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Does Socioeconomic Status Mediate Racial Differences in the Cortisol Response in Middle Childhood?

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Objective: Race/ethnicity and socioeconomic status are both associated with stress physiology as indexed by cortisol. The present study tested the extent to which racial/ethnic disparities in cortisol reactivity are explained by socioeconomic status. ***Method:*** The sample consisted of 296 racially and socioeconomically diverse children ages 8–11 (47% boys). Mothers reported on children's stressors and socioeconomic status; salivary cortisol levels were assessed before and after the Trier Social Stress Test. ***Results:*** Results demonstrated that racial group differences in cortisol reactivity were partially accounted for by differences in socioeconomic status, but racial group differences in cortisol recovery were not. ***Conclusions:*** These findings suggest that cumulative effects of stress and disadvantage may result in differences in stress response physiology as early as middle childhood, and that race-specific mechanisms account for additional variance in cortisol reactivity and recovery.

Keywords: cortisol reactivity and recovery, racial/ethnic differences, stress, socioeconomic status, middle childhood

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A growing line of research indicates that members of racial/ethnic minority groups evidence differences in stress physiology, as often measured via the hormone cortisol (Adam et al., 2015; Bush, Obradović, Adler, & Boyce, 2011; DeSantis et al., 2007). Differences in cortisol function have also been associated with socioeconomic status (SES) and related contextual, or environmental, adversity (Gunnar & Vazquez, 2001; Karb, Elliott, Dowd, & Morenoff, 2012; Rudolph et al., 2014). Explanations of differentiated cortisol functioning in racial and ethnic minority groups have invoked the potential impact and deterioration of stress system functioning that likely occurs for individuals facing higher levels of adversity, suggesting that differences in cortisol functioning reflect adversity "getting under the skin" (Bush et al., 2011; Karb et al., 2012; Skinner, Shirtcliff, Haggerty, Coe, & Catalano, 2011). Adversity and stress in this context includes lower SES and broader contextual inequality and deprivation, as well as other stressors disproportionately affecting members of racial and ethnic minority groups, such as perceived discrimination (Adam et al., 2015; Levy, Heissel, Richeson, & Adam, 2016). Despite the intertwined nature of the explanations for these disparities in cortisol functioning across both racial/ethnic group membership and levels of SES, previous investigations have not focused specifically on disentangling them. In addition, the majority of this work has examined aspects of diurnal or basal cortisol functioning, limiting our understanding of how such explanations manifest in regards to acute cortisol reactivity in response to stress. Examining these questions is even more pressing early in life (e.g., childhood) to isolate the developmental trajectory of potential physiological effects of racial/ethnic and socioeconomic stress. Thus, the primary goal of the present study was to tackle this question: how much are racial/ethnic differences in cortisol stress reactivity, as assessed in the lab with the Trier Social Stress Test for children, accounted for by differences in SES, even as early as middle childhood?

Cortisol Functioning and the Stress Response

Cortisol, the primary product of the hypothalamic-pituitary-adrenal (HPA) axis, is one of the key hormones mediating the biological response to stress (Kirschbaum & Hellhammer, 2000). Cortisol levels increase in response to acute stress, particularly stressors that include either anticipated or actual negative social evaluations (Dickerson & Kemeny, 2004)—this acute stress response represents the aspect of cortisol functioning addressed in the current study. Cortisol reactivity to stress is examined by observing increases in cortisol above basal/diurnal levels, either naturally occurring or experimentally induced, and is often examined in the afternoon hours, when basal levels are less rapidly changing. Acute increases in salivary cortisol are thought to help mobilize the individual for coping with the stressor at hand, providing an energetic boost, narrowing cognition to focus on the stressor at hand, and acutely mobilizing immune resources (Sapolsky, Romero, & Munck, 2000). Recent evidence has suggested that acute cortisol increases also help to boost positive mood state (Hoyt, Zeiders, Ehrlich, & Adam, 2016). With the termination of a stressor, high levels of glucocorticoids provide negative feedback information to the brain, signaling termination of the stress re-

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sponse; a rapid recovery of cortisol levels following acute stress is thought to indicate an efficient, well-functioning negative feedback mechanism (Blair, Granger, & Peters Razza, 2005). A robust increase in cortisol in response to stress, followed by a rapid recovery or decline in cortisol levels poststressor is optimal; insufficient stress responding, and sluggish recovery from stress have been linked to negative cognitive (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007) and health outcomes (Kirschbaum et al., 1995; Phillips, Ginty, & Hughes, 2013). Very small increases and delayed recovery of cortisol levels after exposure to a stressor are both thought to be indications of hypocortisolism, a form of stress-system dysregulation (Adam, Klimes-Dougan, & Gunnar, 2007; Burke, Davis, Otte, & Mohr, 2005; Burke, Fernald, Gertler, & Adler, 2005; Heim, Ehlert, & Hellhammer, 2000; van Goozen et al., 1998).

In addition to increasing in response to stress, cortisol levels show a strong diurnal rhythm, with levels being high on waking, increasing in the 30–40 min postawakening (called the cortisol awakening response), declining quickly in the first few hours after waking, and then more slowly to reach low levels around bedtime (Kirschbaum & Hellhammer, 2000). Similar to the functions of cortisol increases in acute cortisol reactivity, high levels of waking cortisol and a moderately large cortisol awakening response help to positively mobilize the individual for the demands of the day, contributing to boosts in cognition, energy and appetite (Adam, Hawkley, Kudielka, & Cacioppo, 2006); and a robust decline in cortisol levels across the day to low levels in the evening helps to prepare the individual for lower levels of activity and sleep during the evening hours. Low levels of cortisol across the day, particularly during the morning hours when levels are supposed to be high, are also considered indicators of hypocortisolism (Gunnar & Vazquez, 2001), which have been attributed to exposure to extreme adversity or to chronic stress (Adam, 2012).

