Racial and Economic Adversity Differences in Stress Markers and Immune Function Among Urban Adolescents

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Background: Exposure to racism and associated adversities, such as poverty, is hypothesized to contribute to racial inequities in health via stress and immune pathways. Furthermore, the effects of adversity may be more salient during sensitive developmental periods. Our study examined racial differences in stress and immune biomarkers during adolescence and the effects of exposure to economic adversity at distinct developmental time periods and cumulatively in accounting for potential racial differences.

Methods: Secondary analysis of the Adolescent Health and Development in Context study was conducted. Data were derived from self-administered surveys; interviews; smartphone-based, geographic-explicit ecological momentary assessment; stress biomarkers (evening salivary cortisol over six nights and hair cortisol); and immune biomarkers (salivary shedding of Epstein-Barr virus [EBV] DNA among EBV-positive adolescents). Current socioeconomic status measures included annual household income and caregiver education. Caregivers also reported experiences of bankruptcy, difficulty paying bills, receipt of food stamps/Supplemental Nutrition Assistance Program/electronic benefit transfer, and job loss when the child was of ages birth–5 years, 6–10 years, and 11 years or older. An affirmative response to any item was defined as exposure to economic adversity for that developmental time period (yes/no). A cumulative economic adversity measure was calculated as the sum of exposures across developmental periods (0 = *never exposed* to 3 = *exposed across all time periods*). Descriptive and multivariable regression analyses were conducted, accounting for covariates.

Results: Black/African American adolescents had higher salivary cortisol concentration, higher hair cortisol concentration, and an increased odd of salivary shedding of EBV DNA compared to White adolescents. Racial differences were not attenuated by the current socioeconomic status or economic adversity (developmental period or cumulatively).

Discussion: Our study provides evidence that stress and immune biomarkers differ by race as early as adolescence and may be one pathway through which racism and associated adversities contribute to racial health inequities. Further research on the contribution of multiple adversities beyond poverty to racial inequities in physiological stress and health is critical for informing effective prevention and intervention efforts.

Key Words: cortisol • Epstein-Barr virus • immune function • racial differences • racism • stress

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Racial inequities in health are pervasive in the United States as Black/African Americans are more likely than their White counterparts to experience poor birth and pregnancy outcomes, infectious and chronic disease, and premature mortality (National Academies of Sciences, Engineering, and Medicine, 2017). Racism and the associated systematic exposure to adverse social conditions (e.g., poverty, residential and educational segregation, unsafe housing and living environments, violence) are hypothesized to be the root causes of racial inequities in health that often begin early in life and persist across the life span (Bailey et al., 2017; Goosby et al., 2018; Shonkoff et al., 2021; Trent et al., 2019). Life course and stress theories posit that the chronic stress associated with racism and adverse exposures negatively affects health through the damage to organs and tissues that occurs because of recurrent and prolonged activation of physiological stress response systems and subsequent immune dysregulation. Furthermore, the effects of chronic stress may be more salient if exposure to adversity occurs during sensitive developmental

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periods or life transitions, such as childhood and adolescence (Goosby et al., 2018; Shonkoff et al., 2021).

Prior research supports this line of inquiry as Black/African American youth and adults are more likely than their White counterparts to experience dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis. Specifically, researchers have found Black/African American youth and adults are more likely than White youth and adults to experience blunted salivary cortisol diurnal rhythm and elevated cortisol concentration at bedtime (Deer et al., 2018; DeSantis et al., 2007, 2015), decreased morning salivary cortisol concentration (Martin et al., 2012), increased salivary cortisol reactivity with delayed recovery in response to an experimental social stress test (Tackett et al., 2017), and, more recently, elevated hair cortisol concentration (Lehrer et al., 2020; Wosu et al., 2015). Although longitudinal studies investigating the effects of HPA dysfunction on long-term health outcomes are limited, a recent meta-analysis found robust prospective and cross-sectional associations between blunting of the salivary diurnal curve and impaired physical (e.g., immune and inflammatory diseases, cancer, body mass index/obesity, mortality) and mental health (e.g., depression; Adam et al., 2017).

Racial differences in immune function, including reactivation of latent herpes viruses—a marker of cell-mediated immune function—have also been found. Specifically, Black/African American youth and adults are more likely than their White counterparts to experience reactivation of Epstein-Barr virus (EBV; Dowd et al., 2014; Ford & Stowe, 2013, 2017) and cytomegalovirus (Dowd & Aiello, 2009), as manifested by higher levels of antiviral immunoglobulin G (IgG) antibodies or increased odds of salivary shedding of EBV DNA among those with prior latent viral infection. One study found Black–White differences among adults for reactivation of the herpes simplex virus, but not EBV (Stowe et al., 2010). Chronic or repeated latent viral reactivation is of concern as it can lead to prolonged or recurrent inflammation and, depending on the pathogen, increase host susceptibility to other infectious pathogens, cancer, or chronic disease (e.g., cardiovascular disease and autoimmune disorders; Aiello et al., 2010; Ford & Stowe, 2013; Longnecker & Neipel, 2007).

