



**Developmental Histories of Perceived Racial/Ethnic  
Discrimination and Diurnal Cortisol Profiles in Adulthood: A  
20-year Prospective Study**

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## Abstract

Perceived racial/ethnic discrimination (PRD) has been found to predict alterations in cortisol diurnal rhythms in past research, but most research has focused on current perceptions of discrimination. The researchers investigate whether developmental histories of PRD matter for adult diurnal cortisol profiles. One-hundred and twenty ( $N=57$  black,  $N=63$  white) adults ( $M_{age} = 32.36$  years;  $SD = .43$ ) from the Maryland Adolescent Development in Context Study (MADICS) provided saliva samples at waking, 30 minutes after waking, and at bedtime each day for 7 days. Adult diurnal cortisol measures were predicted from measures of PRD obtained over a 20-year period beginning when youth were in 7<sup>th</sup> grade (approximately age 12). Specifically, greater average PRD across the 20-year period predicted flatter diurnal cortisol slopes for both black and white participants. For blacks only, greater average PRD predicted lower waking cortisol and lower total cortisol across the day, a profile considered indicative of chronic stress. The effects of PRD on lower average cortisol across the day for blacks were driven by PRD experiences in adolescence. Young adult PRD, however, for blacks only, was associated with a larger cortisol awakening response. The results suggest that although PRD appears to impact cortisol for both blacks and whites, effects are stronger for black participants. In addition, adolescence may serve as a sensitive period for chronic impacts of PRD on adult stress biology.

## Introduction

Racial and ethnic disparities exist across a wide range of adult health conditions (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005; Williams & Collins, 1995; Williams & Mohammed, 2009). Differing health care access and racial/ethnic differences in health behavior do not appear to fully account for these disparities, leading investigators to propose the possibility that variations in race-based social stress, and the implications of that stress for stress biology, may play a role (Kuzawa & Sweet, 2009; Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009). One biological stress system frequently implicated in theoretical models of the impact of race-based stress on health is the hypothalamic-pituitary adrenal (HPA) axis (Myers, 2009), of which the primary hormonal product is cortisol. Racial/ethnic differences have been found in cortisol diurnal 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 (Fuller-Rowell, Doan, & Eccles, 2012; Kaholokula, Grandinetti, Keller, Nacapoy, & Mau, 2012; Zeiders, Doane, & Roosa, 2012).

Most existing research, however, has focused on current perceptions of discrimination. Although this research suggests that current perceived racial/ethnic discrimination (PRD) can have acute effects on stress biology, it is unclear whether early developmental experiences of PRD or chronic exposure to PRD predict more pronounced or more chronic alterations of stress biology. Research on developmental embedding has suggested that adverse experiences occurring during times of rapid developmental transition may have lasting impacts on adult stress biology (Hertzman & Boyce, 2010; Miller & Chen, 2013). We investigate whether developmental histories of PRD matter for

adult diurnal cortisol profiles, examining the impact of chronic or cumulative exposure to PRD over a 20 year period, as well as the impact of particular developmental timings (adolescence vs. young adulthood) of PRD experiences.

### *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 (Brown & Bigler, 2005; Fishbein, 1996; Major & Kaiser, 2008). From a general stress perspective (Compas, 1987) experiences of discrimination should be considered stressful, regardless of the group membership upon which that discrimination was based (e.g., gender, race, ethnicity, age). Experiences of discrimination are, however, more common for racial and ethnic minorities in the U.S. (Kessler, Mickelson, & Williams, 1999). In addition to discrimination being more common for minorities, historical mistreatment and oppression within U.S. society could make experiences of discrimination particularly relevant and impactful among racial and ethnic minority individuals (Branscombe, Schmitt, & Harvey, 1999; Feagin, Vera, & Batur, 2001), increasing the extent to which such experiences are embodied as alterations in stress physiology and negative health outcomes. Thus, for racial and ethnic minority individuals, such as African Americans, perceived discrimination may be both more frequent and more impactful than for their majority counterparts (Yetman, 1999).