Disentangling Racial/Ethnic and Socioeconomic Differences in Cortisol Functioning

Previous research has identified rather robust evidence for racial/ethnic differences in indices of cortisol functioning from childhood to adulthood, although almost all of this research has focused on basal cortisol functioning rather than reactivity. Most research indicates flatter diurnal cortisol slopes from waking to bedtime across the waking day in racial/ethnic minority adolescents (DeSantis et al., 2007; Martin, Bruce, & Fisher, 2012) and adults (DeSantis, Adam, Hawkley, Kudielka, & Cacioppo, 2015; Skinner et al., 2011). Two studies have provided initial evidence of blunted cortisol reactivity to acute stress among minority racial/ethnic groups (Hostinar, McQuillan, Mirous, Grant, & Adam, 2014), particularly among racial/ethnic minorities exposed to recent race-based stress (Richman & Jonassaint, 2008). Most of the research on basal cortisol has found that associations between race/ethnicity and flatter diurnal cortisol slopes occurs above and beyond the effects of socioeconomic status, and may be best predicted by specific exposure to race-based social stress such as perceived discrimination (Adam et al., 2015). Past research on racial/ethnic differences in cortisol reactivity have, however, not paid sufficient attention to the possible confounding role of socioeconomic status.

Previous research has demonstrated evidence for associations between low socioeconomic status and cortisol functioning. Although these findings have been somewhat mixed, they tend to point in the same direction—lower socioeconomic status reflecting lower basal cortisol levels (Chen & Paterson, 2006), flatter diurnal cortisol slopes (DeSantis, Kuzawa, & Adam, 2015; Hajat et al., 2010), or blunted reactivity (Gunnar & Vazquez, 2001). Research examining the effects of SES and broader contextual stressors on cortisol has found evidence for an impact of proximal effects (e.g., income and education level; Cohen, Doyle, & Baum, 2006; Ranjit, Young, & Kaplan, 2005), as well as distal effects (e.g., neighborhood stress, neighborhood-level social support, and neighborhood violence; Do et al., 2011; Karb et al., 2012). Thus, evidence suggests that socioeconomic status and contextual stress may "get under the skin" across multiple contextual levels, although such work to date has not focused specifically on the acute cortisol stress response, as we do in the present study. It is important to obtain better identification of those socioenvironmental variables most relevant for altered cortisol reactivity to assess specificity of different types of environmental and contextual stressors, with implications for prevention and intervention.

The Present Study

The present study had the primary goal of directly testing the extent to which SES and broader contextual stress, specifically, neighborhood affluence, would account for racial/ethnic differences in the cortisol stress response (focusing here on acute reactivity and recovery) in a moderately large sample of preadolescents. We examined the cortisol stress response using two indices: cortisol reactivity (increase in cortisol levels following an acute stressor) and cortisol recovery (decrease in cortisol levels from the peak, following removal of the stressor). We also examined sociocontextual stress using different indices: an index of SES reflecting both mother's highest level of education and overall family income (referred to in this paper as summed education/income), which is a commonly used approach to indexing SES in the literature (Cohen, Schwartz, et al., 2006; Cohen, Doyle, et al., 2006; Hajat et al., 2010) and an index of SES reflecting overall neighborhood affluence (or, conversely, deprivation) coded from census data, which is another measure that has been used in previous research (Krieger, 1992; McLaughlin, Costello, Leblanc, Sampson, & Kessler, 2012). These measures of SES were selected as they have been used regularly in the broader literature and, thus, facilitate comparisons of findings across studies, but also because they reflect differential factors to the extent that they operate closer to the child, or in the home (maternal education/income) versus further from the child, or outside the home (neighborhood affluence). As another comparison point, we examined a measure of cumulative life stressors as a potentially less contextual stressor that would not be expected to serve as a proxy for broader contextual adversity and disadvantage, as do measures of SES. Specifically, we examined the following hypotheses:

> *Hypothesis 1:* We predicted that low socioeconomic status (low summed education/income and neighborhood affluence) would predict lower cortisol reactivity and recovery, above and beyond prediction by race/ethnic group membership, in hierarchical regression models.

Hypothesis 2: We predicted that, in a series of mediation models, low summed education/income and neighborhood affluence would partially mediate the effects of minority race/ethnic group membership on lowered cortisol reactivity and recovery.

Hypothesis 3: We predicted that life stressors, as an index of stress further removed from the more systemic and entrenched stress associated with broad socioeconomic status and context, would not show a similar pattern of incremental prediction and mediation.

We examined these hypotheses in a sample of 296 highly diverse preadolescents—diverse in terms of both race/ethnicity and socioeconomic status. Diversity in both race/ethnicity and SES domains, in a relatively large sample, offers a critical opportunity to disentangle these associations. In addition, support for these hypotheses in a middle childhood sample would contribute to identifying a potential timeline for such effects, suggesting that they are embedded in physiological functioning even prior to adolescence.

Method

Participants

Of an initial sample of 350 participants, all Black, White, and Hispanic participants were selected for the present study (excluding smaller categories reflecting Asian, Multiracial, and Other groups), resulting in a sample of 296 children and their primary female caregivers (98% mothers). Children were primarily ages 9–10 years (M = 9.82, SD = 0.66; 140 males [47%], full age range 8–11). Participants were recruited from an urban community in the Southwestern United States, using directory information from local school districts and flyers posted in the community and distributed at events. School directory information was employed via a rolling recruitment procedure, such that batches of letters were sent to potential families and followed up via phone to determine interest and eligibility. Recruitment continued until the target sample of 350 families was reached; thus, the aim was not to obtain a perfectly representative sample. Inclusion criteria for the study were fluency in English for the child and fluency in English or Spanish for the female caregiver and exclusion criteria were psychotic disorders, mental retardation, and neurodevelopmental disorders in the child.