Because Black/African American children and adolescents are more likely than their White peers to experience poverty as a consequence of racism (National Academies of Sciences, Engineering, and Medicine, 2019; Trent et al., 2019), deprivation of material and social resources associated with poverty may be one explanation for the racial differences observed in physiological stress (Trent et al., 2019). In addition, researchers have found low socioeconomic status (SES) to be directly associated with irregularities in cortisol concentration and latent viral infection (Deer et al., 2018; DeSantis et al., 2015; Dowd et al., 2012). However, these findings are not ubiquitous as some studies found no direct effect of current SES on stress or immune outcomes (DeSantis et al., 2007; Dowd et al., 2014; Ford & Stowe, 2013), and none found current SES explained the racial differences in these outcomes (Deer et al., 2018; DeSantis et al., 2007, 2015; Dowd et al., 2014; Ford & Stowe, 2013). Thus, a better understanding of the potential effect of low SES experienced at distinct phases of development and cumulatively across early life development in explaining the racial differences in physiological stress and immune function is needed.

The current study builds on this prior research through the examination of the extent to which racial inequities exist across two biomarkers of cortisol activity (salivary and hair cortisol) and one marker of cell-mediated immune function (salivary shedding of EBV DNA among EBV IgG-positive adolescents) in a racially and socioeconomically representative sample of urban adolescents. In addition, we examined the extent to which economic adversity at distinct developmental time periods (infancy to early childhood, middle childhood, and preadolescence to adolescence) and cumulatively accounted for potential racial differences. Our two cortisol markers offer unique contributions as salivary cortisol measures momentary HPA axis activity at bedtime. In contrast, the hair cortisol captures cumulative exposure to cortisol over time as each 1 cm of hair growth approximates 1 month of mean cortisol output (Russell et al., 2012). To date, few studies examine multiple stress biomarkers enabling a robust investigation of potential racial differences in physiological stress.

METHODS

Study Design

A secondary analysis was conducted using data from two linked studies: the Adolescent Health and Development in Context (AHDC) study, a prospective cohort study, and the Linking Biological and Social Pathways to Adolescent Health and Well-Being study, which collected stress biomarkers among a subsample of youth participating in the AHDC study. The study design and sampling procedures have been described in previous analyses (Browning et al., 2017, 2021; Ford et al., 2019; Ford & Stowe, 2017).

Sampling and Data Collection

The studies were conducted in Columbus, Ohio, and several surrounding suburban municipalities in 2014–2016. Sampling, recruitment, and data collection were performed in collaboration with the Center for Human Resources Research—a survey research center housed within the university with experience conducting prospective cohort studies. Youth in the study area were recruited using a mix of vendor- and school-based address lists. Random households were drawn from the list and mailed a flyer that described the study with instructions to call if they were interested in participating. Trained interviewers also called to determine interest and eligibility (youth aged 11–17 years, at least one primary caregiver, and English speaking). If the household had more than one youth eligible for the study, one focal youth was randomly selected for participation in the AHDC study. The university institutional review board approved both studies, and parental consent and youth assent were obtained prior to data collection.

All data were collected in the home by trained interviewers and included (a) an entrance face-to-face interview and self-administered survey with both the focal youth and their primary caregiver; (b) a 7-day smartphone-based Global Positioning System tracking, ecological momentary assessment, and nightly salivary collection for cortisol with the youth; and (c) a face-to-face exit interview and biomarker collection with the youth (hair for cortisol and saliva for EBV antibodies and viral DNA) and a self-administered survey with the primary caregiver. The interviewers reviewed and provided written instructions to the youth on the nightly salivary collection, including the need to record the collection date and time and place the specimen in the home freezer immediately after collection. In addition, these instructions were sent to the youth nightly via the ecological momentary assessment. The interviewer collected saliva from the youth at the exit interview for EBV IgG antibody and viral DNA. The saliva collection for cortisol and EBV measures were collected via passive drool using a saliva collection aid. The youth were instructed to avoid eating, drinking, brushing their teeth 20 minutes before data collection, and to rinse their mouth with water 10 minutes beforehand. The interviewer transported all saliva specimens from the home to the survey research center. They were stored at −20°C and then transferred on dry ice to the −80°C freezers at the university laboratory until assay. The interviewer collected the hair sample at the exit interview with instructions to cut approximately 25–75 mg of hair (0.4–1 cm in diameter) from the posterior vertex region of the scalp, cutting as close to the scalp as possible with thinning shears. Thinning shears were used to maximize the amount of hair collected at once while minimizing the visibility that the hair was cut. Youth were surveyed during the visit on their hair care practices, such as frequency of washing, chemical treatments (e.g., dyes, perms, straighteners), and hair product use. Hair specimens were stored at room temperature prior to assay.

Measures

Dependent Variables

Salivary Cortisol

Before assay, the saliva samples were thawed completely and then vortexed and centrifuged at 1,500*g* for 15 minutes. The saliva was then assayed using the Salimetrics Cortisol Enzyme Immunoassay Kit (Carlsbad, CA). All samples from each participant were assayed simultaneously, on the same plate, and in duplicate. Inter- and intra-assay coefficients of variation were <10%. The My Assay analytic software program using the Salimetrics protocol was used to calculate the cortisol concentrations in μg/dl. Because of the skewed nature of the salivary cortisol measures, the values were natural log-transformed for statistical analysis.

