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Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: A 20-year prospective study

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ABSTRACT

Perceived racial discrimination (PRD) has been associated with altered diurnal cortisol rhythms in past cross-sectional research. We investigate whether developmental histories of PRD, assessed prospectively, are associated with adult diurnal cortisol profiles. One-hundred and twelve ($N = 50$ Black, $N = 62$ White) adults from the Maryland Adolescent Development in Context Study provided saliva samples in adulthood (at approximately age 32 years) at waking, 30 min after waking, and at bedtime for 7 days. Diurnal cortisol measures were calculated, including waking cortisol levels, diurnal cortisol slopes, the cortisol awakening response (CAR), and average daily cortisol (AUC). These cortisol outcomes were predicted from measures of PRD obtained over a 20-year period beginning when individuals were in 7th grade (approximately age 12).

Greater average PRD measured across the 20-year period predicted flatter adult diurnal cortisol slopes for both Black and White adults, and a lower CAR. Greater average PRD also predicted lower waking cortisol for Black, but not White adults. PRD experiences in adolescence accounted for many of these effects. When adolescent and young adult PRD are entered together predicting cortisol outcomes, PRD experiences in adolescence (but not young adulthood) significantly predicted flatter diurnal cortisol slopes for both Black and White adults. Adolescent, but not young adult PRD, also significantly predicted lower waking and lower average cortisol for Black adults. Young adult PRD was, however, a stronger predictor of the CAR, predicting a marginally lower CAR for Whites, and a significantly larger CAR for Blacks. Effects were robust to controlling for covariates including health behaviors, depression, income and parent education levels. PRD experiences interacted with parent education and income to predict aspects of the diurnal cortisol rhythm. Although these results suggest PRD influences on cortisol for both Blacks and Whites, the key findings suggest that the effects are more pervasive for Blacks, affecting multiple aspects of the cortisol diurnal rhythm. In addition, adolescence is a more sensitive developmental period than adulthood for the impacts of PRD on adult stress biology.

1. Introduction

1.1. Overview

Racial and ethnic disparities exist across a wide range of adult health conditions (Williams and Collins, 1995; Mensah et al.,

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2005; Myers, 2009; Williams and Mohammed, 2009). Differing health care access and health behaviors do not appear to fully account for these disparities, leading investigators to propose that race-based social stress such as perceived racial discrimination (PRD) may play a role, by way of its influence on stress biology (Kuzawa and Sweet, 2009; Williams and Mohammed, 2009). The hypothalamic–pituitary–adrenal (HPA) axis and its primary product cortisol are frequently implicated in theoretical models of race-based stress and health (Myers, 2009). Racial/ethnic differences have been found in diurnal cortisol rhythms (Cohen et al., 2006; DeSantis et al., 2007), and perceived discrimination has been associated with altered basal/diurnal levels of cortisol in past research (Kaholokula et al., 2012; Zeiders et al., 2012).

Most existing research on discrimination and cortisol, including the current study, has focused primarily on interpersonal forms of discrimination, including daily hassles and microaggressions related to race/ethnicity (Harrell, 2000; Sue et al., 2007). Past research on discrimination and cortisol has also been cross-sectional, focusing on current or recent PRD, rather than cumulative histories of past PRD exposure. In the current study, we examined the impact of cumulative exposure to PRD assessed prospectively over a 20-year period. We also examined whether PRD experiences measured in adolescence were more strongly related to adult cortisol than PRD experiences measured in early adulthood. We controlled for potential confounds related to cortisol levels and/or discrimination experiences, including health behaviors, socioeconomic variables, and depressive symptoms. We also tested whether effects of histories of PRD on cortisol are stronger for Black adults and for individuals with lower socioeconomic status. Before describing our methods, we give a brief introduction of perceived discrimination and on HPA axis activity, then review past research on associations between them. Methodological issues, such as the importance of considering developmental timing, socioeconomic context, and potential confounds are discussed. Finally, the current project is described.

1.2. Perceived discrimination

Perceived discrimination involves an individual perceiving that they are receiving or have received unfair treatment on the basis of membership in a group (Tajfel, 1982; Fishbein, 1996; Brown and Bigler, 2005; Major and Kaiser, 2008). When asked about perceived discrimination across a range of categories (e.g., gender, race, ethnicity, age, religion, physical appearance, sexual orientation), racial and ethnic minorities in the U.S. describe higher levels of discrimination than their White counterparts (Kessler et al., 1999). Due to the fact that racial/ethnic categorization is relatively stable, and as result of historical mistreatment and oppression based on race within U.S. society, discrimination based on race/ethnicity (perceived racial/ethnic discrimination, PRD) may have particularly strong effects on the well-being of racial/ethnic minority individuals, including Black Americans (Branscombe et al., 1999; Harrell, 2000; Feagin et al., 2001).

1.3. Stress-related changes in diurnal cortisol rhythms

Basal cortisol levels follow a strong circadian or diurnal rhythm, involving high levels upon waking, a substantial (50–60%) increase in the 30–40 min after waking (the cortisol awakening response or CAR), and a subsequent decline across the day, reaching a nadir around midnight (Pruessner et al., 1997; Adam and Kumari, 2009).

Periodic activation of the HPA axis is considered adaptive and necessary to cope with acute stress, and cortisol levels are particularly responsive to stress of a social-evaluative nature (Dickerson and Kemeny, 2004). Stress-related changes in several aspects of the diurnal cortisol rhythm have been identified. The CAR has been found to increase in the presence of acute daily stressors (Adam et al., 2006; Chida and Steptoe, 2009; Fries et al., 2009). It has been found to be lower, however, in the presence of traumatic stress, particularly when accompanied by post-traumatic stress symptoms or disorders (Rohleder et al., 2004; Wessa et al., 2006). Chronic stress has also been associated with lower waking cortisol levels flatter diurnal cortisol slopes (Adam, 2012; Doane et al., 2013). Flatter cortisol slopes have been linked to higher depression (Doane et al., 2013), fatigue (Bower et al., 2005; Kumari et al., 2009), and cardiovascular disease (Matthews et al., 2006; Kumari et al., 2011). Chronic, and particularly traumatic stress, also predicts overall reductions in cortisol across the day, known as hypocortisolism (Heim et al., 2000; Fries et al., 2005; Miller et al., 2007). Hypocortisolism is associated with the presence of fatigue and pain syndromes, and overactivation of immune and inflammatory systems (Fries et al., 2009).

1.4. Perceived discrimination and diurnal cortisol rhythms

Past research has consistently found flatter diurnal cortisol rhythms in African Americans as compared to Whites (Cohen et al., 2006; DeSantis et al., 2007, 2015; Skinner et al., 2011; Martin et al., 2012). Higher PRD has been proposed as a potential mediator between race/ethnicity and flatter diurnal cortisol slopes (DeSantis et al., 2007). There are relatively few empirical studies of associations between PRD and diurnal cortisol rhythms. Some studies have examined major life events related to racism, although most have focused primarily on everyday interpersonal discrimination (daily race-related hassles and microaggressions).

One study of young adults (Skinner et al., 2011) assessed retrospective lifetime racism and racial daily hassles and found that discrimination predicted flatter diurnal cortisol slopes among both Black and White youth. Another study found associations between everyday PRD and flatter diurnal cortisol slopes among racial/ethnic minority young adults, but not racial/ethnic majority group members (Zeiders et al., 2014). This latter result is in line with prior evidence showing that racial/ethnic minorities are more sensitive to the effects of stress on the cortisol diurnal rhythm (DeSantis et al., 2015). In pregnant women, Suglia et al. (2010) found cumulative stress, including measures of major and everyday PRD, predicted lower morning cortisol and flatter waking to bedtime cortisol slopes for Black, but not Hispanic women. By contrast, one study of adults found that everyday discrimination predicted flatter diurnal cortisol slopes in White adults, but steeper diurnal cortisol slopes in Black adults (Fuller-Rowell et al., 2012b). Another study of preadolescents did not find significant associations between everyday discrimination and cortisol diurnal rhythms (Martin et al., 2012).

Less research has focused on PRD and average cortisol levels, however a study of Hawaiians found perceived racism to be associated with an overall lowering of cortisol levels across the day among Native Hawaiians (Kaholokula et al., 2012). Thus, with a few exceptions, existing research suggests that PRD is associated with a flattening of the diurnal cortisol rhythm and potentially an overall lowering of the diurnal cortisol curve across the day, with some evidence that effects are stronger for racial/ethnic minorities.