Caregivers reported the following demographics on a questionnaire: race/ethnicities for their children, marital status, education, employment, and annual household income. The sample of 296 children were reported as 35.1% Black/African American, 29.4% Hispanic/Latin American, and 35.5% White/European American. Additional demographic information can be found in Table 1.

Measures

Life Events Questionnaire (LEQ). Primary female caregivers were asked to complete the 50-item LEQ (adapted from the Life Events Interviews; Billig, Hershberger, Iacono, & McGue, 1996) to assess the presence or absence of specific life events at any point during the child's life. For this study, a family level events score (FAM; Kushner & Tackett, 2015) was calculated using life event items related to family financial problems (e.g., parent lost job), family legal problems (e.g., family member arrested), family mental health problems (e.g., family member treated for emotional problems), and parental discord/divorce/changes that happened at any point during the child's life. The scores on the FAM Scale ranged from 0 to 9 in this sample. In the current study, the FAM score had a coefficient alpha of .79.

Socioeconomic Status (SES). Two scores were created to capture SES in this sample. The first score was based on caregiver

Table 1
Descriptive Characteristics of the Sample on Demographic Measures

	Black (n = 104)				Hispanic (n = 87)				White (n = 105)			
	M	*SD*	*n*	%	*M*	*SD*	*n*	%	*M*	*SD*	*n*	%
Sex (female)			53	51.0%			44	50.6%			59	56.2%
Age	9.31	.68			9.29	.68			9.27	.67		

Family Income	Under $20,000	$20,001 – 40,000	$40,001 – 60,000	$60,001 – 80,000	$80,001 – 100,000	Over $100,000	Did not report
	20.9%	16.9%	9.5%	9.5%	8.8%	28.4%	6.1%

Marital Status	Married or living with a partner	Never married	Divorced	Separated	Widowed	Did not report
	62.6%	13.5%	13.9%	4.7%	1.4%	3.7%

Education	Graduate or professional school	Some graduate or professional school	Post-secondary degree or diploma	Some college	High school	Some high school	8th grade or less	Did not report
	7.6%	12.5%	26.4%	16.2%	17.2%	6.1%	.7%	3.4%

Employment	Full time	Part time	Stay-at-home caregiver	Unemployed	Student	Medical disability	Retired	Did not report
	46.3%	18.9%	16.9%	9.5%	3%	1.4%	.3%	3.7%

report. Following recommendations in the literature, this score was a combination of the primary female caregiver's highest level of education and average household income, which were standardized and summed, labeled summed education/income (Cohen, Doyle, et al., 2006). Education was coded as 1 = *Grade 8 or less*, 2 = *some high school*, 3 = *completed high school*, 4 = *some university*, 5 = *college diploma*, 6 = *university degree*, 7 = *some graduate school/professional school*, and 8 = *completed graduate/professional school*. Caregivers reported income in $10,000 increments, which were coded into six $20,000 increments ranging from "under $20,000" to "over $100,000." This coding was used to better equalize distribution of responses across the categorical options.

The second score was based on census tract information. Addresses were entered into the Federal Financial Institutions Examination Council (FFIEC) geocoding/mapping system or the address search tool in the American FactFinder to obtain corresponding census tract income level (an index of neighborhood affluence). The census assigns each tract an income level by comparing the median family income of a census tract to the median family income of the metropolitan statistical area or metropolitan division in which the census tract is situated. Tract income levels are classified as 1 = *low* (tract median income is less than 50% of that corresponding metropolitan statistical area [MSA]), 2 = *moderate* (tract median income is greater than or equal to 50% but less than 80% of that corresponding MSA), 3 = *middle* (tract median income is greater than or equal to 80% but less than 120% of that corresponding MSA), or 4 = *upper* (tract median income is greater than 120% of that corresponding MSA).

Trier Social Stress Test for Children (TSST-C). Participants engaged in an adapted version of the TSST-C (Buske-Kirschbaum et al., 1997), a laboratory paradigm consisting of a storytelling and math task designed to elicit a stress response to a psychosocial stressor. Participants were told that their storytelling performance would be judged. They were given a story prompt and then had 3 min to prepare a middle and end for the story. They were told they should try to do better than all the other children that had participated in the study. After the preparation time, the participants were asked to finish telling the story to two storytelling experts (i.e., unacquainted judges) for 5 min. The judges were instructed to maintain a serious, unsmiling, and neutral demeanor without giving verbal or nonverbal feedback. One judge turned on a video camera and pretended to turn on a microphone that was handed to the participant. Participants were prompted to continue if they paused for longer than 20 seconds. After 5 min, participants were presented with the second half of the paradigm, an unexpected 5-min-long arithmetic task, (i.e., "Starting with the number 758 we want you to count down by subtracting 7"). If the participant made an error, they were asked to start over. After 5 errors, the judges provided new starting and count numbers (i.e., "Starting with the number 307 we want you to count down by subtracting 3"). At the end of the task, judges enthusiastically praised the participant's performance. Caregivers observed their child's performance through a one-way mirror to facilitate careful observation of the participant's distress level throughout the task. There were 64 instances where the task was discontinued either by the tester, the parent, or the child participant. Among these 64 instances, 18 were explicitly discontinued by the child participant. Parents and testers chose to discontinue the task if they deemed that the child participant appeared distressed (i.e., the participant was on the verge of tears or began to cry), erring on the side of discontinuation if in doubt. Analyses indicated that this subsample showed significant reactivity to task exposure (see details in the online supplemental information), thus, we retained the data of those 64 participants in all analyses.