Hair Cortisol

Hair was prepared for assay at the Ohio State University College of Nursing Stress Science Laboratory using an adapted protocol (Meyer et al., 2014) as described in prior research (Ford et al., 2019). Prior to the assay, the hair sample (1–3 cm) was washed with high-performance liquid chromatography-grade isopropanol and dried over 1–3 days. The hair was then minced in a microcentrifuge tube before being grounded into powder using a Retsch Mixer Mill MM 400 for approximately 5 minutes. A total of 1.1 ml of high-performance liquid chromatography-grade methanol was added to the ground sample and incubated for 18–24 hr at room temperature with constant agitation. The tubes were then centrifuged at 5,000*g* for 5 minutes at room temperature to pellet the powdered hair. The entire amount (~1 ml) of supernatant was transferred to a clean microcentrifuge tube, and the methanol was removed by evaporation using a stream of air for 6–8 hr at room temperature. The cortisol extract was immediately reconstituted in 100 μl of Salimetrics immunoassay cortisol analysis diluent buffer. Hair samples were assayed in duplicate using the Salimetrics high sensitivity enzyme immunoassay cortisol kit. Inter- and intra-assay coefficients of variation were <10%. The My Assay analytic software program using the Salimetrics protocol was used to calculate the cortisol concentrations in μg/dl. Hair cortisol concentration was converted to pg/mg using the formula provided by Meyer et al. (2014). Because of the skewed nature of the hair cortisol measures, the values were natural log-transformed for statistical analysis.

Salivary Shedding of EBV DNA

A primary objective of this study was to examine the potential for EBV reactivation through salivary shedding of EBV DNA; thus, the sample for this analysis included only those youth who were EBV viral capsid antigen IgG positive (had evidence of past primary EBV infection), determined through an adapted enzyme-linked immunosorbent assay method using saliva (Stowe et al., 2014). In preparative pilot work, we found a 0.92 correlation coefficient between serum and salivary EBV IgG antibodies in which an antibody titer greater than 0.02 was suggestive of prior EBV infection. Thus, youth with a salivary EBV viral capsid antigen IgG antibody level of greater than 0.02 were considered EBV positive (Ford & Stowe, 2017; Stowe et al., 2014).

Salivary shedding of EBV DNA was measured dichotomously because of the skewed nature of the distribution (36% of the youth had no evidence of salivary shedding); thus, youth who had 10 or more copies (10 copies was the lower bound detectable level) of EBV DNA in their saliva (1 = *yes*) were compared to youth who were below the detectable level. The EBV DNA viral load assessment was accomplished using polymerase chain reaction (PCR) methodology at Microgen Laboratories (Mehta et al., 2013; Stowe et al., 2007). DNA was isolated from saliva using the QiaAmp blood kit (Qiagen, Valencia, CA). EBV copy numbers were measured in samples using real-time PCR with PCR primers that amplify a portion of the BALF5 gene. Real-time fluorescence measurements were taken over 40 cycles using an Mx3005P real-time PCR instrument, and unknowns were compared to a standard curve (serially diluted plasmids containing single-copy viral genes). Copy numbers were then calculated automatically using the StrateGene software (Bellingham, WA).

Independent Variables of Interest

Race

Dichotomous measures based on self-report included non-Hispanic Black/African American and non-Hispanic White (reference) adolescents.

Current SES

Caregiver reported measures of current SES included *annual household income* (categorical measure: $0–$30,000, $30,001–$60,000, and $60,001 and greater [reference]) and *educational attainment* (less than high school, high school degree, some college, bachelor's degree, and graduate or professional degree [reference]).

Economic Adversity

Caregivers were queried on youth exposure (yes/no) to the following four economic adversities during the subsequent time periods, birth–5 years (infancy to early childhood), 6–10 years (middle childhood), and 11 years and greater (preadolescence to adolescence): experienced bankruptcy, had difficulty paying bills, received food stamps/Supplemental Nutrition Assistance Program/electronic benefit transfer, and a parent lost their job. Youth exposed to any one of the four items were defined as having been exposed to economic adversity for that developmental time period (yes/no for each period). A cumulative economic adversity measure was calculated using a sum score of exposure across the developmental periods (0 = *never exposed* to 3 = *exposed across all time periods*).

Covariates Additional measures in the analysis included the following: *sex*—youth self-reported as male or female (reference), *age* (continuous measure 11–17 years), *caregiver married* (yes vs. no), *season* of data collection (fall, winter, spring vs. summer [reference]), *household size* (caregiver-reported number of people living in the household), *weight status* (objective height and weight collected and calculated according to the Centers for Disease Control and Prevention child and adolescent body mass index guidelines with overweight and obese categories compared to the reference underweight/normal weight; Kuczmarski et al., 2002), and *pubertal development* (youth self-reported gender-specific scales adapted from Petersen et al., 1988; Ford & Stowe, 2017; Petersen et al., 1988) in which youth were asked about their perceptions of pubertal development (males and females were both asked about growth spurt in height, growth of pubic hair, and skin changes; males were asked about voice change and facial hair, and females were asked about breast growth and menstruation onset). Response options for each item ranged from 1 (*no development*) to 4 (*development complete*). To create the composite, the item scores were summed and averaged for those who had complete data on all five items; those missing a response to any item were set to missing on the scale. For the hair cortisol analysis, additional covariates included *hair length* in centimeters, *hair chemically treated* (1 = *yes*), and *daily hair washing* (1 = *yes*). For the salivary cortisol analysis, additional covariates include day-level measures of *time since waking* in hours and *waking time*.