1.5. The importance of developmental histories

Prior empirical research on discrimination and cortisol has focused on recent discrimination, rather than taking into account histories of exposure. As a result, past work has not been able to assess effects of chronic PRD exposure, or the relative impacts of PRD exposure at different developmental stages. Life-span developmental theories suggests that experiences may have cumulative impacts on biology over time; events occurring during times of

rapid developmental transition are, however, likely to have larger effects, becoming "built-in" to an individual's changing biology or psychology (Halfon and Hochstein, 2002). The biological "embedding" of experiences during infancy and early childhood have received considerable attention (Hertzman, 1999; Shonkoff et al., 2009; Miller and Chen, 2013; Nelson, 2013). More recently, adolescence has been recognized as an additional sensitive period, in part due to notable changes in brain and neuroendocrine development during this time period (Spear, 2000; Chambers et al., 2003; Dahl, 2004).

Adolescence is also a key period in the development of identity (Kroger, 2006), and for racial/ethnic minority youth, developing a racial/ethnic identity becomes salient (French et al., 2006; Umaña-Taylor et al., 2014). The presence of a strong racial/ethnic identity, conferred in part through racial/ethnic socialization by parents, has been found to be protective against the negative impact of discrimination (Neblett et al., 2008, 2012). For adolescents, however, racial/ethnic socialization and racial/ethnic identity development are still in progress (Boykin and Toms, 1985). Adolescents may also be less ready to employ important coping skills, ranging from support seeking to emotion regulation (Brondolo et al., 2009). PRD experiences may thus have stronger effects in adolescence as compared to adulthood, due to immature buffering mechanisms and coping resources. Adolescent PRD may also affect adult outcomes by becoming "built in" to developing biological and psychological systems (for example, by impacting self-esteem). As a result, we hypothesize that PRD experiences during adolescence will be a stronger predictor of adult HPA axis functioning than PRD experiences in young adulthood, particularly for Black Americans.

1.6. Contexts of race-based discrimination

Racial discrimination and racial disparities in stress hormones are not experienced in a vacuum; they occur in particular neighborhood, family and socioeconomic contexts (Brondolo, 2015). The socioeconomic context in which perceived discrimination occurs may be particularly important in several ways (Harrell, 2000; Meyers, 2009). First, given that Blacks are more likely to live in lower income families, it is important to ensure that effects attributed to perceived racial discrimination for Blacks are not accounted for by the economic conditions of the family, and co-occurring stressors (Meyers, 2009). Second, the additional stress associated with living in a low-income environment may exacerbate the negative effects of perceived discrimination, multiplying the negative impacts of PRD. By contrast, individuals living in higher SES environments may be exposed to more or differing kinds of racial discrimination, due to exposure to more multiracial contexts (Harrell, 2000). Research should therefore both covary the effects of SES and consider interactions between SES and PRD when considering its relationships with stress biology and health outcomes (Harrell, 2000; Myers, 2009).

1.7. Current research

We examined relations between histories of PRD reported from adolescence through young adulthood and diurnal cortisol rhythms measured in adulthood. We accomplished this by adding diurnal cortisol measures to the Maryland Adolescent Development in Context Study (MADICS) (Eccles et al., 1997; Wong et al., 2003; Eccles et al., 2006). MADICS is a longitudinal study in which PRD was measured over a 20-year period from early adolescence through approximately age 32. In addition to examining average/cumulative measures of PRD over a 20-year period, we examined whether PRD in two different time periods – adolescence and young adulthood – differentially predicted adult diurnal cortisol profiles. We also examined whether associations between perceived discrimination and adult stress biology differed for self-identified Blacks and Whites, and whether effects were moderated by socioeconomic status. Finally, we examined whether racial disparities existed in diurnal cortisol rhythms, and whether histories of racial discrimination from adolescence through adulthood helped to explain these disparities.

2. Method

2.1. Study overview

Participants and data were drawn from the MADICS Study, a longitudinal study of 1482 adolescents ($n = 879$ Black, 49% women) from Prince Georges County, Maryland (Eccles et al., 1997, 2006; Wong et al., 2003). Participants were recruited in 7th grade, at age 12, and followed for 20 years, through approximately age 32. There were eight waves of data collection across the follow-up period, including assessments in the 7th grade (Waves (W) 1 and 2), 8th grade (W3), 11th grade (W4), 1 year after high school (W5), 3 years after high school (W6), approximately age 30 (W7), and approximately age 32 (W8) (Brodish et al., 2011; Fuller-Rowell et al., 2012a).

At W8, a subset of participants, selected based on past histories of discrimination were invited to enroll in an add-on study in which biomarkers of stress and health were assessed. Participants in the add-on study completed a variety of measures, including a 7-day cortisol data collection protocol. The current study examines the relation between PRD, as reported across waves, and individuals' W8 diurnal cortisol profiles. All procedures were carried out with the adequate understanding and written consent of the participants, and were approved by Institutional Review Boards at Northwestern University, the University of Michigan, Harvard University, and the University of California at San Francisco.

2.2. Participants

One hundred and twenty four participants were enrolled in the MADICS health study. Based on a variable reflecting cumulative history of discrimination across the first seven waves of the MADICS study (see Appendix A of Supplementary information for discrimination questions), we recruited approximately equal numbers of Blacks and Whites, and both males and females with low, medium, and high levels of perceived racial discrimination. Individuals were excluded from the study due to use of corticosteroid-based medication ($N = 2$) or illicit substance use ($N = 1$). Individual days of data from the week-long diary study were excluded if that day was missing a morning or an evening cortisol sample, if it had a wake time before 0400 h or after 1400 h, if the individual slept less than four or more than twelve hours the prior night, or if the individual stayed awake for more than 20 h. One participant was excluded for not having any valid days of data. Eight additional participants were excluded for having extensive missing data on perceived discrimination: either more than 50% of their items missing or fewer than 2 waves of data available. Ultimately, our sample included 112 individuals: 32 Black females, 36 White females, 18 Black males, and 26 White males across low, medium and high PRD groups.

2.3. Demographic data

Most of the demographic data were taken from the W1 MADICS youth and parent surveys. Race and ethnicity were assessed by having participants self-report whether they identified as Black, White, Asian, Latino, or other. Parent education level at W1 was reported on a scale ranging from "Less than High School" to "College or More" (see Table 1). The highest of mother's and father's education levels

Table 1
Descriptive information on full study sample, Black sample, and White sample, including comparisons of Black and White samples.

	Full sample		Black sample		White sample		Comparison	
	Mean (or %)	SD		SD	Mean (or %)	SD	*t*	*p*
Adolescent control variables								
Female (percent)	60.714	49.058	64.000	48.487	58.065	49.748	−0.635	0.527
W1 income (in 000's of $)	51.205	19.693	46.600	22.923	54.919	2.017	2.263	0.026
Parent education (percentages)[a]								
Less than HS	21.429	41.279	35.135	48.398	10.638	10.638	−2.810	0.006
HS diploma	23.810	42.848	35.135	48.397	14.894	35.987	−2.199	0.031
Some college	27.381	44.859	16.216	16.216	36.170	48.569	2.063	0.042
College or more	27.381	44.859	13.514	34.658	38.298	49.137	2.600	0.011
Proportion of friends that are Black*[b]	3.080	1.331	4.075	0.162	2.234	0.133	−8.866	0.000
Proportion of friends that are White*[c]	2.874	1.292	1.775	0.110	3.809	0.128	11.832	0.000
Neighborhood school quality* (scale 1–4)[d]	2.965	0.832	3.150	0.700	2.804	0.910	−1.952	0.054
Neighborhood safety* (scale 1–3)[e]	2.632	0.508	2.525	0.554	2.723	0.452	1.839	0.069
Percent Black HH in neighborhood*[f]	39.171	30.358	60.495	26.916	20.561	2.535	−8.815	0.000
Percent Black students in school at W3*[g]	65.782	18.718	76.612	14.704	55.830	16.447	−5.594	0.000
Concurrent control variables								
Age	32.358	0.440	32.349	0.406	32.365	0.469	0.198	0.844
W8 income (in 000's of $)	76.336	45.627	77.164	45.162	75.669	46.355	−0.172	0.864
Smoker (percent)	20.536	40.578	22.000	41.845	19.355	39.830	−0.342	0.733
Birth control use (percent)	11.607	32.175	12.000	32.826	11.290	31.906	−0.116	0.908
Sleep & cortisol variables								
Waking cortisol level (μg/dl)	0.257	0.109	0.236	0.107	0.274	0.108	1.840	0.068
Wake + 30 cortisol (μg/dl)	0.363	0.129	0.347	0.151	0.376	0.108	1.181	0.240
Bedtime cortisol (μg/dl)	0.066	0.072	0.085	0.094	0.050	0.043	−2.596	0.011
Wake time (in decimal-hours)	7.324	1.337	7.637	1.521	7.072	1.119	−2.265	0.026
Total hours of sleep	6.282	1.110	6.200	1.192	6.348	1.045	0.698	0.487
Time between first and second samples	0.578	0.310	0.633	0.458	0.534	0.050	−1.684	0.095
Time between first and third samples	15.937	1.163	16.062	1.398	15.837	0.932	−1.018	0.311
Depression								
Cumulative depression (W3–7)	0.000	1.000	−0.028	1.063	0.022	0.954	0.262	0.794
Adolescent depression (W3–4)[h]	0.000	1.000	0.023	1.074	−0.019	0.943	−0.209	0.835
Young adult depression (W5–7)	0.000	1.000	−0.033	1.061	0.026	0.956	0.310	0.757
PRD		1.000						
Cumulative PRD (W3–7)	0.000	1.000	0.468	1.104	−0.377	0.719	−4.880	0.000
Adolescent PRD (W3–4)	0.000	1.000	0.215	1.140	−0.174	0.841	−2.078	0.040
Young adult PRD (W5–7)	0.000	1.000	0.553	1.060	−0.446	0.681	−6.042	0.000
N		112	50		62			