Procedures

Caregivers completed informed consent and the majority of questionnaires upon arrival to the lab. All hormone samples were collected between noon and sundown (range: 12:00:00 to 19:07:16, M = 14:59:45, SD = 1:50:46) to account for the diurnal variation in cortisol levels (Kirschbaum & Hellhammer, 1994). The exact timing of sundown was determined by consulting online references for that particular assessment date. Female participants who had begun menstruating were scheduled during the first 10 days of their cycle (follicular phase), when hormones are most stable (Liening, Stanton, Saini, & Schultheiss, 2010). All parents were instructed not to have their child eat or drink for 2 hours before the assessment. Once the child had given assent, they rinsed their mouth out with water and were instructed to drink a small cup of water. After 30 min of sedentary activity (questionnaire and computer task completion), the baseline saliva sample was collected. Saliva samples were then collected 20 min after the start of the TSST (excluding speech preparation time, so from the beginning of the speech delivery; see also Buske-Kirschbaum et al., 1997; Dickerson & Kemeny, 2004; T2), 40 min after the start of the TSST (T3), and 60 min after the start of the TSST (T4). Participants drooled through a sanitary straw into a 2-ml IBL vial. All samples were then frozen at −20° C, and shipped on dry ice to Clemens Kirschbaum at the Technical University of Dresden, where they were immunoassayed for cortisol measured in nmol/L units (IBL Int., Hamburg, Germany). The average inter- and intraassay coefficients for cortisol were below 8%. All study methods and materials received approval from the University of Houston institutional review board. Parents were compensated with a $75 gift card, and the child was compensated with a small prize. The study took approximately 3.5 hours, and included questionnaires and behavioral tasks.

Results

Descriptive statistics (mean values and standard deviations) for all study variables can be found in Table 2. Zero-order correlations between race, gender, age, LEQ, SES, and hormone variables are presented in Table 3. Cortisol samples for three children were dropped from analyses because they were either receiving insulin (n = 2) or steroid treatment (n = 1). Variable frequencies were examined and 23 extreme values for cortisol (more than three standard deviations above the mean) were winsorized before analyses (replaced with the highest value in the data less than 3 SD). Cortisol variables were log (base 10)-transformed to reduce skew. Cortisol reactivity was calculated by subtracting baseline cortisol (T1) from cortisol at T2. Cortisol recovery was calculated by subtracting cortisol at T4 from cortisol at T2. All statistical analyses were performed using Statistical Package for the Social Sciences Version 22 (SPSS 22). All mediation analyses were performed using the PROCESS macro for SPSS (Hayes, 2013).

Table 2
Descriptive Characteristics of the Sample on Hormones and SES Variables

Number of participants for cortisol	Baseline cortisol	Cortisol reactivity (T2–T1)	Cortisol recovery (T2–T4)	Number of participants for EI	EI	Number of participants for FAM	FAM
	M (*SD*)	*M* (*SD*)	*M* (*SD*)		*M* (*SD*)		*M* (*SD*)
Total (N = 284)	5.34 (4.45)	3.08 (6.30)	3.61 (5.06)	Total (N = 284)	.01 (1.73)	Total (N = 257)	2.54 (2.34)
Male (N = 130)	5.57 (4.66)	3.07 (6.25)	3.76 (5.30)	Male (N = 132)	.01 (1.72)	Male (N = 121)	2.68 (2.49)
Female (N = 154)	5.15 (4.26)	3.09 (6.36)	3.49 (4.88)	Female (N = 147)	.00 (1.76)	Female (N = 136)	2.43 (2.21)
Black (N = 97)	5.12 (4.84)	1.23 (3.88)	2.17 (4.08)	Black (N = 97)	−.77 (1.64)	Black (N = 85)	2.88 (2.56)
White (N = 101)	5.63 (4.19)	4.46 (7.17)	4.65 (5.30)	White (N = 104)	1.27 (1.24)	White (N = 94)	2.21 (2.26)
Hispanic (N = 86)	5.25 (4.31)	3.61 (7.00)	4.06 (5.46)	Hispanic (N = 83)	−.67 (1.43)	Hispanic (N = 78)	2.58 (2.17)
NA = 1 (N = 39)	4.11 (2.37)	1.78 (4.46)	2.03 (3.61)	NA = 1 (N = 39)	−1.39 (1.40)	NA = 1 (N = 34)	2.85 (2.31)
NA = 2 (N = 80)	4.95 (3.27)	1.70 (3.80)	2.50 (3.46)	NA = 2 (N = 78)	−.87 (1.31)	NA = 2 (N = 69)	2.91 (2.40)
NA = 3 (N = 48)	6.90 (7.47)	1.69 (6.73)	3.80 (6.07)	NA = 3 (N = 50)	−.24 (1.68)	NA = 3 (N = 45)	2.89 (2.70)
NA = 4 (N = 115)	5.42 (3.88)	4.98 (7.39)	4.74 (5.55)	NA = 4 (N = 115)	1.21 (1.29)	NA = 4 (N = 107)	2.07 (2.10)

Note. SES = socioeconomic status; EI = summed z-score of education and income variables; FAM = family level stress variable from the Life Events Questionnaire; NA = neighborhood affluence; cortisol are raw values in nmol/l units, however, transformed values are used in analyses.