### *Diurnal Cortisol Rhythms*

Cortisol, the end product of the HPA axis, is the most frequently measured HPA-axis marker in research on chronic stress and health, in part because the non-invasive nature of salivary cortisol collection allows repeated measurement of cortisol in the context of daily life (Adam, Sutton, Doane, & Mineka, 2008; Kirschbaum & Hellhammer, 2000). Cortisol

levels increase in response to certain types of stress, particularly stress of a social-evaluative nature (Dickerson & Kemeny, 2004), but also follow a strong circadian rhythm. The typical basal/diurnal cortisol rhythm involves high levels upon waking, a substantial (50-60%) increase in the 30-40 minutes after waking (the cortisol awakening response or CAR), and a subsequent decline over the remainder of the day, reaching a low point or nadir around midnight (Kirschbaum & Hellhammer, 1989; Pruessner et al., 1997; Weitzman et al., 1971).

Periodic activation of the HPA axis is considered adaptive and necessary to cope with acute stress. In acute stress situations, levels increase temporarily above the typical basal rhythm before returning to typical basal levels (Adam, 2006; Kirschbaum, Pirke, & Hellhammer, 1993). In addition, the cortisol awakening response has also been found to increase in the presence of acute anticipated or recent daily stressors (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Chida & Steptoe, 2009; Fries, Dettenborn, & Kirschbaum, 2008). In the face of repeated or chronic stress, however, chronic alterations in diurnal cortisol rhythms can be found. Changes in the rate of decline in cortisol from waking to bedtime (referred to as the diurnal cortisol slope; Adam and Kumari, 2009), represents an important indicator of stress-related alteration of the diurnal cortisol rhythm. Both acute and chronic stress exposures have been linked to flatter diurnal cortisol slopes (Adam, 2012; Miller et al., 2007; Miller, et al., 2002; Suglia et al. 2010) and flatter cortisol slopes have been linked to worse mental health (Havermans et al., 2011), higher fatigue (Bower et al. 2005), increased breast cancer mortality (Sephton et al., 2000), and cardiovascular disease (Matthews, Schwartz, Cohen, & Seeman, 2006). These findings are consistent with theoretical models suggesting that environmental factors contribute to alterations in the

diurnal functioning of the HPA axis, and that changes in diurnal cortisol slopes may be key mediators linking environmental stressors to health outcomes (e.g., Gunnar and Quevedo, 2007; Adam and Kumari, 2009).

In addition to flatter diurnal cortisol slopes, chronic stress is thought to contribute to changes in average cortisol levels across the day. Although acute stressors typically result in short-term elevations in cortisol, over a longer time frame, under chronic stress, levels can drop below normal, resulting in an overall lowering of cortisol levels, or hypocortisolism (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Heim, Ehlert, & Helhammer, 2000; Miller, Chen, & Zhou, 2007). An overall lowering of cortisol in response to chronic stress has also been termed “the attenuation hypothesis” (Susman, 2006). Hypocortisolism, in turn, has been found to matter for health, being associated with fatigue and pain syndromes, and overactivation of immune and inflammatory systems (Fries, Dettenborn, & Kirschbaum, 2009).

#### *Perceived Discrimination and Cortisol*

Past research has consistently found racial/ethnic differences in cortisol diurnal rhythms, and in particular, flatter diurnal cortisol rhythms in African Americans as compared to Whites (Cohen et al., 2006; DeSantis et al., 2008). Higher perceived discrimination has been proposed as a potential mediator between race/ethnicity and flatter diurnal cortisol slopes (DeSantis et al., 2007). Relatively few empirical studies, however, have examined the link between perceived discrimination and diurnal cortisol slopes. This is surprising given that stressors that are characterized as uncontrollable and socially evaluative, both of which apply to perceptions of discrimination, are some of the strongest activators of the HPA axis (Dickerson & Kemeny, 2004).