[a] Parent education- analytic sample *N* = 84 (37 Black, 47 White).
[b] Proportion of friends that are Black- analytic sample *N* = 87 (40 Black, 47 White). Variable refers to youth-report of how many of their friends that they spend most of their time with are Black (1 = none of them 2 = a few of them 3 = about half of them 4 = most of them 5 = all of them).
[c] Proportion of friends that are White- analytic sample *N* = 87 (40 Black, 47 White). Variable refers to youth-report of how many of their friends that they spend most of their time with are White (1 = none of them 2 = a few of them 3 = about half of them 4 = most of them 5 = all of them).
[d] Neighborhood school quality- analytic sample *N* = 86 (40 Black, 46 White). Variable refers to parent-report of neighborhood's school quality on a scale from 1 to 4 (1 = poor 2 = fair 3 = good 4 = excellent).
[e] Neighborhood safety- analytic sample *N* = 87 (40 Black, 47 White). Variable refers to parent-report of neighborhood safety, as compared with "other neighborhoods,ön a scale from 1 to 3 (1 = less safe than most 2 = about the same 3 = more safe than most).
[f] Percent Black households in a neighborhood- analytic sample *N* = 103 (48 Black, 55 White). Variable refers to the percentage of black households in each participants' neighborhood.
[g] Percent Black students in school- analytic sample *N* = 71 (34 Black, 37 White). Variable refers to the percentage of black students within each school.
[h] Adolescent depression (W3–4)- analytic sample *N* = 99 (45 Black, 54 White).

was used (Adam et al., 2011). Parents self-reported on the total family income level at W1 on a scale ranging from 1 (Less than $5,000) to 16 (More than $75,000). Income at W8 (simultaneous with cortisol measurement) was self-reported on a scale in $5,000 increments, ranging from "less than $5,000" to "more than $200,000". The mean of the selected income category was used to examine family income in dollar units. Participant age at W8 was also self-reported.

2.4. Perceived racial discrimination (PRD)

Current PRD was assessed by youth self-report in Waves 3–7, using a variety of questions reflecting the extent to which individuals perceived unfair treatment due to race. Questions for W3 through W6 were designed by our team and have been used in prior publications (Wong et al., 2003). At W7, in addition to 4 questions designed by our team, 14 questions modified slightly from the Daily Life Experiences (Racial Hassles) scale of the Racism and Life Experience Scales (RaLES) were used (Harrell et al., 1997; Utsey, 1998). Questions varied over time to take into account the changing developmental stages and social contexts present at each wave. Various types of PRD were assessed, including interpersonal discrimination within the school setting (e.g., How often have you felt that teachers/counselors discourage you from taking certain classes because of your race?), racism-related daily hassles (e.g., how often do you feel that kids do not want to hang out with you because of your race?) and microaggressions (e.g., Because of your race, how often have other reacted to you as if they were afraid or intimidated?). See Appendix A of Supplementary information for the full set of PRD questions, and means and comparisons for each item by race/ethnicity.

PRD items were standardized and averaged within each wave, and then averaged together across waves to create three different standardized PRD history measures. First, a cumulative PRD history measure was created (W3 through W7; $\alpha = 0.92$ across all included items). Next, an adolescent PRD measure was created by averaging the scales assessed at W3 and W4 ($\alpha = 0.87$). Finally, a young adult PRD measure was created by averaging the scales assessed at W5–W7 ($\alpha = 0.94$). Individuals with missing items in a given wave

had the item replaced with the individual's average for that wave; the proportion of missing data replaced per wave ranged from 0 to 2% of items, given 98–100% item completion rates for waves in which individuals were present. For individuals with missing waves of data the mean of their available waves was utilized. The majority (74%) of the sample had least 4 out of 5 waves of data present: 52% had all 5 waves of data, 22% had 4 waves of data, 21% had 3 waves of data, and 7% had 2 waves of data. Adolescent and young adult PRD were only moderately correlated with one another ($r = 0.42$, $p = 0.000$), suggesting that they are related, but sufficiently distinct that they could make unique contributions to the prediction of adult cortisol.

2.5. Salivary cortisol

Saliva samples were gathered three times daily each day for one week: at waking, 30 min after waking, and at bedtime (Adam and Kumari, 2009). The passive drool technique was used, in which participants expelled unstimulated saliva through a small plastic straw into a 2 mL polypropylene vial. During a reminder call the evening prior to data collection, participants were instructed to place sampling materials by their bed and to take their first sample as soon as possible after opening their eyes. A kitchen timer preset to 30 min was provided to aid in the timing of the 2nd sample. Participants were instructed not to eat, drink, or brush their teeth during the 30 min prior to the sample collection times, and to record their exact times of collection on labels provided for each vial. They were asked to store samples in their refrigerators after collection, and to return samples to us by regular postal mail. Cortisol samples are stable in saliva at room temperature for several days and are not affected by a regular postal journey (Clements and Parker, 1998). Samples were stored at −20 °C, then shipped on dry ice to Trier, Germany, and were assayed in duplicate using time-resolved fluorescent-detection immunoassay (DELFIA; Dressendörfer et al., 1992). Intra-assay variation ranged from 4.0% to 6.7%, while inter-assay variation ranged from 7.1% to 9.0%. Cortisol values are reported in μg/dl units. Log-transformed cortisol values were used in analysis; descriptive statistics and figures are presented in raw units (μg/dl) for ease of interpretation. Objective monitoring of compliance was not available in this study. However, due to the importance of accurate sample timing for estimation of the CAR (Kudielka et al., 2003), 30-min post-awakening samples that were reported to be taken more than 10 min early or 10 min late were eliminated from analysis.

2.6. Health covariates

Health covariates known to affect concurrent cortisol levels were measured by questionnaire at W8, including time of waking on the days of cortisol testing, birth control use and smoking status. Depressive symptoms were also measured multiple times from Wave 3 through W7, using items from the Children's Depression Inventory (Kovacs, 1992). The number of items assessed at each Wave ranged from 6 to 26 items (alphas ranged from 0.82 to 0.87). Items were averaged and standardized within each wave, and then averaged together across waves to create measures of cumulative (Waves 3–7), adolescent (Waves 3 and 4), and young adult (waves 5–7) depressive symptoms.

2.7. Contextual variables

To better describe the contexts in which participants live and discrimination experiences were encountered, we assessed several aspects of social, school and neighborhood contexts at W3 and W4. Participants reported the proportions of their friends that were Black and White at W3. Parents reported the perceived quality of the schools in their neighborhood and the perceived safety of their neighborhood (W3). Census data was obtained at W3 regarding the percent of black households in participants' neighborhoods (W3). The percent of students in participants' schools who were Black was obtained from school administrative data at W4. See Table 1 for means by race/ethnicity.

