the relatively crude nature of the measure and what it conceptually conveys (i.e. 'respect' and 'courtesy,' with discrimination only offered as a specific selection if the respondent indicated 'sometimes' or 'often' on the first

question—likely why so few did), we feel it does not discount the possibility that our results are operating through a social bias channel.

With respect to the links between discrimination and cardiovascular outcomes like hypertension, recent research suggests that racial bias is associated with leukocyte telomere length (an indicator of general systemic aging—Chae et al. 2014) and C-reactive protein levels related to inflammation and cardiovascular health (Goosby et al. 2015). The results of these studies suggest that discrimination manifests in physiological responses that in turn affect cardiovascular outcomes, and may offer insights for the results we obtain here. With the increasing availability of supplementary biometric data, future research could model these processes in sketching out the specific mechanisms embedded in the relationship between coloration and wellbeing.

Conclusion

In this paper, we find that even among full siblings and after controlling for a range of relevant factors, darker skin tone is associated with a greater likelihood of having been diagnosed with hypertension. This finding supports extant evidence of color bias, though with our data we cannot determine whether this may be a function of inter- and/or intraracial processes, or definitively rule out a more complex causal pathway that does not directly involve discrimination. We also offer additional evidence that these results are not an artifact of genetic pleiotropy between skin tone and hypertension causing alleles. Our results are the first we are aware of to bring a sibling fixed-effects analytic strategy (as well as genetic data) to bear on examining health outcomes patterned on complexion to support a social explanation for why darker-skinned people disproportionately suffer from stress-related cardiovascular health issues. These findings are yet more evidence that skin color should be considered as a relevant factor—independent of racial or ethnic affiliation alone, and

particularly in an increasingly multiracial society—in reproducing pernicious inequalities in health and wellbeing.

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TABLE 1. Variables and Descriptive Statistics (Full Sibling Sample)

	Question Text	Variable	Wave	Mean	SD	N
Hypertension	Has a doctor, nurse or other health care provider ever told you that you have or had: high blood pressure or hypertension? [If female add, when you were not pregnant] 0=No 1=Yes	H4ID5C	IV	.105	.307	1879
Hypertension, Stage I	Self-reported hypertension, with measured blood pressure over Stage I thresholds in the biometric component replacing otherwise negative indicators; 0=No 1=Yes	C4VAR045	IV	.236	.424	1879
Hypertension, Stage II	Self-reported hypertension, with measured blood pressure over Stage II thresholds in the biometric component replacing otherwise negative indicators; 0=No 1=Yes	C4VAR046	IV	.123	.329	1879
Skin Tone	What is the respondent's skin color? [Interviewer coded] 1-5 (Recoded from original variable as: 1=white; 2=light brown; 3=medium brown; 4=dark brown; 5=black)	H3IR17	III	4.350	1.126	1630
Sex	Respondent's biological sex [Interviewer coded and asked if necessary] 0=Male 1=Female	BIO_SEX4	IV	.518	.500	1879
Age	Age derived from DOB at administration of Wave IV interview	H4OD1Y	IV	28.946	1.748	1879
Outdoor	During a typical summer week, how many hours do you spend outdoors in the sun during the day?	H4DA17	IV	14.936	17.211	1830
Body Mass Index	Body mass index calculated from height and weight in the biometric component of the survey	H4BMI	IV	29.175	7.742	1847

TABLE 2. Main Results—Hypertension Predicted by Skin Tone Among Full Siblings

	Hypertension Self Report				Hypertension Constructed (Stage 1)				Hypertension Constructed (Stage 2)			
	FE		FE—Cond. Logit		FE		FE—Cond. Logit		FE		FE—Cond. Logit	
Darker Tone	.043 (.030)	.088* (.038)	.388 (.315)	.867* (.439)	.063† (.035)	.091* (.042)	.413† (.247)	.824* (.380)	.051 (.031)	.094* (.039)	.410 (.294)	.706† (.374)
Age	.010 (.006)	.020 (.013)	.153† (.081)	.158 (.146)	.016† (.009)	.044** (.017)	.118* (.058)	.383** (.121)	.012† (.007)	.025 (.014)	.164* (.074)	.218† (.127)
Female	-.031 (.026)	.012 (.052)	-.419 (.321)	.234 (.658)	-.135*** (.036)	-.138† (.071)	-.921*** (.238)	-1.329** (.505)	-.041 (.028)	.003 (.058)	-.551† (.304)	-.209 (.547)
Outdoors	<-.001 (.001)	-.001 (.002)	-.006 (.009)	-.008 (.016)	<-.001 (.001)	.001 (.003)	-.003 (.007)	.005 (.015)	<.001 (.001)	<-.001 (.003)	-.003 (.008)	<.001 (.015)
BMI	.009*** (.002)	.011* (.005)	.094*** (.023)	.067* (.032)	.012*** (.003)	.016** (.005)	.069*** (.016)	.102*** (.031)	.010*** (.002)	.011* (.005)	.094*** (.022)	.066* (.030)
Full Sample	✓		✓		✓		✓		✓		✓	
Black/Latinx		✓		✓		✓		✓		✓		✓
F	5.12***	2.35*	—	—	9.14***	6.24***	—	—	6.01***	2.52*	34.64***	13.01*
X ²	—	—	29.46***	12.71*	—	—	48.53***	29.93***	—	—	—	—
N	1496	446	268	102	1496	446	477	160	1496	446	306	119

NOTE: Robust standard errors in parentheses (OLS/FE specifications). For logit and conditional logit specifications, we give odds ratios and robust standard errors.

† $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$