One study of young adults (Skinner, Shirtcliff, Haggerty, Coe, & Catalano, 2011) examined the relation between a variety of environmental stressors that included perceived discrimination and diurnal cortisol among White and Black youth and found that perceived discrimination related to flatter diurnal cortisol slopes in both groups. A study conducted in Dominica found that among women with a high level of internalized racism, perceived stress was related to flatter diurnal cortisol slopes, whereas among women with low levels of internalized racism, no relation was found between perceived stress and cortisol slopes (Tull, Sheu, Butler, & Cornelious, 2005). Another study of young adults found associations between discrimination and flatter diurnal cortisol slopes among racial/ethnic minority group members, but not racial/ethnic majority group members (Zeiders, Doane, & Adam, In press). In contrast, a study of adults found that discrimination predicted flatter diurnal cortisol slopes in White adults, but steeper diurnal cortisol slopes in Black adults (Fuller-Rowell et al., 2012). A recent 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). Taken together, this research, with some exceptions, suggests that perceived discrimination is associated with a flattening of the diurnal cortisol rhythm, and potentially an overall lowering of the diurnal cortisol curve across the day -- a pattern consistent with hypocortisolism.

### *Effects of Chronic Discrimination*

Prior research has focused on current or recent discrimination, rather than taking into account histories of exposure; hence, this work has not been able to assess the toll that chronic experiences of discrimination may take on an individuals' health. Moreover, prospective longitudinal studies of discrimination and cortisol diurnal rhythms are lacking.



Acute discrimination experiences may contribute to short-term activations of the HPA axis, but we argue that chronic histories of discrimination, in line with the work on hypocortisolism noted above, are likely to be associated with flatter diurnal cortisol rhythms and lower overall cortisol levels. We hypothesize that this pattern of response to chronic exposure to discrimination is especially likely to be found among racial/ethnic minority group members, due to their greater exposure to, and the greater psychological impact of, discrimination for these groups.

### *Developmental Timing*

In addition to a lack of focus on the chronicity of discrimination experiences, research on the developmental timing of discrimination experiences is also lacking. Developmental theory has long suggested that the developmental timing of experiences matters for long-term health and well-being, and that experiences occurring during times of rapid developmental transition are likely to have larger effects, as they become “built-in” to the changing biology or psychology of the individual. The biological “embedding” of early childhood experience has received considerable attention over the past few decades (Adam, 2012; Hertzman, 1999; Miller & Chen, 2013; Nelson, 2013; Shonkoff, Boyce, & McEwen, 2009). More recently, adolescence has been recognized as an additional critical period of neurobiological plasticity, given changes in brain development and the neuroendocrine system over this time period (Chambers, Taylor, & Potenza, 2003; Dahl, 2004; Spear, 2000). Adolescence is also a key period in the development of identity in general (Kroger, 2003; Meeus, Iedema, Helsen, & Vollebergh, 1999), and of racial/ethnic identity in particular (French, Seidman, Allen, & Aber, 2006; Phinney, 1989) As a result, we predict that adolescence will be a period during which exposure to discrimination may

have particularly important effects, and we thus adolescent PRD will be a stronger predictor of adult HPA axis functioning than PRD experiences reported in young adulthood.

### *Current Research*

In the current research, we focused on relations between histories of perceived discrimination reported from adolescence through young adulthood and cortisol diurnal rhythms measured in adulthood. We accomplished this by adding measures of diurnal cortisol to a longitudinal study in which reporting on perceived discrimination was obtained over a 20-year period from early adolescence through approximately age 32. In addition to examining measures of cumulative discrimination over a 20-year period, we also examined whether discrimination experienced 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. Finally, we examined whether racial/ethnic disparities existed in diurnal cortisol rhythms, and whether cumulative histories of racial/ethnic discrimination from adolescence through adulthood helped to explain these disparities.

### **Method**

Participants and data were drawn from the Maryland Adolescent Development in Context Study (MADICS), a prospective longitudinal study of 1,482 adolescents ( $n = 879$  Black, 49% women) from Prince Georges County, Maryland (Eccles, Early, Fraser, Belansky, & McCarthy, 1997; Eccles, Wong, & Peck, 2006; Wong, Eccles, & Sameroff, 2003). Participants were recruited in 7th grade, at age 12, and followed for the subsequent 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 1 and 2) 8th grade (Wave 3), 11th grade (Wave 4), 1 year after high school (Wave 5), 3 years after high school (Wave 6), approximately age 30 (Wave 7), and approximately age 32 (Wave 8) (Brodish et al., 2011; Fuller-Rowell et al., In press).