2.8. Analysis

A 3-level multilevel model was run in HLM 7 in order to model each individual's diurnal cortisol levels across the day and to predict individual differences in the diurnal cortisol rhythm. This approach, which has been utilized and recommended in past diurnal cortisol research (Hruschka et al., 2005; Adam, 2006), models the non-independence associated with the nested data structure (Raudenbush and Bryk, 2002) and has the ability to model the diurnal rhythm of cortisol while adding in moment-level (Level 1), day-level (Level 2), and person-level (Level 3) predictors. The general decline of cortisol levels across the day was modeled by regressing time of day of sampling (calculated as time since waking and entered at Level (1) on each individual's cortisol level (the dependent variable). A slowing of the decline was modeled by including quadratic time term (time since waking squared, entered at Level 1). Time was centered as hours since waking (e.g., waking time = 0), so that the intercept reflected the cortisol level at waking. To model the size of the CAR, a dummy variable was added at Level 1 (sample 2 = 1, all other samples = 0). At Level 2, time of waking and length of sleep for each individual, each day, were entered as day-level covariates. At Level 3, we entered PRD variables, race (0 = White, 1 = Black), SES variables, interactions between race and PRD, gender and PRD, and SES and PRD, and person-level control variables (e.g., gender, age, oral contraceptive use, average time of waking).

The race and gender dummy variables and the time variables were centered at their own zero points. Day-level variables were group-mean centered, and person-level variables were grand-mean centered. Analyses proceeded in the following order. First, we examined descriptive information on how our cortisol variables and covariates vary by race. Next, we analyzed how race, PRD, and our set of race, gender, and SES by PRD interactions related to morning cortisol (the intercept), the CAR, and the diurnal cortisol slope, controlling for our set of covariates. Covariates were only retained in the model if they showed significant associations with at least one of the cortisol outcomes, and interactions were retained only for cortisol outcomes for which they were significant.

In addition to focusing on waking levels, the size of the CAR, and slope, we calculated an area under the curve from the available data points each day in order to model the average elevation of the diurnal cortisol curve across the day (average or total cortisol levels). We then conducted a 2-level HLM model predicting total cortisol (AUC) from the cumulative, adolescent, and young adult PRD measures and covariates, from race, from race, gender and SES by PRD interactions, and our set of covariates.

Analyses are conducted first for average/cumulative PRD, followed by models with adolescent and young adult PRD serving as the predictors. Finally, to examine whether histories of PRD account for racial/ethnic disparities in cortisol rhythms, we compared the effects of race on cortisol diurnal rhythms in models without PRD variables, to the effects of race on cortisol diurnal rhythms in models including the PRD variables.

3. Results

3.1. Descriptive information

Descriptive information on levels of cortisol, PRD, covariates, and contextual variables for the full sample and for Black and

White participants are presented in Table 1. Independent sample *t*-tests revealed that Blacks had marginally lower waking cortisol (0.236 vs. 0.274 μg/dL) and significantly higher bedtime cortisol (0.085 vs. 0.050 μg/dL) than Whites. Racial differences were also apparent for time of waking; Black participants reported later waking times than White participants. Racial differences were observed in all of the PRD variables, and particularly in young adult PRD; Black participants reported 0.85 standard deviations (SDs) higher levels of cumulative PRD, 0.39 SD higher PRD in adolescence, and 1.00 SD higher levels of young adult PRD than White participants.

For the covariates, Blacks and Whites did not differ in levels of depression in adolescence or young adulthood, nor did they differ on smoking or birth control use. There were significant differences between Blacks and Whites on the demographic and contextual variables. Whites had significantly higher family income than Blacks at W1. These income differences were no longer present at Wave 8. Blacks came from families with significantly lower parent education at W1. Blacks reported significantly more friends that were Black and fewer friends that were White, and Whites reported significantly more friends that were White and fewer that were Black. There were non-significant trends for Blacks to report higher school quality and lower neighborhood safety than Whites. Blacks, as compared to Whites, lived in neighborhoods with a higher proportion of Black households (60% for Blacks vs. 21% for Whites), and in schools with a higher proportion of Black students (77% for Blacks vs. 56% for Whites). Thus, although racial/ethnic segregation in friendships, neighborhood and school contexts is apparent, there is still evidence of opportunity for interracial encounters between Blacks and Whites.

3.2. Preliminary analyses

Preliminary analyses revealed that the social, school and neighborhood context variables had limited effects on the cortisol outcomes, and associations between PRD and cortisol were not altered notably by their inclusion in the models. As a result, these variables are not considered further in our analyses. However, key demographic variables such as gender, family income at W1 and W8 and parent education at W1 were included. We tested interactions between gender and PRD in predicting cortisol; no significant effects were found. As a result, only main effects of gender are retained as covariates. Several non-significant covariates were removed from the model (i.e., hours of sleep, age, and oral contraceptive use). The remaining covariates (i.e., gender, W1 and W8 income, and both day-level and average wake times) were significant in at least one model and therefore retained in all HLM models. Depressive symptoms were included in the final models to demonstrate that associations between PRD and cortisol were not explained by the potentially confounding impact of depressive symptoms on PRD reporting and cortisol.

3.3. Covariates effects

There were relatively few significant effects of demographic and health covariates (see Table 2 for wake, CAR and slope results; AUC results are reported in text). Females had 2% flatter diurnal cortisol slopes than males ($p = 0.030$). They also had a significantly larger CAR. Day-specific waketimes predicted a smaller CAR ($p < 0.001$); CARs decreased by 10% for every hour later waketime. AUC cortisol was lower on days that individuals woke up later ($b = -3.54$, SE = 0.21, $p < 0.001$). Later average waketimes across the 7 days predicted flatter slopes; 1% flatter per hour later average waking. Finally, there was trend for higher baseline family income to predict lower AUC cortisol ($b = -0.09$, SE = 0.05, $p = 0.099$).

3.4. Race, cumulative discrimination and cortisol

Our key models examined race, cumulative PRD, and their interactions in predicting diurnal cortisol rhythms. Interactions between PRD and income and parent education are also examined.

3.4.1. Waking cortisol

As seen in Table 2, waking cortisol levels for Whites were on average 0.23 μg/dl. Blacks had, on average, 16% lower waking cortisol than Whites. There were no significant effects of cumulative PRD on waking cortisol for Whites. A significant race by cumulative PRD interaction revealed that for Blacks, waking cortisol decreased 17% for every 1 SD higher cumulative PRD from adolescence through young adulthood. There was also a significant PRD by parent education interaction, with cumulative PRD predicting a 10% greater drop in waking cortisol for each SD higher parent education.

3.4.2. Cortisol awakening response

There was a significant CAR present for both males and females; males' cortisol levels increased 54% from waking to 30 min after waking and females' cortisol levels increased 71%. There were no main effects of race on the CAR. There was however a significant effect of cumulative PRD on the CAR, with the CAR being 18% lower for every SD increase in cumulative PRD. There was also a significant income by cumulative PRD interaction: effects of PRD on the CAR were lessened by higher family income at W1, with the impact of cumulative PRD on the CAR being 4% less for every $10,000 increase in family income.

3.4.3. Diurnal cortisol slope

There was, as expected, a significant decline in cortisol levels across the day, with cortisol levels declining 26% per hour (at waking), and that decline decelerating (due to a significant quadratic term) 1% per hour starting at waking. There was a significant effect of cumulative PRD on diurnal cortisol slope, with cortisol slopes declining 1% per hour more slowly (being 1% flatter) for every SD increase in cumulative PRD from adolescence through young adulthood for both Blacks and Whites. The interaction between race and cumulative PRD was not significant in predicting diurnal cortisol slopes ($b = 0.012$, SE = 0.008, $p = 0.149$), but there was a significant PRD by parent education interaction, with the impact of PRD on cortisol slopes being 1% greater (1% flatter) for every SD increase in parent education.

3.4.4. Average cortisol across the day (AUC)

There were no significant differences between Blacks and Whites in average cortisol levels across the day ($b = 2.37$, SE = 2.02, $p = 0.24$), nor was there a significant effect of cumulative PRD on AUC cortisol ($b = 1.22$, SE = 2.69, $p = 0.65$) or a race by cumulative PRD interaction ($b = -2.70$, SE = 2.13, $p = 0.21$).

3.5. Developmental timing of discrimination effects

In order to consider whether the developmental timing of PRD exposure mattered, we examined whether adolescent PRD (W3 & W4) and young adult (W5–W7) PRD, as well as their interactions with race, income and parent education, were associated with diurnal cortisol rhythms (Table 3). Results for covariates are not described again as they were very similar to those in the cumulative PRD model.