Hierarchical regression models were estimated to examine the incremental variance in cortisol reactivity and recovery predicted by summed education/income, neighborhood affluence, or FAM over and above race/ethnicity. Dependent variables were cortisol reactivity and cortisol recovery at T4. At Step 1, all models included the covariates sex, age, and time of waking.[1] At Step 2, the first two regressions included only dummy-coded race/ethnicity (separately for Black and Hispanic children, with White as the comparison group) to examine the main effect of race/ethnicity on cortisol reactivity and recovery. At Step 2, additional regressions each included either summed education/income, neighborhood affluence, or FAM to examine the main effect of these variables on cortisol reactivity and recovery. These regressions also included race/ethnicity at Step 3 to examine the incremental contributions of summed education/income, neighborhood affluence, or FAM controlling for race/ethnicity effects. Results are presented by step of the hierarchical regression models (see Table 4).

Presented first are results of the models with cortisol reactivity as the dependent variable. The first regression showed a main effect of race/ethnicity on cortisol reactivity. Specifically, at Step 2, cortisol reactivity was lower in Black than in White children (b = −0.14, SE_b = 0.04, 95% confidence interval (CI) [−0.21, −0.07], $p < .001$) and did not differ between Hispanic and White children (b = −0.04, SE_b = 0.04, 95% CI [−0.11, 0.03], p = .271). Regression models including SES variables showed main effects of summed education/income and neighborhood affluence, respectively, on cortisol reactivity. Specifically, at Step 2, cortisol reactivity was positively predicted by summed education/income (i.e., higher SES; b = 0.03, $p < .001$) and by neighborhood affluence (i.e., higher SES; b = 0.05, $p < .001$). The regression including FAM did not show a main effect of FAM on cortisol reactivity (b = −0.01, p = .449). To examine incremental contributions of summed education/income, neighborhood affluence, or FAM controlling for race/ethnicity effects, we included race/ethnicity in Step 3 of the regression models that included SES variables. Results showed that cortisol reactivity was positively predicted by summed education/income (higher SES; b = 0.03, p = .005) and neighborhood affluence (b = 0.03, p = .038) over and above race/ethnicity (and race/ethnicity largely remained a significant predictor as well, with the exception of the model with summed education/income).

Presented next are results of the models with cortisol recovery as the dependent variable. The first regression model showed a main effect of race/ethnicity on cortisol recovery. Specifically, at Step 2, cortisol recovery was lower in Black than White children (b = −0.08, SE_b = 0.03, 95% CI [−0.13, −0.03], p = .001) and did not differ between Hispanic and White children (b = −0.01, SE_b = 0.03, 95% CI [−0.06, 0.04], p = .613). Of the regression models including SES variables, only neighborhood affluence showed a main effect on cortisol recovery. Specifically, at Step 2, cortisol recovery was positively predicted by neighborhood affluence (i.e., higher SES with higher recovery; b = 0.03, p = .009). The remaining regression models did not support a main effect of summed education/income (b = 0.01, p = .108) or FAM (b = 0.00, p = .756) on cortisol recovery, respectively. To examine incremental contributions of summed education/income, neighborhood affluence, or FAM controlling for race/ethnicity effects, we included race/ethnicity in Step 3 of the regression models that included SES variables. Cortisol recovery was not positively predicted by either summed education/income, neighborhood affluence, or FAM over and above race/ethnicity; race/ethnicity remained a significant predictor.

Finally, mediation models (Hayes, 2013) were estimated to examine whether the race—cortisol associations were mediated by summed education/income, neighborhood affluence, or FAM. We tested for mediation among all racial groups despite not all racial groups differing from one another on cortisol, given that an indirect effect is possible even in the context of a nonsignificant total effect (see Hayes, 2013). The hypothesized mediation model is displayed in Figure 1. In both racial group comparisons, the association between race and cortisol reactivity was mediated by summed education/income (Black: ab = −0.06; CI [−0.11, −0.02]; Hispanic: ab = −0.06, CI [−0.10, −0.02]) and by neighborhood affluence (Black: ab = −0.05, CI [−0.09, −0.01]; Hispanic: ab = −0.03, CI [−0.06, −0.01]), but not by FAM (Black: ab = 0.00; CI [−0.01, 0.01]; Hispanic: ab = 0.00, CI [−0.01, 0.00]). The direct

[1] We reran our analyses using BMI as an additional covariate and found an identical pattern to the one reported in the main manuscript, with one exception: summed education/income now also predicted cortisol recovery.

Table 3
Correlation Coefficients

	1	2	3	4	5	6	7	8	9
1. Black	—								
2. Hispanic	−.48***	—							
3. Gender	−.03	−.03	—						
4. Age	.02	.00	−.06	—					
5. FAM	.10	.01	−.05	.13	—				
6. EI	−.31***	−.26***	−.01	−.05	−.25***	—			
7. NA	−.45***	−.10	.02	−.13*	−.15*	.59***	—		
8. Cortisol reactivity	−.21***	.06	.00	.09	−.05	.20**	.22***	—	
9. Cortisol recovery	−.21**	.06	−.03	.03	.07	.14*	.22***	.81***	—

Note. Black = dummy coded variable where 1 = Black; Hispanic = dummy coded variable where 1 = Hispanic; FAM = family level stress variable from the Life Events Questionnaire; EI = summed z-score of education and income variables; NA = neighborhood affluence.
* $p < .05$. ** $p < .01$. *** $p < .001$.

effect of race on cortisol reactivity was no longer significant once the indirect effect of summed education/income was accounted for (Black: $c' = -0.07$, $p = .078$; Hispanic: $c' = 0.02$, $p = .718$); however, the direct effect of race was still significant after accounting for the indirect effect of neighborhood affluence (Black: $c' = -0.09$, $p = .030$; Hispanic: $c' = -0.02$, $p = .699$). These mediation results are displayed in Figure 2. The association between race and cortisol recovery was not mediated by summed education/income (Black: ab = −0.01, CI [−0.04, 0.02]; Hispanic: ab = −0.01, CI [−0.04, 0.02]), neighborhood affluence (Black: ab = −0.02, CI [−0.05, 0.01]; Hispanic: ab = −0.01, CI [−0.03, 0.01]), or FAM (Black: ab = 0.00; CI [−0.01, 0.01]; Hispanic: ab = 0.00, CI [0.00, 0.01]).