Analytic Samples

The sample size for each outcome varied slightly due to timing of collection and methodological challenges (e.g., insufficient hair length, refusals, and outlier results). The analytic sample across all outcomes included only youth who self-identified as non-Hispanic Black/African American or non-Hispanic White and excluded youth on corticosteroids because of their effect on cortisol and immune response. The final analytic sample sizes were (a) salivary cortisol, n = 2,648 salivary cortisol samples nested within 494 adolescents (5.4 samples on average per participant, range 1–6); (b) hair cortisol, n = 453 adolescents; and (c) EBV reactivation, n = 426 adolescents who were EBV positive (79% of the eligible sample).

Analytic Strategy

Univariate analysis was conducted to describe the characteristics of the total sample for each outcome—salivary cortisol, hair cortisol, and salivary shedding of EBV DNA. Racial differences in current SES and economic adversity were described, and statistical significance was tested via chi-square and bivariate linear regression. In addition, multivariable logistic regression was conducted to examine racial differences in salivary shedding of EBV DNA, multivariable linear regression to examine racial differences in hair cortisol concentration, and multilevel linear regression analysis to examine racial differences in salivary cortisol concentration. Four models were analyzed for racial differences in each outcome: without covariates (Model 1); with current SES and covariates (Model 2); with current SES, economic adversity by developmental period (birth–5 years, 6–10 years, and 11 years and greater), and covariates (Model 3); and with current SES, cumulative economic adversity, and covariates (Model 4). In addition, we conducted post hoc sensitivity analysis to examine separate relationships for current household income and caregiver level of education and each of the three outcomes; the findings of the post hoc analysis were consistent with those presented in Model 2 for all three outcomes in which current household income and

TABLE 1. Descriptive Characteristics of the Adolescents for Each Sample by Outcome

	Salivary cortisol sample *n* = 494		Hair cortisol sample *n* = 453		Salivary EBV sample *n* = 426	
	n	%	*n*	%	*n*	%
Youth race						
Black/African American	174	35.2	158	34.9	162	38.0
White	320	64.8	295	65.1	264	62.0
Caregiver education						
Less than high school	20	4.0	15	3.3	18	4.2
High school degree	69	14.0	62	13.7	63	14.8
Some college	149	30.2	132	29.1	130	30.5
Bachelor's degree	142	28.7	135	29.8	119	27.9
Graduate/prof degree	109	22.1	109	23.2	93	21.8
Missing	5	1.0	4	0.9	3	0.7
Household income						
$0–$30,000	141	28.5	119	26.3	122	28.6
$30,001–$60,000	101	20.5	92	20.3	85	19.9
$60,001 and higher	219	44.3	210	46.3	191	44.9
Missing	33	6.7	32	7.1	28	6.6
Economic adversity: developmental period						
0–5 years						
Yes	185	37.5	164	36.2	170	39.9
No	296	59.9	279	61.6	241	56.6
Missing	13	2.6	10	2.2	15	3.5
6–10 years						
Yes	176	35.6	153	33.8	159	37.3
No	306	61.9	288	63.6	249	58.5
Missing	12	2.4	12	2.6	18	4.2
11 years and more						
Yes	159	32.2	131	28.9	133	31.2
No	324	65.6	310	68.4	276	64.8
Missing	11	2.2	12	2.6	17	4.0
Economic adversity: cumulative						
Never	220	44.5	213	47.0	172	40.4
1 time period	97	19.6	87	19.2	84	19.7
2 time periods	62	12.6	55	12.2	64	15.0
3 time periods	97	19.6	82	18.1	80	18.8
Missing	18	3.6	16	3.5	26	6.1
Youth sex						
Male	237	48.0	232	51.2	219	51.4
Female	257	52.0	221	48.8	207	48.6
Caregiver married						
Yes	303	61.3	282	62.2	259	60.8
No	188	38.1	167	36.9	159	37.3
Missing	3	0.6	4	0.9	8	1.9
Youth weight status						
Underweight	10	2.0	11	2.4	10	2.4
Normal weight	299	60.5	269	59.4	241	56.6
Overweight	74	15.0	73	16.1	79	18.5
Obese	96	19.4	90	19.9	78	18.3
Missing	15	3.0	10	2.2	18	4.2
Season of collection						
Winter	132	26.7	120	26.5	144	33.8
Spring	147	29.8	141	31.1	36	8.5

(continues)

TABLE 1. Descriptive Characteristics of the Adolescents for Each Sample by Outcome, Continued

	Salivary cortisol sample *n* = 494		Hair cortisol sample *n* = 453		Salivary EBV sample *n* = 426	
	n	%	*n*	%	*n*	%
Summer	104	21.0	98	21.6	116	27.2
Fall	111	22.5	94	20.8	130	30.5
Chemical use in hair						
Yes			76	16.8		
No			370	81.7		
Missing			7	1.5		
Washes hair daily						
Yes			178	39.3		
No			257	56.7		
Missing			18	4.0		
Salivary shedding of EBV DNA						
Yes					272	63.9
No					154	36.2
	n	Mean (*SD*)	*n*	Mean (*SD*)	*n*	Mean (*SD*)
Logged salivary cortisol[1]	2648	0.12 (0.15)	453	0.98 (1.1)	—	—
Time since waking	2540	13.5 (2.5)	—	—	—	—
Waking time	2540	7.8 (1.8)	—	—	—	—
Age (range 11–17 years)	494	14.6 (1.8)	453	14.6 (1.8)	426	14.4 (1.8)
Household size	491	4.6 (1.6)	453	4.58 (1.6)	426	4.63 (1.6)
Pubertal development (range 1–4)	386	3.05 (0.71)	356	3.04 (0.7)	326	3.02 (0.7)
Logged hair cortisol[a]			453	0.98 (1.1)		
Hair length in cm			448	2.98 (1.1)		