3.5.1. Waking cortisol

When adolescent PRD and young adult PRD were entered separately in the model predicting waking cortisol, there were no main effects of PRD, but there were significant interactions with race. Adolescent PRD predicted lower waking cortisol levels for Blacks

Table 2
Multilevel model of the associations between cumulative perceived racial/ethnic discrimination and adult cortisol.

Fixed effect	Coefficient	SE	*t*	*P*	Interpretation
Model for waking cortisol level, π_0					
Average waking cortisol level, β_{00}					
Intercept, γ_{000}	−1.451	0.059	−24.520	<0.001	Waking level=0.23 μg/dl*
Female, γ_{001}	−0.011	0.067	−0.162	0.872	n.s.
Black, γ_{002}	−0.173	0.080	−2.158	0.033	−16% for Black respondents**
W1 income, γ_{003}	−0.004	0.002	−1.928	0.057	n.s.
W8 income, γ_{004}	0.001	0.001	0.968	0.335	n.s.
Wake, γ_{005}	−0.012	0.033	−0.367	0.714	n.s.
Parent education, γ_{006}	−0.041	0.051	−0.797	0.427	n.s.
Cumulative depression, γ_{007}	−0.007	0.033	−0.195	0.846	n.s.
Cumulative PRD, γ_{008}	0.002	0.046	0.033	0.974	n.s.
Blackcumulative PRD, γ_{009}	−0.185	0.067	−2.783	0.006	−17% for every +1SD for Black respondents
Parent education × cumulative PRD, γ_{0010}	−0.100	0.048	−2.089	0.039	−10% for every +1SD in parent education
Wakeup time, β_{01}					
Intercept, γ_{010}	−0.008	0.025	−0.313	0.754	n.s.
Model for cortisol awakening response, π_1					
Average cortisol awakening response, β_{10}					
Intercept, γ_{100}	0.429	0.054	7.946	<0.001	+54% CAR for males
Female, γ_{101}	0.160	0.059	2.728	0.008	+17% larger CAR for females
Black, γ_{102}	0.019	0.069	0.273	0.785	n.s.
W1 income, γ_{103}	0.000	0.001	−0.130	0.897	n.s.
W8 income, γ_{104}	0.001	0.001	0.971	0.334	n.s.
Wake, γ_{105}	0.014	0.024	0.585	0.560	n.s.
Parent education, γ_{106}	0.034	0.036	0.948	0.345	n.s.
Cumulative depression, γ_{107}	−0.012	0.031	−0.397	0.692	n.s.
Cumulative PRD, γ_{108}	−0.197	0.073	−2.684	0.008	−18% for every +1SD
Black × cumulative PRD, γ_{109}	0.073	0.060	1.224	0.224	n.s.
W1 income × cumulative PRD, γ_{1010}	0.003	0.001	3.002	0.003	+4% for every $10,000 in parental income
Wakeup time, β_{11}					
Intercept, γ_{110}	−0.109	0.022	−4.973	<0.001	−10% for every hour later waking
Model for time since waking, π_2					
Average effect of time since waking, β_{20}					
Intercept, γ_{200}	−0.296	0.029	−10.381	<0.001	−26% per hour at waking
Female, γ_{201}	0.019	0.008	2.202	0.030	+2% flatter for females
Black, γ_{202}	0.014	0.010	1.360	0.177	n.s.
W1 income, γ_{203}	0.000	0.000	−0.237	0.813	n.s.
W8 income, γ_{204}	0.000	0.000	−0.486	0.628	n.s.
Wake, γ_{205}	0.010	0.004	2.293	0.024	1% flatter per hour later average waking
Parent education, γ_{206}	0.004	0.005	0.831	0.408	n.s.
Cumulative depression, γ_{207}	0.001	0.005	0.248	0.804	n.s.
Cumulative PRD, γ_{208}	0.012	0.004	2.686	0.008	1% flatter per +1SD
Parent education × cumulative PRD, γ_{209}	0.012	0.004	2.674	0.009	+1% for every +1SD in parent education
Wakeup time, β_{21}					
Intercept, γ_{210}	0.000	0.003	0.165	0.869	n.s.
Model for time since waking squared, π_3					
Intercept, β_{30}					
Intercept, γ_{300}	0.011	0.002	6.280	<0.001	1% deceleration in slope per hour after waking

All level 1 predictors are uncentered; level 2 variables are group mean centered; income, average wake time, and PRD variables are grand mean centered in level 3 while Female and Black were uncentered. Day-level predictors of waking values, slopes, and CAR were fixed at levels 2 and 3, while all other coefficients are set as random. Random effects for the level 2 intercept was significant at the $p<0.05$ level [$r_0=0.132$, $\chi2(560)=643.779$, $p=0.008$]. Random effects for all of the four level 3 intercepts were significant at the $p<0.05$ in the full model [$u_{00}=0.297$, $\chi2(99)=341.570$, $p<0.001$; $u_{01}=0.173$, $\chi2(100)=253.781$, $p<0.001$; $u_{20}=0.144$, $\chi2(99)=150.519$, $p<0.001$; $u_{30}=0.010$, $\chi2(109)=239.375$, $p<0.001$].

* Because the outcome was log-transformed, the exponential function was applied to the coefficient to transform the units back to the original scale of measure.

** The following transformation was applied: $B_{\%change}=[\exp(B_{raw})]-1$

($b=-0.15$, SE = 0.056, $p=0.01$). There was a trend for young adult PRD to also predict lower waking cortisol ($b=-0.15$, SE = 0.079, $p=0.06$).

When adolescent and young adult PRD were entered simultaneously to examine their unique effects (Table 3), adolescent PRD continued to predict lower morning cortisol for Blacks ($b=-0.13$, SE = 0.060, $p=0.04$). Morning cortisol levels were 12% lower for every SD increase in adolescent PRD for Black participants. There were no significant unique effects of young adult PRD on waking cortisol. There was, however, a significant parent education by adolescent PRD interaction, with adolescent PRD being associated with a larger decline in waking cortisol at higher levels of parental education.

3.5.2. Cortisol awakening response

When adolescent and young adult PRD were entered separately, there was a trend for higher adolescent PRD to predict a lower cortisol awakening response ($b=-0.15$, SE = 0.079, $p=0.054$), with no significant race by adolescent PRD interaction ($b=-0.05$, SE = 0.06, $p=0.43$). Higher young adult PRD predicted a significantly greater CAR ($b=-0.23$, SE = 0.08, $p=0.003$), however there was also significant race by PRD interaction ($b=0.152$, SE = 0.06, $p=0.016$), with Whites with higher young adult PRD having a lower CAR, but Blacks with high young adult PRD having a significantly larger CAR.

When both adolescent and young adult PRD were entered simultaneously in the model (Table 3), there was a trend for young adult PRD to be associated with a lower CAR for Whites ($b=-0.171$,

Table 3
Multilevel model of the associations between adolescent and young adult PRD and adult cortisol patterns.