Discussion

Results from the present study supported the primary hypothesis, such that racial group differences in cortisol reactivity (but less so for recovery) were partially accounted for by differences in maternal education/family income and neighborhood affluence. These findings support the "wear-and-tear" theory regarding the effects of socioeconomic status and contextual stress on physiological functioning (McEwen, 2004), even as early as preadolescence. They also build on recent work identifying other stressors (e.g., perceived discrimination) that disproportionately affect members of racial and ethnic minority groups and have deleterious consequences for individuals' physiological functioning and, ultimately, health and mental health outcomes by demonstrating substantial (but only partial) explanatory power of socioeconomic status for racial/ethnic differences in the acute cortisol stress response. Specifically, a full account of relevant processes must incorporate both race-specific stressors and broad stressors such as those related to low socioeconomic status. These findings offer the first rigorous investigation of SES and race/ethnicity as independent factors impacting the acute cortisol stress response, with previous research primarily focusing on aspects of diurnal cortisol

Table 4
Hierarchical Regression Analyses Predicting Cortisol Response From Race/Ethnicity, Education + Income (EI), Neighborhood Affluence (NA), and Family-Level Events (FAM)

	Model											
	Including EI (n = 257–258)				Including NA (n = 264–265)				Including FAM (n = 235)			
Step	Variable	B	SE_B	95% CI	Variable	B	SE_B	95% CI	Variable	B	SE_B	95% CI
						DV: Cortisol reactivity						
2	EI	.03***	.01	.02, .05	NA	.05***	.01	.03, .08	FAM	−.01	.01	−.02, .01
3	Black	−.07	.04	−.16, .01	Black	−.09*	.04	−.18,−.01	Black	−.17***	.04	−.24,−.09
	Hispanic	.02	.04	−.07, .10	Hispanic	−.02	.04	−.09, .06	Hispanic	−.06	.04	−.14, .02
	EI	.03**	.01	.01, .05	NA	.03*	.02	.00, .06	FAM	.00	.01	−.02, .01
						DV: Cortisol recovery						
2	EI	.01	.01	.00, .02	NA	.03**	.01	.01, .04	FAM	.00	.01	−.01, .01
3	Black	−.07*	.03	−.13,−.01	Black	−.06*	.03	−.12,−.003	Black	−.09**	.03	−.15,−.04
	Hispanic	.00	.03	−.06, .06	Hispanic	−.01	.03	−.06, .05	Hispanic	−.02	.03	−.08, .03
	EI	.00	.01	−.01, .02	NA	.01	.01	−.01, .04	FAM	.00	.01	−.01, .01

Note. Separate hierarchical regression analyses (indicated by letters next to the step) were conducted for each combination of race/ethnicity and EI, race/ethnicity and NA, and race/ethnicity and FAM to examine their incremental validity in predicting cortisol reactivity and recovery. For all models, age, sex, and time of waking were entered in Step 1. None of the covariates robustly predicted cortisol reactivity and recovery and were therefore omitted from the table. Model estimates are displayed for variables of interest at each step (omitted estimates available upon request).
* $p < .05$. ** $p < .01$. *** $p < .001$.

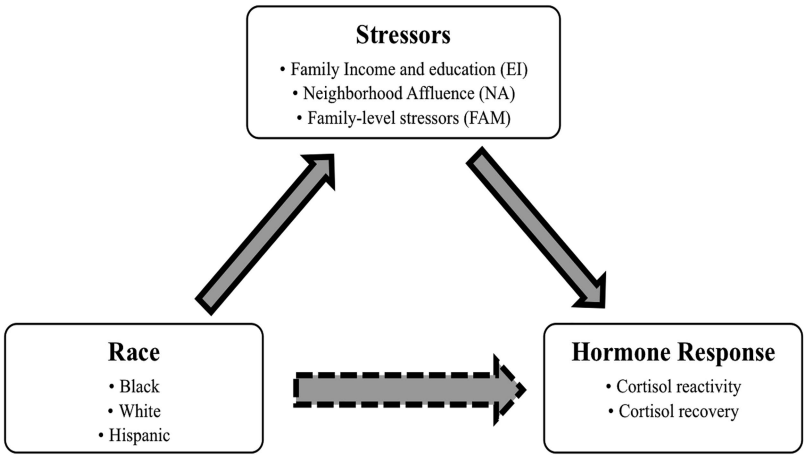

Figure 1. Theoretical mediation model illustrating the mediation of the relationship between race and hormone response by stress variables under consideration in the present study.

functioning or basal cortisol levels. Most importantly, these findings offer the first empirical effort to determine whether racial differences in cortisol reactivity are potentially reflecting the impact of socioeconomic status and contextual adversity. Such work is critical for better understanding pathways to adaptive and maladaptive health outcomes, as altered cortisol functioning may play a key role in mapping these pathways (e.g., Blair et al., 2005; Burke et al., 2005; Conradt et al., 2014; Phillips et al., 2013; Skinner et al., 2011).