Note. EBV = Epstein-Barr virus.
[a]Salivary cortisol expressed as μg/dl and hair cortisol as pg/mg.

caregiver level of education are modeled simultaneously. Finally, missing responses to the independent variables were examined, and multiple imputations of independent variables using 25 imputations were conducted for all regression analyses. The puberty measure had the highest proportion of missing data (21%–23% across the three outcomes), with most because of unknown responses. Analysis was conducted using SAS 9.4 (Cary, NC) for the hair cortisol and salivary shedding of EBV DNA outcomes using Proc MI for the multiple imputation and R was used for the salivary cortisol outcomes with multiple imputation via chained equations using the Multivariate Imputation by Chained Equations (MICE) package (25 multiple imputations used for all analyses; van Buuren & Groothuis-Oudshoorn, 2011).

RESULTS

The descriptive characteristics for the total sample of adolescents for all three outcomes are presented in Table 1. The mean logged cortisol level for saliva was 0.12 μg/dl, and for hair, it was 0.98 pg/mg. Nearly 64% of the adolescents were shedding EBV DNA in the saliva, suggestive of EBV reactivation. Across the outcomes, approximately 35%–38% of the youth self-identified as Black/African American. Current SES was fairly consistent across the outcomes with 3%–5% of the primary caregivers having less than a high school degree, 13%–14% with a high school degree, 29%–30% with some college, 27%–29% with a bachelor's degree, and 21%–24% with a graduate or professional degree. Furthermore, across outcomes, the annual household income was less than $30,000 a year for 26%–28% of the adolescents, between $30,001–$60,000 for approximately 20% of the adolescents, and $60,001 and higher for 44%–46% of the adolescents. Across all the outcomes, approximately a third of the adolescents experienced economic adversity from birth to 5 years of age (36%–40%), 6–10 years (33%–37%), and 11 years and higher (28%–33%) with a slightly higher proportion prior to age of 5 years. For cumulative economic adversity, 40%–47% of the youth across outcomes never experienced economic adversity, approximately 19% experienced it at one developmental time period, 12%–15% experienced it at two time periods, and 18%–19% experienced economic adversity across all three time periods. Across the outcomes, 48%–51% of the adolescents were male, the average age was 14 years, approximately 60% of the primary caregivers were married, the average household size was about four, and the average pubertal development was 3 (range 1–4). Approximately 2% of the adolescents were underweight, 56%–60% were normal weight, 15%–18% were overweight, and 18%–19% were obese. Season of collection had some variation across outcomes because of timing of collection. Table 1 includes descriptive characteristics for additional covariates specific to the cortisol collections.

Table 2 presents the descriptive statistics for current SES and economic adversity by developmental period and cumulatively stratified by race. Across all outcomes, Black/African American adolescents were significantly more likely to experience low SES and economic adversity than White adolescents. For example (across outcomes), 7%–8% of Black/African American adolescents had a primary caregiver with less than a high school degree compared to less than 2% of White adolescents; approximately 50% of Black/African American adolescents lived in households with an annual income of less than $30,000 compared to 12%–16% of White adolescents; 48%–52% of Black/African American adolescents experienced economic adversity between birth and 5 years of age compared to 28%–32% of White adolescents, 44%–46% of Black/African American adolescents experienced economic adversity between 6 and 10 years of age compared to 27%–32% of White adolescents, and 41%–43% of Black/African American adolescents experienced economic adversity at 11 years of age and older compared to 22%–26% of White adolescents;

TABLE 2. Bivariate Results of Racial Differences in Current SES and Economic Adversity for Each Sample, By Outcome

	Salivary cortisol sample				Hair cortisol sample				Salivary EBV sample			
	Black/AA *n* = 174		White *n* = 320		Black/AA *n* = 158		White *n* = 295		Black/AA *n* = 162		White *n* = 264	
SES characteristics	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Caregiver education[a,b,c]												
Less than high school	14	8.1	6	1.9	11	7.0	4	1.4	13	8.0	5	1.9
High school degree	34	19.5	35	10.9	34	21.5	28	9.5	41	25.3	22	8.3
Some college	84	48.3	65	20.3	76	48.1	56	19.0	66	40.7	64	24.2
Bachelor's degree	30	17.2	112	35.0	25	15.8	110	37.3	29	17.9	90	34.1
Graduate/prof degree	12	6.9	97	30.3	12	7.6	93	31.5	13	8.0	80	30.3
Missing	0	0	5	1.6	0	0	4	1.3	0	0	3	1.1
Household income[a,b,c]												
$0–$30,000	90	51.7	51	15.9	82	51.9	37	12.5	82	50.6	40	15.2
$30,001–$60,000	54	31.0	47	14.7	49	31.0	43	14.6	46	28.4	39	14.8
$60,001 and higher	19	11.0	200	62.5	20	12.7	190	64.4	21	13.0	170	64.3
Missing	11	6.3	22	6.9	7	4.4	25	8.5	13	8.0	15	5.7
Economic adversity: developmental period												
0–5 years[a,b,c]												
Yes	85	48.9	100	31.3	79	50.0	85	28.8	85	52.5	85	32.2
No	78	44.8	218	68.1	70	44.3	209	70.9	65	40.1	176	66.7
Missing	11	6.3	2	0.6	9	5.7	1	0.3	12	7.4	3	1.1
6–10 years[a,b,c]												
Yes	79	45.4	97	30.3	71	44.9	82	27.8	75	46.3	84	31.8
No	83	47.7	223	69.7	76	48.1	212	71.9	70	43.2	179	67.8
Missing	12	6.9	0	0	11	7.0	1	0.3	17	10.5	1	0.4
11 years and more[a,b,c]												
Yes	75	43.1	84	26.3	65	41.1	66	22.4	67	41.4	66	25.0
No	88	50.6	236	73.7	82	51.9	228	77.3	79	48.8	197	74.6
Missing	11	6.3	0	0	11	7.0	1	0.3	16	9.8	1	0.4
Economic adversity: cumulative[a,b,c]												
Never	54	31.0	166	51.9	49	31.0	164	55.6	41	25.3	131	49.6
1 time period	28	16.1	69	21.6	26	16.5	61	20.7	26	16.0	58	22.0
2 time periods	22	12.7	40	12.5	20	12.7	35	11.9	22	13.6	42	15.9
3 time periods	54	31.0	43	13.4	48	30.4	34	11.5	50	30.9	30	11.4
Missing	16	9.2	2	0.6	15	9.5	1	0.3	23	14.2	3	1.1