Fixed effect	Coefficient	SE	*t*	*P*	Interpretation
Model for waking cortisol level, π_0					
Average waking cortisol level, β_{00}					
Intercept, γ_{000}	−1.455	0.061	−23.758	<0.001	Waking level = 0.23 μg/dl*
Female, γ_{001}	0.001	0.066	0.022	0.982	n.s.
Black, γ_{002}	−0.179	0.088	−2.040	0.044	−16% for Black respondents**
W1 income, γ_{003}	−0.004	0.002	−1.840	0.069	−4% for every $10,000 in family income
W8 income, γ_{004}	0.001	0.001	0.744	0.459	n.s.
Wake, γ_{005}	−0.002	0.034	−0.063	0.950	n.s.
Parent education, γ_{006}	−0.032	−0.049	0.639	0.525	n.s.
Adolescent depresssion, γ_{007}	−0.005	0.051	−0.103	0.918	n.s.
Young adult depresssion, γ_{008}	−0.012	0.039	−0.323	0.748	n.s.
Adolescent PRD, γ_{009}	−0.048	0.049	−0.980	0.330	n.s.
Young adult PRD, γ_{0010}	0.034	0.052	0.649	0.518	n.s.
Black × adolescent PRD, γ_{0011}	−0.126	0.060	−2.111	0.037	−12% for every +1SD for Black respondents
Black × young adult PRD, γ_{0012}	−0.080	0.071	−1.129	0.262	n.s.
Parent education × adolescent PRD, γ_{0013}	−0.115	0.056	−2.064	0.042	−11% for every +1SD in parent education
Parent education × young adult PRD, γ_{0014}	0.030	0.036	0.843	0.401	n.s.
Wakeup time, β_{01}					
Intercept, γ_{010}	−0.008	0.025	−0.317	0.752	n.s.
Model for cortisol awakening response, π_1					
Average cortisol awakening response, β_{10}					
Intercept, γ_{100}	0.431	0.057	7.611	<0.001	+54% CAR for males
Female, γ_{101}	0.158	0.058	2.725	0.008	+17% larger CAR for female
Black, γ_{102}	0.006	0.069	0.087	0.931	n.s.
W1 income, γ_{103}	0.000	0.001	−0.222	0.825	n.s.
W8 income, γ_{104}	0.001	0.001	1.222	0.225	n.s.
Wake, γ_{105}	0.016	0.025	0.628	0.531	n.s.
Parent education, γ_{106}	0.050	0.035	1.446	0.151	n.s.
Adolescent depresssion, γ_{107}	−0.029	0.040	−0.718	0.475	n.s.
Young adult depresssion, γ_{108}	0.004	0.037	0.106	0.916	n.s.
Adolescent PRD, γ_{109}	−0.085	0.100	−0.855	0.395	n.s.
Young adult PRD, γ_{1010}	−0.171	0.091	−1.885	0.062	−16% for every +1SD
Black × adolescent PRD, γ_{1011}	−0.099	0.073	−1.355	0.179	n.s.
Black × young adult PRD, γ_{1012}	0.174	0.065	2.685	0.009	+19% for every +1SD for Black respondents
W1 income × adolescent PRD, γ_{1013}	0.002	0.002	1.498	0.137	n.s.
W1 income × young adult PRD, γ_{1014}	0.002	0.001	1.722	0.088	+2% for every $10,000 in parental income
Wakeup time, β_{11}					
Intercept, γ_{110}	−0.109	0.022	−4.936	<0.001	−10% for every hour later waking
Model for time since waking, π_2					
Average effect of time since waking, β_{20}					
Intercept, γ_{200}	−0.294	0.029	−10.257	<0.001	−25% for every hour at midday
Female, γ_{201}	0.014	0.008	1.696	0.093	+1% flatter for females
Black, γ_{202}	0.016	0.011	1.485	0.141	n.s.
W1 income, γ_{203}	0.000	0.000	−0.636	0.526	n.s.
W8 income, γ_{204}	0.000	0.000	−0.272	0.786	n.s.
Wake, γ_{205}	0.009	0.004	2.056	0.042	1% flatter per hour later average waking
Parent education, γ_{206}	0.004	0.005	0.776	0.439	n.s.
Adolescent depresssion, γ_{207}	−0.006	0.006	−1.114	0.268	n.s.
Young adult depresssion, γ_{208}	0.006	0.006	0.984	0.328	n.s.
Adolescent PRD, γ_{209}	0.008	0.003	2.437	0.017	1% flatter per +1SD
Young adult PRD, γ_{2010}	0.007	0.005	1.521	0.131	n.s.
Wakeup time, β_{21}					
Intercept, γ_{210}	0.000	0.003	0.145	0.885	n.s.
Model for time since waking squared, π_3					
Intercept, β_{30}					
Intercept, γ_{300}	0.011	0.002	6.177	<0.001	1% deceleration in slope per hour after waking

All level 1 predictors are uncentered; level 2 variables are group mean centered; Income, average wake time, and PRD variables are grand mean centered in level 3 while Female and Black were uncentered. Day-level predictors of waking values, slopes, and CAR were fixed at levels 2 and 3, while all other coefficients are set as random. Random effects for the level 2 intercept was significant at the $p < 0.05$ level [$r_0 = 0.132$, $\chi2(560) = 648.172$, $p = 0.006$]. Random effects for all of the four level 3 intercepts were significant at the $p < 0.05$ in the full model [$u_{00} = 0.294$, $\chi2(95) = 339.849$, $p < 0.001$; $u_{01} = 0.174$, $\chi2(99) = 253.230$, $p < 0.001$; $u_{20} = 0.157$, $\chi2(95) = 151.723$, $p < 0.001$; $u_{30} = 0.010$, $\chi2(109) = 240.844$, $p < 0.001$].

* Because the outcome was log-transformed, the exponential function was applied to the coefficient to transform the units back to the original scale of measure.

** The following transformation was applied: $B_{\%change} = [\exp(B_{raw})] - 1$.

SE = 0.09, $p = 0.062$), and a significant Black by young adult PRD interaction ($b = .174$, SE = .07, $p = .009$), with Blacks showing a 19% larger CAR for every SD increase in PRD (see Table 3). There was a trend for higher income to be associated with greater increases in the CAR per unit of young adult PRD ($b = .002$, SE = .001, $p = .088$).

3.5.3. Diurnal cortisol slope

When entered separately, both adolescent and young adult PRD showed significant main effects on diurnal cortisol slopes (PRD, $b = .010$, SE = .004, $p = .011$ and $b = .010$, SE = .005, $p = .043$, respectively), with higher PRD during both age periods predicting flatter diurnal cortisol slopes. When entered simultaneously (Table 3), there was a significant effect of adolescent PRD on flatter slopes ($b = .008$, SE = .003, $p = .017$), however young adult PRD was no longer significant ($b = .007$, SE = .005, $p = .131$). There were no significant race by PRD interactions for either adolescent or young adult PRD ($b = 0.008$, SE = 0.007, $p = .259$; $b = .004$, SE = .10, $p = .663$).

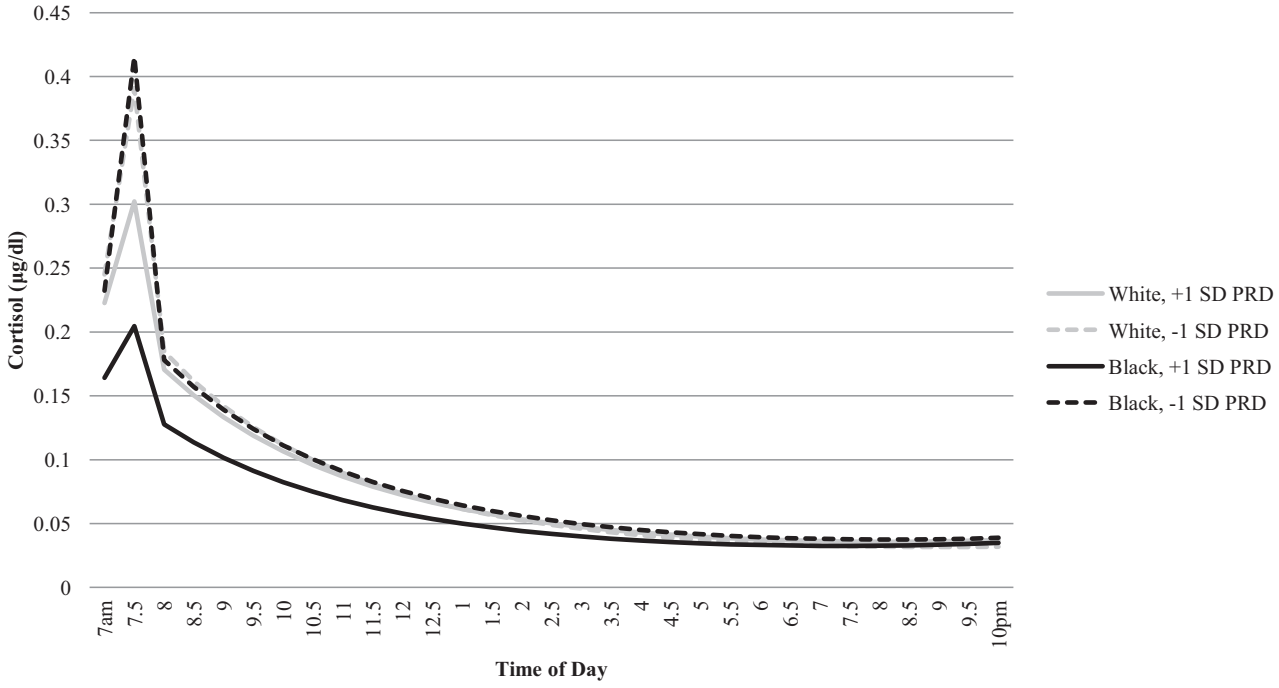

Fig. 1. Adolescent perceived racial/ethnic discrimination and adult cortisol rhythms across the day by race/ethnicity.