The overall pattern of findings between racial/ethnic groups was similar to previous research on the impact of SES and related stressors on altered cortisol levels and patterns: Black children showed the lowest levels of cortisol reactivity and recovery, relative to both White and Hispanic children, who did not significantly

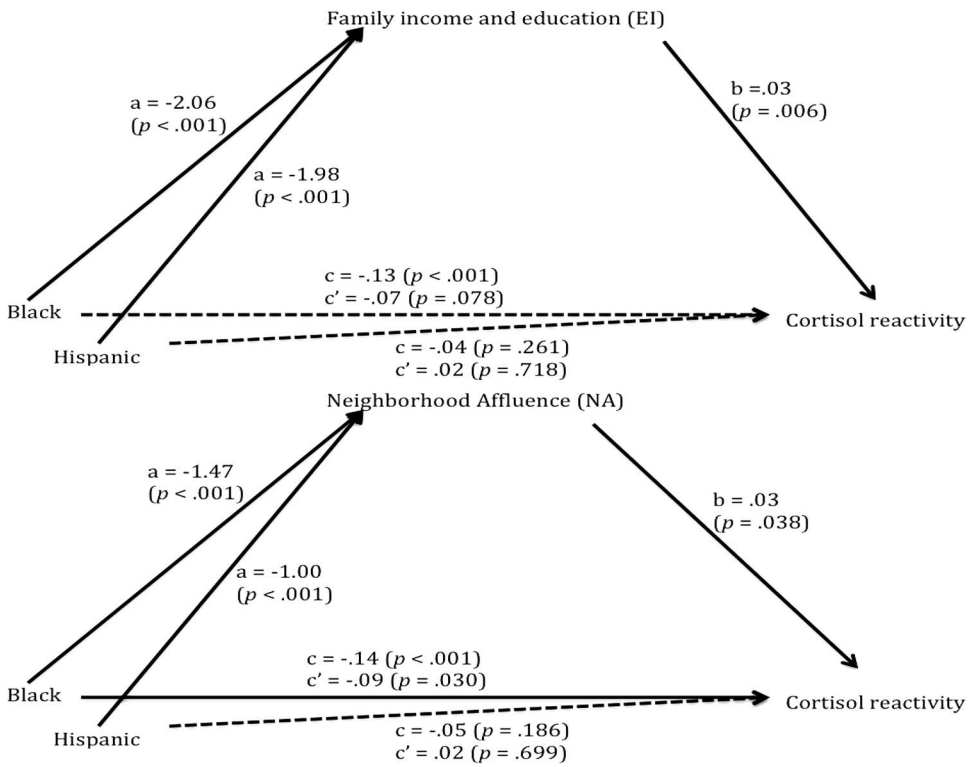

Figure 2. Graphical representation of the mediation results of the present study. Family income and education (EI) and neighborhood affluence (NA) mediate the pathway from race to cortisol reactivity.

differ from each other. The primary goal of this study was to take this finding a step further, and try to unpack potential mechanisms underlying these group differences by directly comparing race/ethnicity and SES in hierarchical regression analyses, and examining evidence for SES mediation of race/ethnicity associations with acute cortisol reactivity. Three types of stress/adversity were examined: levels of general life stressors, maternal highest level of education and family income, and neighborhood affluence (or, conversely, deprivation). As expected, the experience of general life stressors did not incrementally predict altered cortisol reactivity and recovery, above and beyond race/ethnic group membership, nor did general life stressors mediate the association between race/ethnic group membership and cortisol reactivity and recovery. Thus, altered cortisol reactivity and recovery were not a function of more normative, and widely distributed, experiences of life stress and hassle.

Both measures of SES, however, did lend support to our primary hypothesis. Both proximal (summed education/income) and distal (neighborhood affluence) SES indicators incrementally predicted cortisol reactivity in expected directions (higher SES leads to higher cortisol reactivity), even after controlling for race/ethnic group membership. Prediction of cortisol recovery did not show this pattern, however. In addition, race/ethnicity showed unique predictive variance, even after controlling for SES, in all analyses with the exception of the model including summed education/income predicting cortisol reactivity. These findings suggest that SES plays an important and incremental role in understanding race/ethnicity differences in cortisol reactivity, but also indicate that additional variance may be unexplained by the SES variables measured here. The next set of analyses directly tested evidence for mediation of stress and SES in accounting for the associations between race/ethnic group membership and cortisol reactivity and recovery. Once again, as expected, general life stressors did not mediate the association between race/ethnicity and cortisol reactivity or recovery. Both measures of SES, however, partially mediated these associations. Proximal SES (summed education/income) and distal SES (neighborhood affluence) partially mediated the association between race/ethnic group membership and cortisol reactivity. Mediation was not found for associations between race/ethnic group membership and cortisol recovery. These results further suggest that SES represents an especially deleterious and impactful proxy for stressful experiences powerful enough to disrupt adaptive physiological functioning, even as early as middle childhood.

In the context of recent work (Adam et al., 2015; Skinner et al., 2011), which has highlighted specific psychological constructs such as perceived discrimination as resulting in long-term differences in cortisol functioning, these measures of SES may reflect broader proxies for multiple types of psychological adversity (potentially also including greater severity of life hassles, which may be experienced more chronically by low SES individuals—a distinction not often well captured in global checklists of life stress exposure). Perceived discrimination was not measured in the current study, but reflects another systemic and consequential stressor that is disproportionately experienced by members of racial/ethnic minority groups, similar to SES. In general, these findings highlight the importance of considering potential pathways through which SES affects health, as well as evidence for race-specific stressors operating beyond the impact of SES. Socioeconomic status may increase exposure to stressors (e.g., relationship problems, psychological symptoms; Santiago, Wadsworth, & Stump, 2011), which in turn may impact health. Socioeconomic status also shows influence on motivation, attention, and cognitive control (e.g., Blair et al., 2008; Pilgrim, Marin, & Lupien, 2010), which may have downstream consequences on physiology and health. Yet, these findings also suggest that stressors that may be experienced broadly across SES strata (e.g., family conflict), as measured with the LEQ in the current study, may not be the most relevant or deleterious aspects of environmental stress impacting children's health and physiological functioning. Closer examination of potential mechanisms will be an important direction for future research.