Note. SES = socioeconomic status; EBV = Epstein-Barr virus; AA = African American.
[a] $p < .001$ for racial differences in current SES and economic adversity for salivary cortisol sample.
[b] $p < .001$ for racial differences in current SES and economic adversity for hair cortisol sample.
[c] $p < .001$ for racial differences in current SES and economic adversity for salivary EBV sample.

and approximately 30% of Black/African American adolescents experienced economic adversity across all three developmental periods compared to 11%–13% of White adolescents.

Table 3 presents the results for the multilevel linear regression analysis of racial differences in evening salivary cortisol concentration. Across Models 1–4, Black/African American adolescents had higher salivary cortisol concentration in comparison to White adolescents and the size of the effect was not attenuated by the SES measures: in the baseline model (Model 1), controlling only for daily measures of time since waking and wake-up time ($b = 0.18$, $SE = 0.01$, $p = .002$); in Model 2, accounting for current SES and covariates ($b = 0.25$, $SE = 0.08$, $p = .001$); in Model 3, accounting for current SES, developmental period economic adversity, and covariates ($b = 0.25$, $SE = 0.08$, $p = .001$); and in Model 4, accounting for current SES, cumulative economic adversity, and covariates ($b = 0.25$, $SE = 0.08$, $p = .001$). Adolescents who had a primary caregiver with less than a high school degree had lower salivary cortisol concentration than those with a graduate or professional degree (Models 2–4). Results for the covariates are available upon request.

Table 4 presents the results for the linear regression analysis of racial differences in hair cortisol concentration. Across all Models 1–4, Black/African American adolescents had higher hair cortisol concentration in comparison to White adolescents, and the size of the effect was not attenuated by the SES measures: in the baseline model, without covariates ($b = 0.48$, $SE = 0.02$, $p < .001$); in Model 2, accounting for current SES and covariates ($b = 0.41$, $SE = 0.14$, $p = .003$); in Model 3, accounting for current SES, economic adversity by developmental period, and covariates ($b = 0.42$, $SE = 0.14$, $p = .003$); and in Model 4, accounting for current SES, cumulative economic adversity, and covariates ($b = 0.41$, $SE = 0.14$, $p = .001$). Marginally significant direct associations were found for SES and hair cortisol concentration in which adolescents who had a caregiver with a high school education or more had marginally higher hair cortisol concentration than those with a graduate or professional degree (Models 2–4, $p = .06$). In comparison, adolescents who experienced economic adversity between birth and 5 years of age had marginally lower hair cortisol concentration than those who did not experience this adversity (Model 3, $p = .06$). Results for the covariates are available upon request.

Table 5 presents the results for the logistic regression analysis of racial differences in salivary shedding of EBV DNA. Across all Models 1–4, Black/African American adolescents had an increased odds of salivary shedding of EBV DNA compared to White adolescents, and the size of the effect was not attenuated

TABLE 3. Multilevel Linear Regression Results of the Racial Differences in Logged Salivary Cortisol Among Adolescents, *N* = 2,248 Salivary Samples Nested Within 464 Adolescents

	Model 1	Model 2	Model 3	Model 4
	***b* (*SE*)**	***b* (*SE*)**	***b SE*)**	***b* (*SE*)**
Youth race				
Black/African American	0.18 (0.006)**	0.25 (0.08)**	0.25 (0.08)**	0.25 (0.08)**
White (ref)				
Caregiver education				
Less than high school		–0.38 (0.16)*	–0.36 (0.16)*	–0.37 (0.16)*
High school degree		–0.01 (0.09)	0.00 (0.09)	–0.01 (0.10)
Some college		–0.13 (0.09)	–0.12 (0.09)	–0.013 (0.09)
Bachelor's degree		–0.05 (0.07)	–0.05 (0.07)	–0.04 (0.07)
Graduate/professional degree (ref)				
Household income				
\$0–\$30,000		0.06 (0.09)	0.06 (0.10)	0.07 (0.10)
\$30,001–\$60,000		0.04 (0.08)	0.04 (0.08)	0.05 (0.08)
\$60,001+ (ref)				
Economic adversity: developmental period				
0–5 years			–0.004 (0.07)	
6–10 years			–0.08 (0.08)	
11 years and more			0.05 (0.07)	
Economic adversity: cumulative				–0.01 (0.03)
Constant	–2.53 (0.03)***	–3.10 (0.26)***	–3.05 (0.26)***	–3.09 (0.26)***
Ins1_1_1 constant	0.29 (0.03)***	0.27 (0.03)***	0.27 (0.02)***	0.27 (0.02)***
Insig_e constant	0.34 (0.02)***	0.34 (0.02)***	0.34 (0.02)***	0.34 (0.02)***

Note. Controlling for sex, age, caregiver marital status, season of collection, weight status, pubertal development, household size, time since waking, and waking time.