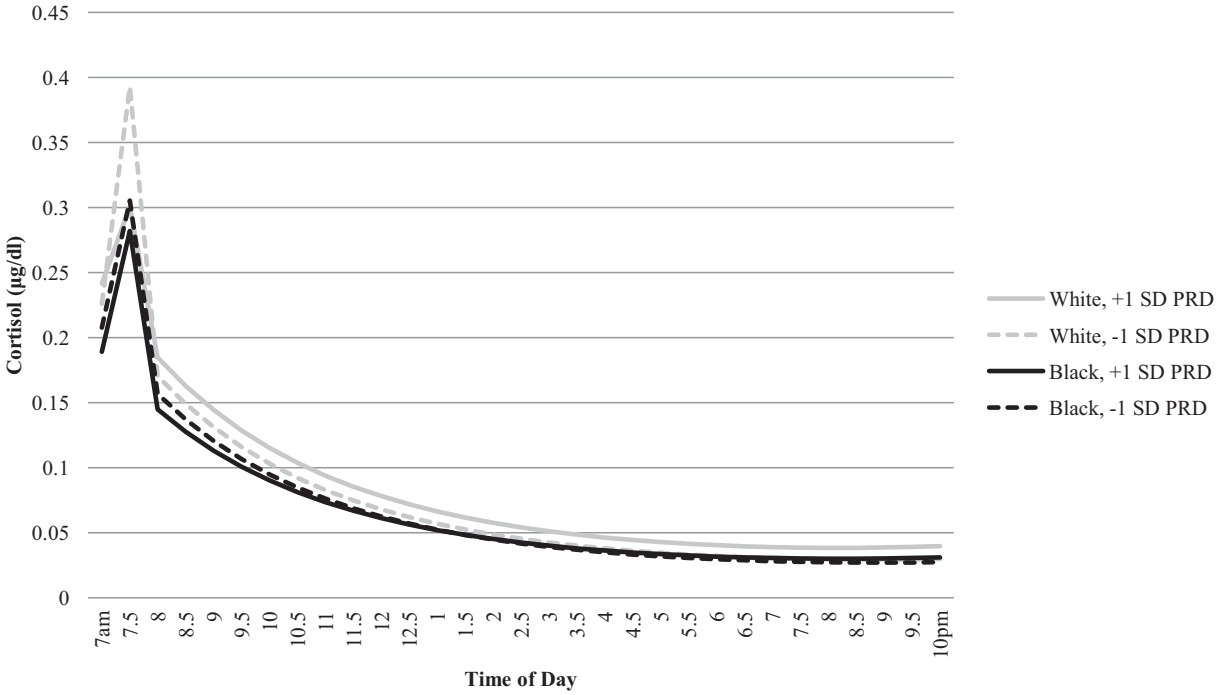

Fig. 2. Young adult perceived racial/ethnic discrimination and adult cortisol rhythms across the day, by race/ethnicity.

3.5.4. Average cortisol across the day (AUC)

When adolescent and young adult PRD were entered in the model separately, adolescent PRD was associated with lower average cortisol across the day for Blacks ($b = -3.71$, SE = 1.55, $p = .019$), but not for Whites ($b = .72$, SE = 2.29, $p = .75$). Young adult PRD ($b = .73$, SE = 2.92, $p = .802$) and its interaction with race ($b = -.94$, SE = 2.28, $p = .681$) were not significant predictors of AUC cortisol.

When adolescent and young adult PRD were entered simultaneously, there was again a significant race by adolescent PRD interaction, with adolescent PRD ($b = -4.12$, SE = 1.64, $p = .014$), but not young adult PRD ($b = .992$, SE = 2.29, $p = .667$) predicting lower average cortisol levels for Blacks. The lower AUC for Blacks with high adolescent PRD is depicted in Fig. 1; the non-significant association between young adult PRD and AUC cortisol is seen in Fig. 2.

3.6. Does discrimination account for racial/ethnic disparities in diurnal cortisol rhythms?

In a reduced model examining associations between race and cortisol diurnal rhythms without any discrimination variables,

strong effects of race were evident for waking cortisol and the diurnal cortisol slope, but not the CAR or AUC. Specifically, Black respondents' waking cortisol levels were 25% lower ($b = -.285$, SE = .09, $p = .002$) and their diurnal cortisol slopes were 3% flatter per hour compared to White participants ($b = .026$, SE = .010, $p = .009$). In models that included cumulative PRD, the size of these effects were reduced slightly, to Blacks having 16% lower waking cortisol levels ($b = -.173$, SE = .08, $p = .033$) and 1.5% flatter slopes, with the main effect of race on cortisol slope no longer being significant ($b = .019$, SE = .069, $p = .785$). Slightly stronger reductions in effect size were found for the model that included adolescent and young adult PRD, with the association between race and morning cortisol being reduced to a trend level, and associations between race and diurnal cortisol slope no longer being significant (see Table 3).

4. Discussion

Our results suggest that greater developmental histories of PRD are related to a pattern of flatter diurnal cortisol slopes, lower waking cortisol levels, and lower average cortisol across the waking day. The pattern of flatter slopes with high PRD was present for both Blacks and Whites, whereas the lowering of waking and average cortisol levels was specific to Blacks reporting high PRD. These results are in accord with past studies that have found higher PRD to be associated with flatter diurnal cortisol slopes (Suglia et al., 2010; Skinner et al., 2011 Zeiders et al., 2014) and lower average cortisol (Kaholokula et al., 2012). They contrast somewhat with one study that found flatter diurnal cortisol slopes for Whites but steeper slopes for Blacks (Fuller-Rowell et al., 2012b). Overall, the bulk of the evidence, including the current study, suggests a pattern of flatter diurnal cortisol slopes and lower average cortisol with higher PRD.

A pattern of lower average cortisol across the day is an indicator of hypcortisolism—a pattern of low and less dynamic cortisol levels that is thought to result from past chronic stress or traumatic stress, and is associated with negative health outcomes (Heim et al., 2000; Fries et al., 2005). It has been suggested that patterns of low or attenuated cortisol levels may emerge over time after a period of over-activation of the HPA axis due to extreme or chronic stress experiences (Miller et al., 2007). Susman et al. have referred to this as the attenuation hypothesis (Susman, 2006; Trickett et al., 2010).

Most of the tendency towards hypocortisolism appears to be driven by experiences of discrimination occurring during adolescence, more so than young adulthood. When entered simultaneously with young adult PRD levels, only adolescent PRD significantly predicted lower waking cortisol, a flatter diurnal cortisol slope and lower AUC cortisol. Adolescence is a developmental period in which the ability to perceive and understand experiences of discrimination increases (Brown & Bigler, 2005; Eccles et al., 2006; Wong et al., 2003; Umaña-Taylor et al., 2014). For Black adolescents, this developing awareness of both current and historical racial discrimination could make experiences of discrimination during adolescence particularly impactful. In addition, the protective mechanisms (such as socialization of a strong/racial ethnic identity) that have been shown to buffer youth from the negative impact of discrimination are likely to be less well established in adolescence (Boykin and Toms, 1985; Coard and Sellers, 2005; Eccles et al., 2006; Neblett et al., 2012). Rapid changes in social, emotional, identity and biological processes are also occurring during the adolescent time period. Consistent with the notion of adolescence as a sensitive period (Halfon and Hochstein, 2002), racial discrimination experiences during adolescence may become embedded in developing identities, emotion and coping processes, and neurobiology of racial/ethnic minority youth. Finally, given that the discrimination questions used in the current study changed with development, it is possible that the questions asked during adolescence captured more physiologically activating types of discrimination experiences.

Results for the cortisol awakening response were complicated, and were the exception to the rule of greater impacts of adolescent as compared to young adult PRD. Higher cumulative discrimination predicted a lower CAR, on average. When adolescent and young adult PRD were examined, however, PRD experiences in young adulthood were stronger predictors of the CAR than adolescent PRD. In addition, the direction of effect was opposite for Blacks and Whites. For Whites, higher young adult PRD marginally predicted a lower CAR, whereas for Blacks, higher young adult PRD predicted a significantly larger CAR. The CAR is hypothesized to increase in response to anticipated daily challenges, and to serve as a preparatory response for coping with the demands of the day (Adam et al., 2006; Fries et al., 2009). Perhaps Blacks who are reporting PRD experiences in young adulthood are actively mobilizing to cope with the anticipated discriminatory experiences.