In addition, previous studies examined measures of cortisol functioning other than stress reactivity, and largely measured cortisol functioning in adulthood. Thus, the current research is consistent with this evidence but extends it as well. It provides input for developmental theories regarding the impact of high adversity on cortisol functioning. For example, suggestions that conditions of high stress may result in a shift from hypercortisolism to hypocortisolism in childhood (Lupien, King, Meaney, & McEwen, 2000) should be informed by the current study, which might indicate that such a shift, if it does occur, happens much earlier than middle childhood. Future studies will hopefully build on this growing evidence base and further link individual studies to one another, in hopes of gaining a broader picture of stress experienced by racial/ethnic minority group members, and its impact on physiological functioning, across the life span.

Limitations and Future Directions

As with all psychological research, this study is not without its limitations, which point to important future directions for research. The importance of examining multiple aspects of cortisol functioning has been highlighted, and indeed, separate examination of cortisol recovery (not just reactivity) may sometimes be illuminating (e.g., Tackett et al., 2014). In the present investigation, however, we found little evidence for individual differences in SES impacting cortisol recovery (although race/ethnicity did predict recovery patterns), with hypothesized effects for SES largely emerging for cortisol reactivity. Future research should continue to map effects of SES and contextual stress across different aspects of cortisol functioning to better understand the underlying physiological mechanisms involved in "getting under the skin." It is also important to note that, whereas the recovery response can be differentiated from the reactivity response, overall diminished reactivity (as seen in Black children in the current study) may account for restricted variance in recovery, limiting its potential in testing the hypotheses presented here. Furthermore, most of the existing literature has examined basal cortisol rather than reactivity. Future studies should incorporate both for a better understanding of multiple aspects of HPA axis functioning.

Another important area for future research is evaluating threat responses to the TSST-C among youth. Research evaluating threat response and discrimination has suggested that individuals may experience a greater stress response when they are given feedback that is discrepant to their performance, especially when the feedback is provided by individuals of different racial/ethnic backgrounds (Major et al., 2016). Although we did not explicitly code

praise and performance on the task, it is important for future research to determine whether increased threat is being invoked for minority youth and how that maps onto the current findings.

Some of the advances offered in the current study—namely, examination of effects in middle childhood and across multiple levels of stress, socioeconomic status, and contextual adversity—also highlight how little we know about the development of these effects across time and via various environmental contexts. That is to say, longitudinal work as well as cross-sectional work on focal age groups will be critical to further understand how early these effects emerge. For example, comparing these findings to studies with adolescents (DeSantis et al., 2007), we might wonder whether Hispanic children show a more delayed developmental impact of SES on cortisol functioning. Although the current study found no differences between Hispanic and White children's cortisol reactivity, DeSantis and colleagues (2007) found both Hispanic and African American adolescents to show flatter cortisol slopes than White adolescents.

In addition, we offer one example of the potential utility of examining levels of stress and adversity that span the individual and proximal and distal sociocultural context, but many more environmental stressors and contexts need to be examined to better understand those sociocontextual features carrying much of the explanatory power (Bush et al., 2011), as well as the extent to which contextual factors may interact with one another (e.g., Fuller-Rowell, Doan, & Eccles, 2012) and with race (Skinner et al., 2011). Examination of the impact of race-specific stressors on HPA axis function remains a critical area for future research. Furthermore, examination of stigma, including structural stigma (Hatzenbuehler & McLaughlin, 2014), and variables such as racial composition and segregation of neighborhoods is also important for future research in this area. Inclusion of such constructs in empirical designs would offer the opportunity to explore potential mechanisms, or alternative explanations, for broader contextual/neighborhood effects such as those reported here. Ultimately, fully contextualized longitudinal examinations (e.g., Adam et al., 2015) will be needed to elucidate transactional processes developing across time, and their resultant impact on developing stress physiology. In addition, future work should also examine these variables in the context of social deprivation theory (Crosby, 1976), and specifically examine whether differences emerge for subjective versus objective SES and subjective-objective SES discrepancies. These represent important areas for future research.

Conclusion

The current study sought to challenge the robust existing evidence for racial/ethnic differences in cortisol functioning, by asking whether such differences may be partially accounted for by differences in SES. In a racially/ethnically and socioeconomically diverse sample in middle childhood, effects of stress, SES, and contextual adversity were further examined across different levels, from individual-level life stressors, to proximal SES (maternal education and household income), to distal SES (neighborhood affluence). These findings suggest that some racial differences in cortisol reactivity are mediated by differences in distal and proximal SES, which are further emphasized by the emergence of these effects relatively early in the life span, in middle childhood. However, some racial differences in cortisol (particularly those for recovery) remain above and beyond the effects of SES. Thus, socioeconomic status and contextual adversity appear to be a major driving force in observed differences in stress physiology functioning that may substantially account for some differences between racial groups. These findings have policy and intervention implications as well, underscoring the relevance of environmental/contextual factors in this process. Interventionists and policymakers must continue focusing effort toward broader, systemic, sociocultural changes that target sources of psychosocial adversity related to low SES. Results from this, and similar, research suggest that interventions should be targeting individuals in early childhood, with greater attention to mitigating the consequences of low SES and contextual adversity for those at greatest risk.

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