*$p < .05$.

**$p < .01$.

***$p < .001$.

TABLE 4. Linear Regression Results of the Racial Differences in Hair Cortisol Among Adolescents, *N* = 453

	Model 1		Model 2		Model 3		Model 4	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Youth race								
Black/African American	0.48	(0.02)***	0.41	(0.14)**	0.42	(0.14)**	0.41	(0.14)**
White (ref)								
Caregiver education								
<High school			0.21	(0.33)	0.21	(0.33)	0.18	(0.33)
High school degree			0.38	(0.19)~	0.36	(0.20)~	0.37	(0.20)~
Some college			0.15	(0.17)	0.13	(0.17)	0.14	(0.17)
Bachelor's degree			0.21	(0.14)	0.22	(0.14)	0.21	(0.14)
Grad/prof degree (ref)								
Household income								
\$0–\$30,000			–0.10	(0.17)	–0.10	(0.18)	–0.07	(0.18)
\$30,001–\$60,000			–0.02	(0.16)	–0.02	(0.16)	–0.01	(0.16)
\$60,001+ (ref)								
Economic adversity: developmental period								
0–5 years					–0.25	(0.13)~		
6–10 years					0.22	(0.15)		
11 years and more					0.02	(0.14)		
Economic adversity: cumulative							–0.002	(0.05)
Constant	0.81	(0.01)***	0.80	(0.48)	0.76	(0.49)	0.80	(0.49)

Note. Controlling for sex, age, caregiver marital status, season of collection, weight status, pubertal development, household size, hair length, frequency of hair washing, and chemical use in hair.
*$p < .05$.
**$p < .01$.
***$p < .001$.
~$p < .10$.

by the SES measures: in the baseline model, without covariates (*OR* = 1.91, 95% CI [1.04, 3.53], *p* = .038); in Model 2, accounting for current SES and covariates (*OR* = 1.94, 95% CI [1.05, 3.57], *p* = .034); in Model 3, accounting for current SES, developmental period economic adversity, and covariates (*OR* = 1.91, 95% CI [1.03, 3.54], *p* = .039); and in Model 4, accounting for current SES, cumulative economic adversity, and covariates (*OR* = 1.91, 95% CI [1.04, 3.52], *p* = .038). Significant direct associations between SES and salivary shedding of EBV DNA were found as adolescents who had a caregiver with some college had lower odds of salivary shedding of EBV DNA than those with a graduate or professional degree (Models 2–4), whereas adolescents who experienced economic adversity between birth and 5 years of age had higher odds of salivary shedding of EBV DNA than those who did not experience this adversity (Model 3). Results for the covariates are available upon request.

DISCUSSION

Significant racial differences in cortisol and immune biomarkers were found in our study. Black/African American youth had higher salivary and hair cortisol concentration and increased odds of salivary shedding of EBV DNA compared to their White peers. Our findings are consistent with prior research documenting racial inequities in physiological stress and immune function (DeSantis et al., 2007, 2015; Dowd et al., 2014; Ford & Stowe, 2013; Lehrer et al., 2020; Tackett et al., 2017; Wosu et al., 2015) and provide novel contributions of robust Black–White differences across multiple stress biomarkers among the adolescents in this study. Notably, extant research documents how dysregulation of the HPA axis and the immune response are associated with numerous poor health outcomes (Adam et al., 2017; Longnecker & Neipel, 2007). Thus, our study adds to the evidence that elevated stress and immune biomarkers differ by race as early as adolescence and may be one pathway through which racism and associated adversities contribute to the Black–White health inequities observed in the United States.

In addition, we found economic adversity experienced between birth and 5 years of age was significantly associated with salivary shedding of EBV DNA and marginally associated with lower hair cortisol; these findings are consistent with prior research (Bunea et al., 2017; Ehlert, 2013; Finegood et al., 2017; Kalmakis et al., 2015; Khoury et al., 2019; Simmons et al., 2016). It is important to note that both high and low cortisol concentrations have been found to be associated with activation of the immune response and poor health (Steudte-Schmiedgen et al., 2016). Low cortisol concentration is thought to be the result of glucocorticoid resistance or abnormalities in the HPA negative feedback loop because of chronic stress, which may explain in part the lower salivary cortisol concentration found among

TABLE 5. Logistic Regression Results of the Racial Differences in Salivary Shedding of EBV DNA Among Adolescents, $N = 426$