At Wave 8, a subset of participants, selected based on past histories of discrimination (see below) were invited to enroll in an add-on study in which biomarkers of stress and health were assessed. Participants in the health add-on study completed a variety of measures, including a week-long cortisol data collection protocol. The primary aim of the current work was to consider the relation between PRD, as reported across waves, and individuals' diurnal cortisol profiles, as assessed in the Wave 8 add-on study.

### *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 additional details on discrimination measures, below and in Appendix A), we recruited approximately equal numbers of Blacks and Whites, and both males and females with low, medium, and high levels of perceived racial/ethnic 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 4:00AM or after 2:00PM, 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 hours. One participant was excluded for not having any valid days of data. Ultimately, our sample included 120 individuals: 35 Black females, 36

White females, 22 Black males, and 27 White males distributed across low, medium, and high discrimination groups.

#### *Demographic data*

Demographic data were taken from the Wave 1 MADICS survey. Specifically, in order to assess race and ethnicity, participants were asked whether they identified as Black, White, Asian, Latino, or other. Also at Wave 1 (1991), parents reported on the total family income level on the following scale ranging from 1 (Less than \$5,000) to 16 (More than \$75,000).

#### *Perceived racial/ethnic discrimination.*

Current perceived racial/ethnic discrimination (PRD) was assessed by youth report at each of Waves 3, 4, 5, 6 and 7, using a variety of questions reflecting the extent to which individuals perceived unfair treatment due to race. Given that there were no existing scales to assess PRD among adolescents within the school context when these data were collected, Eccles and her colleagues developed new measures based on open-ended survey questions from Wave 1 and interviews with both participants and African American informants. See Wong et al., 2003 and Eccles et al., 2006 for details of scale creation, development, and both validity and reliability assessments. Measures differed slightly at each wave due to the changing age, developmental stage, and contexts present at each wave (see Appendix A for full set of PRD questions). Example questions include: “How often do you feel that you get disciplined more harshly by teachers than other kids because of your race?” (Wave 3, 8<sup>th</sup> grade; Wave 4, 11<sup>th</sup> grade) and “At work, how often have you experienced what you perceived as racist behavior or treatment?” (Wave 6, 3 years post high school, approximately age 21). Differing numbers of items were available at each wave: Wave 3

and 4 each included 8 questions ( $\alpha = 0.88$  for Wave 3,  $0.89$  for Wave 4), Wave 5 had one or two questions depending on whether the student was in school or work (no alpha reportable), Wave 6 had 3 questions ( $\alpha = 0.47$ ), and Wave 7 had 19 questions ( $\alpha = 0.94$ ). Wave 8 PRD was not included in our PRD history variable because Wave 8 was approximately concurrent with the health outcome measurement, and we wanted our variable to reflect past histories of PRD.

PRD items were standardized and averaged within each wave, and then averaged together across waves to create three different PRD history measures reflecting the developmental timing of PRD history exposures. First, a *cumulative PRD history* measure was created (averaged across Waves 3 through 7 wave scales;  $\alpha = .58$  across scales,  $\alpha = 0.90$  across all items). Next, an *adolescent PRD* measure was created by averaging the scales assessed at Waves 3 and 4 ( $\alpha = 0.87$ ). Finally, a *young adult PRD measure* was created by averaging the scales assessed at Waves 5, 6, and 7 ( $\alpha$  across scales =  $.56$ ,  $\alpha$  across all items =  $0.94$ ). Each wave had an equal weight in the final data. Individuals with missing items in a given wave had the item replaced with the individual's wave average. Individuals with missing waves had that wave replaced with their average across their available waves. Adolescent and young adult PRD were only moderately correlated with one another ( $r = .43$ ,  $p = .000$ ), suggesting that they are related, but sufficiently distinct that they could make unique contributions to the prediction of adult cortisol.