Do the observed HPA axis changes found to be associated with PRD matter for health or developmental wellbeing? Both an overall lowering of cortisol and a flattening of the diurnal slope have been found to have important health implications. Flatter diurnal cortisol slopes have been linked to a wide range of negative health outcomes, including increased risk for cardiovascular disease, metabolic disorders, and mortality (Sephton et al., 2000; Steptoe et al., 2004; Kumari et al., 2009). An overall lowering of the diurnal cortisol curve (hypocortisolemic pattern) has further been linked with fibromyalgia, higher fatigue and chronic fatigue syndrome, autoimmune disorders, and PTSD (Crofford et al., 1994; Yehuda et al., 1996; Heim et al., 2000; Fries et al., 2005). The higher CAR found among Blacks with high young adult PRD may also have mental health relevance. Past evidence has linked an elevated CAR to the onset of depression and anxiety disorders in adolescents and young adults (Adam et al., 2010, 2014; Vrshek-Schallhorn et al., 2013).

There are numerous strengths to the current study, the largest of which is our use of 20 years of prospective longitudinal data to measure histories of discrimination. We also collected 7 days of cortisol data to measure diurnal cortisol rhythms, and employed sophisticated analytic techniques controlling for multiple demographic and health covariates, including both baseline and follow-up socioeconomic characteristics. In line with recent models that emphasize the cumulative impact of multiple stressors, including both socioeconomic, and race-related stressors (Myers, 2009; Brondolo, 2015), we also tested interactions between PRD and socioeconomic variables.

Our covariate effects were consistent with prior research showing that waketimes have a strong impact on cortisol rhythms, with later waking predicting a smaller CAR and AUC and flatter slope (Federenko et al., 2004; Zeiders et al., 2012). We also replicated prior research showing a slightly larger CAR for females as compared to males (Fries et al., 2009). More novel are findings of females having flatter slopes; this latter finding should be replicated in future research.

Our PRD-cortisol findings were not accounted for histories of depression occurring at the same time as the PRD measurement, despite prior research showing associations between depression and diurnal cortisol (Doane et al., 2013). We did not, however, assess developmental histories of trauma and PTSD symptoms. Given that individuals with PTSD show a pattern of hypocortisolism similar to that seen in the current study (Rohleder et al., 2004; Wessa et al., 2006), the possible role of PTSD symptoms in explaining associations between PRD and cortisol diurnal rhythms should be assessed in future research.

Our findings for socioeconomic variables were mixed. Higher parent education was associated with a greater lower of waking

cortisol with higher cumulative and adolescent PRD, and a stronger flattening of the diurnal cortisol slope with higher cumulative PRD. The findings for education suggest that, if anything, there may be greater impacts of PRD on cortisol at higher levels of education. One possibility is that individuals with higher education live and work in contexts in which they are exposed to greater PRD, particularly microaggressions. For income, there was a trend for higher baseline family income to predict lower AUC cortisol. Higher baseline income was also associated with a significant reduction in the effect of cumulative PRD on a lower CAR. Thus, there is some sign of income conferring some protection against the impact of PRD on cortisol. However, these findings should be replicated, and additional research should explore differences in the amount, type, and impact of PRD encountered at different levels of income and education.

There are a number of limitations to the current study. First, although we included 20 years of prospective longitudinal data in perceived discrimination, only one wave of cortisol was available in adulthood. Thus, we were unable to control for individuals' prior diurnal cortisol patterns. As a result, we were unable to assess changes in cortisol over time. Second, we did not have information on even earlier PRD experiences – those occurring during early childhood or the prenatal period (maternal PRD exposure). These earlier PRD exposures may influence children's developing HPA axes (Thayer and Kuzawa, 2015), and should be considered in future research. However, it is worth noting that the one study that has examined PRD and cortisol in preadolescence did not find associations (Martin et al., 2012). An increasing awareness of PRD during the adolescent period, paired with greater emotional and biological reactivity (Stroud et al., 2009) may help potentiate larger effects of PRD during the adolescent time period. Third, our sample is non-representative and geographically constrained; as such, it is not clear whether results will generalize to other geographic areas or populations. Fourth, although we examined some contextual factors that may interact with PRD exposures to affect outcomes (Myers, 2009; Brondolo, 2015), future research should examine additional contextual factors such as non-racial stressors from employment and family domains. Fifth, there were limitations to our cortisol measurement. We did not use objective measures of compliance with the requested timing of cortisol samples, and our cortisol measurement involved a fairly minimal protocol, in terms of number of samples per day. However, similar protocols have been widely used in past research (Adam et al., 2006; Adam and Kumari, 2009) and our measurement of cortisol over a full 7 days helps to increase the reliability of the cortisol measures (Hellhammer et al., 2007).

Sixth, our measures of discrimination were for the most part, designed for our study, although a standardized measure was introduced in more recent assessments. Missing from our measure were questions on targeting of and discrimination against Black youth by police, which could be a major source of stress. Also, although our PRD measures contained a mixture of items that assess overt and more subtle discrimination, they were not systematically designed to assess subtypes of discrimination. Future research should more systematically assess, and clearly differentiate between subtypes of PRD experiences, and should also examine how multiple subtypes of PRD might interact to affect outcomes. Nonetheless, the fact that our incomplete measure of discrimination still showed significant effects suggests that the impacts of discrimination on cortisol are notable; even larger effects might have been found with a more comprehensive measure.

Finally, absent from the current analysis is a detailed examination protective factors that may buffer youth, and particularly minority youth, from the negative impact of race-based social stress. These may include race-based protective factors, such as strong racial socialization and a strong racial–ethnic identity, as well as more general protective factors such as strong social support (Neblett et al., 2008, 2012; Brondolo et al., 2009). An important next step in research will be to identify resilience-promoting factors that may prevent or lessen the impact of PRD on stress, stress biology, and stress-related health and behavioral outcomes.

Despite these limitations, this study is groundbreaking in that it is the first prospective study to examine developmental histories of PRD (over a 20 year period) in relation to cortisol profiles in adulthood. Past studies examining the effects of PRD on cortisol have focused on concurrent experiences of discrimination, thus missing the effects of a potential accumulation of race-related stress over time on cortisol levels, and being unable to test the effects of particular developmental timings of PRD exposure. We found that high cumulative PRD significantly predicted flatter diurnal cortisol slopes relative to those with lower PRD. Our PRD measures accounted for much of the Black–White difference in the slope of diurnal cortisol rhythm that was present prior to entering the PRD variables in the model, suggesting that cumulative histories of discrimination may be an important factor accounting for racial/ethnic differences in cortisol slopes found in other studies. We find that PRD during both adolescence and young adulthood are associated with adult cortisol functioning. Adolescent PRD had more pervasive effects, however, particularly for Blacks, contributing to a hypocortisolemic pattern of lower average cortisol that has been associated with numerous health risks. The extent to which PRD contributes to racial/ethnic disparities in mental health, physical health, and academic attainment by way of altered HPA axis functioning, remains to be established in future work.

Contributors

EKA conceived of and designed the study, oversaw the acquisition of data and data analysis, and took the lead on the first draft and revisions of the paper. JAH ran the analysis, helped write the results section, prepared the tables and graphs and commented on multiple drafts of this paper. KHZ wrote part of the introduction, contributed to data analysis and commented on and edited drafts of this paper. JAR contributed to study design and commented on drafts of this paper. ECR oversaw data collection, helped with table preparation, and commented on drafts of this paper. KBE contributed to data collection and data analysis and commented on drafts of this paper. DJL assisted with data analysis, helped with table preparation, and commented on drafts of this paper. MK contributed to study design, helped to oversee data collection, and commented on drafts of this paper. ABB contributed to study design, helped to oversee data collection, and commented on drafts of this paper.OM contributed to study design, helped to oversee data collection, and commented on drafts of this paper. SP contributed to study design, helped to oversee data collection, and commented on drafts of this paper. TEF contributed to study design, helped to oversee data collection and commented on drafts of this paper. JSE oversaw the design of this study, helped to oversee data collection and commented on drafts of this paper. All authors approved the final submitted version of the manuscript.

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Conflicts of interest

None.

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The first 6 waves of data are archived at the Henry A. Murray Research Archive at Harvard University. <http://www.murray.harvard.edu/contact-us/>.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.psyneuen.2015.08.018.

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