	Model 1		Model 2		Model 3		Model 4	
	OR	95% CI	*OR*	95% CI	*OR*	95% CI	*OR*	95% CI
Youth race								
Black/African American	1.91	(1.04, 3.53)	1.94	(1.05, 3.57)	1.91	(1.03, 3.54)	1.91	(1.04, 3.52)
White (ref)								
Caregiver education								
Less than high school			1.98	(0.21, 18.40)	1.80	(0.19, 17.0)	1.74	(0.20, 15.1)
High school degree			0.52	(0.20, 1.30)	0.48	(0.19,1.24)	0.50	(0.20, 1.26)
Some college			0.33	(0.15, 0.73)	0.32	(0.15,0.72)	0.32	(0.14, 0.71)
Bachelor's degree			0.56	(0.29, 1.08)	0.54	(0.28, 1.04)	0.56	(0.29, 1.07)
Graduate/prof degree (ref)								
Household income								
\$0–\$30,000			1.56	(0.69, 3.51)	1.59	(0.67, 3.78)	1.53	(0.66, 3.56)
\$30,001–\$60,000			0.73	(0.35,1.52)	0.73	(0.34,1.57)	0.71	(0.33, 1.51)
\$60,001+ (ref)								
Economic adversity: developmental period								
0–5 years					1.91	(1.04, 3.50)		
6–10 years					0.72	(0.35, 1.48)		
11 years and more					0.89	(0.45, 1.77)		
Economic adversity: cumulative							1.10	(0.87, 1.39)
Constant	0.83	(0.09, 7.69)	0.89	(0.10, 8.16)	0.76	(0.08, 7.39)	0.83	(0.09, 7.69)

Note. Controlling for sex, age, caregiver marital status, season of collection, weight status, pubertal development, and household size. EBV = Epstein-Barr virus; *OR* = odds ratio; CI = confidence interval.

adolescents who had a caregiver with less than a high school education (vs. graduate/professional degree) and the lower hair cortisol concentration (marginally significant) found among adolescents exposed to economic adversity earlier in life. Although racism is also a chronic stressor, Black/African Americans experience the deleterious effects of racism daily, which may account for the racial differences in higher cortisol concentrations observed in our study and extant research. Future research of the dynamics of physiological stress over time is greatly needed to understand better the effects of social adversity on racial inequities in stress and health across the life span.

Consistent with prior research (DeSantis et al., 2007; Dowd et al., 2014; Ford & Stowe, 2013), neither current SES nor life course economic adversity (developmental period or cumulative exposure) attenuated the racial differences in salivary and hair cortisol or salivary shedding of EBV DNA among the adolescents in our study despite the vast racial differences they experience in economic adversity. Explanations for the inequities are most likely multifactorial as Black/African American youth also have increased exposure to multiple types of other adversities compared to White youth. For example, in addition to poverty, Black/African American youth are more likely than White youth to be exposed to violence in their residential neighborhood (Census tract) and routine activity locations (activity space; Browning et al., 2017); live in areas with fewer social, educational, economic, and health-related resources (using the Child Opportunity Index; Acevedo-Garcia et al., 2020); experience parental death, parental incarceration, parental divorce/separation, and racial discrimination; and witness domestic violence as well as witness or be a victim of neighborhood violence (Maguire-Jack et al., 2020). Moreover, they also are more likely to experience these adverse exposures at much earlier ages (Maguire-Jack et al., 2020), which may contribute to a more significant cumulative burden over time, as well as the potential for more harmful effects because of exposure during a sensitive developmental period (Shonkoff et al., 2021). These increased exposures stem from centuries of structural racism and discriminatory practices historically embedded in our housing, education, health, and correctional systems with significant negative and inequitable health and social consequences for communities, families, and people of color (Bailey et al., 2017; Goosby et al., 2018; Shonkoff et al., 2021; Trent et al., 2019). Research investigating the contribution of these multiple adversities at distinct developmental time periods and across the life span to racial inequities in physiological stress and health is critical for informing effective prevention and intervention efforts.

Several study limitations warrant further discussion. First, our study is cross-sectional in design, and we measure physiological stress and immune function at one time point during adolescence, precluding causal inference. Second, our study focused on the direct relationships between race and physiological stress and immune function. However, there may be heterogeneity in this relationship by other sociodemographic factors (e.g., age, gender, sexual orientation, gender identity); thus, further research on the effects of these intersections on physiological stress and immune function is needed. Last, we collected salivary cortisol only at bedtime because of methodological challenges of collecting saliva samples at multiple time points daily over the weeklong data collection period (e.g., school attendance, storage, cost). Although the single time point of collection precluded our examination of the diurnal curve, prior studies found blunting of the diurnal curve with higher nighttime cortisol levels among low-income and Black/African American samples (Cohen et al., 2006).

Conclusion

Our study provides novel contributions to examining racial inequities in physiological stress and immune function in adolescence. Future research is needed to identify individual, social, and structural factors that may prevent and/or buffer stress to develop effective multilevel interventions. To date, interventions have focused primarily on individual resilience. Although they may be effective for coping with stress at the individual level, we must address the underlying factors contributing to racial inequities in stress and health—structural racism and its associated adversities—to improve the health and well-being of Black/African Americans and all people of color. Evidence from a review of interventions targeting adverse childhood experiences found professionally led, multicomponent interventions that included parenting education, mental health counseling, social service referral, and/or social support improved child behavioral/mental health well-being and the parent–child relationship (Marie-Mitchell & Kostolansky, 2019). However, the interventions were targeted primarily to families with children 5 years of age or less. Though this is a period of development where intervention is critical to improving lifelong health and well-being, interventions targeting older children, adolescents, and adults across the life course are also needed.

Furthermore, structural interventions and financial investment at the local, state, and national levels targeting racism and the associated adversities are critical for *preventing* racial inequities in stress and health across the life span, ultimately improving all Americans' health and well-being.

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