### *Salivary cortisol*

Saliva samples were gathered three times daily for one week: at waking, 30 minutes after waking, and at bedtime (Adam & 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 beginning 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 minutes 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 minutes prior to the expected sample collection times. They were asked to store samples in their refrigerators after collection, and returned samples to our team by regular postal mail. Cortisol samples are stable in saliva at room temperature for several days, and are not affected by the handling and temperature variations associated with a regular postal journey (Clements & Parker, 1998). Samples were stored at -20°C after being returned to the lab. They were later shipped on dry ice to Trier, Germany, and were assayed in duplicate using time-resolved fluorescent-detection immunoassay (DELFI; Dressendörfer, Kirschbaum, Rohde, Stahl, & Strasburger, 1992). Intra-assay variation ranged from 4.0% to 6.7%, while inter-assay variation ranged from 7.1% to 9.0%.

### *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 (Adam, 2006; Hruschka et al., 2005), models the non-independence associated with the nested structure of the data (cortisol samples nested within days, nested within individuals; 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. In line with prior work, the current analyses modeled the

general decline of cortisol levels across the day 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 (the increase in cortisol from waking to 30 minutes after waking), 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, perceived discrimination, race (dummy coded 0 = White, 1 = Black), their interactions, and person-level control variables (e.g., SES, gender, age, oral contraceptive use and average time of waking) were entered. Covariates that were significant predictors of any of the Level 1 coefficients were retained in the model; covariates that were not significant predictors of any of the Level 1 coefficients were removed from the model.

The race and gender dummy variables and the time variables were centered at their own zero points (i.e., no centering was added in the HLM model). Day-level variables were group-mean centered, and person-level variables were grand-mean centered. Analyses proceeded in the following order. First, we provided descriptive information on how our cortisol variables and covariates vary by race. Next, we examined how race, perceived discrimination, and the interactions between race and perceived discrimination related to morning cortisol (the intercept), the CAR, and the diurnal slope, controlling for our set of covariates. To maintain the most parsimonious models, covariates were only retained in the model if they showed significant associations with at least one of the cortisol outcomes,

and interactions between race and perceived discrimination are retained in the model only for the 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 better 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, and from race by PRD interactions and our set of covariates.

Finally, in order to examine the extent to which histories of perceived discrimination account for racial/ethnic disparities in cortisol rhythms, we examined the effects of race on cortisol diurnal rhythms in models *without* the perceived discrimination variables, and compared the effect sizes for the associations between race and cortisol outcomes for the models with and without the perceived discrimination variables.

## **Results**

Descriptive information on levels of cortisol, PRD, and covariates for the full sample and separately for Black and White participants are presented in Table 1. Diurnal cortisol rhythms, on average, followed the expected daily pattern of moderately high levels on waking (average of .255  $\mu\text{g}/\text{dl}$ ), followed by a rapid increase in cortisol (cortisol awakening response or CAR) to .363  $\mu\text{g}/\text{dl}$  at 30 minutes after waking, and a decline in levels across the day to low levels at bedtime (average of .067  $\mu\text{g}/\text{dl}$ ). Independent sample t-tests revealed significant differences in cortisol levels across the day for Blacks as compared to Whites: Blacks had significantly lower waking cortisol values (.234 vs. .275) and significantly higher bedtime cortisol values (.084 vs. .051) than Whites. Racial/ethnic



differences were also apparent for time of waking, with Blacks waking up 36 minutes later than Whites. Baseline levels of family income were different across race, with the average income for families of Black participants being slightly lower than those of White participants (\$46,404 vs. \$54,802). Levels of perceived discrimination also varied according to race, with Blacks being .71 SD higher than Whites on cumulative perceived discrimination. There were no significant differences between Blacks and Whites in perceived discrimination during adolescence, but strong and significant differences in perceived discrimination during the young adult years (see Table 1), with Blacks being .86 SD higher than Whites on perceived discrimination during young adulthood.

#### *Race, cumulative discrimination and cortisol*

Next, we turn to the model examining associations between race, cumulative discrimination, and their interactions in predicting diurnal cortisol rhythms (see Table 2). Associations between these variables, and relevant covariates, are presented for waking level of cortisol, the size of the cortisol awakening response, and the slope of decline in cortisol levels across the day. Non-significant covariates were removed from the model (i.e., hours of sleep, age, and oral contraceptive use). The remaining covariates, including gender, family income and both daily and average wake time were significant in at least one model and were retained in all HLM models as a result.

*Waking cortisol.* Adjusting for covariates (gender, income, and time of waking), waking levels of cortisol were on average .23  $\mu\text{g}/\text{dl}$ . Blacks had, on average, 15% lower waking cortisol levels than Whites. Controlling for race and other covariates, there was a trend ( $p = .08$ ) for higher family income at baseline predicting lower waking cortisol in adulthood, with waking cortisol being 3% lower for every \$10,000 additional family

income. There were no significant main effects of cumulative perceived discrimination (across Waves 3 to 7) on waking cortisol, but there was a significant interaction between race and cumulative discrimination, with waking cortisol being 13% lower for every 1 SD higher cumulative discrimination from adolescence through young adulthood reported by Black participants. There were no significant effects of gender or either typical or day-specific times of waking on waking cortisol.

*Cortisol awakening response.* There was a significant increase in cortisol levels after waking, with cortisol levels increasing 55% from waking to 30 minutes after waking for males, and increasing 72% on average for females. The CAR was significantly larger for females than for males. There were no effects of race, or of cumulative perceived discrimination on the size of the CAR, nor were there effects of average or typical waketimes. There were however significant effects of day-to-day changes in time of waking. On days that participants woke up later, their CARs 10% lower for every hour later time of waking.

*Diurnal cortisol slope.* There was, as expected, a strong and significant decline in cortisol levels across the day, with cortisol levels declining 26% per hour at the time of the waking for Whites. There was a trend ( $p=.07$ ) for slopes to decline more slowly across the day, with slopes of linear decline being 2% less per hour for Blacks than for Whites. There was a significant effect of cumulative exposure to perceived discrimination on diurnal cortisol slopes, with slopes declining 1% per hour more slowly (being 1% flatter) for every one SD higher on cumulative perceived discrimination 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 ( $b = .008, SE = .007, p = .257$ ).

There was a trend for females to have flatter diurnal cortisol slopes than males ( $p=.08$ ), and slopes were significantly flatter for individuals with later average waketimes. There were no significant effects of family income or daily changes in time of waking on diurnal cortisol slopes (see Table 2).

The quadratic term for the diurnal cortisol slope was significant and positive, indicating that, as expected, cortisol slopes followed a quadratic pattern of decline across the day, with slopes decelerating at a rate of 1% per hour starting at waking. We did not examine predictors of the quadratic effect of time of day on cortisol, as we had no theoretical model guiding predictions of the quadratic effect.

*Average cortisol across the day.* In order to examine associations between cumulative PRD and average cortisol levels across the whole day, we conducted a 2-level HLM model predicting the area under the curve of cortisol levels (AUC cortisol) from race, perceived discrimination, their interaction, and our set of covariates (results presented only in text). There were no significant differences between Blacks and Whites in average cortisol levels across the day ( $b = 3.12, SE = 1.82, p=.09$ ) nor was there a significant effect of cumulative PRD on average cortisol levels across the waking day ( $b = .48, SE = 1.58, p = .76$ ), nor a significant race by cumulative PRD interaction ( $b = -1.92, SE = 2.10, p = .36$ ).

#### *Developmental timing of discrimination effects*

In order to consider whether the developmental timing of PRD exposure matters, we examined whether adolescent PRD (Waves 3 & 4) and young adult (Waves 5, 6, & 7) PRD, as well as their interactions with race, were associated with diurnal cortisol rhythms (see Table 3). The same set of covariates – income, average and day-specific time of waking, and gender, were included. Results for covariates showed a very similar pattern of

significance and effect sizes as in the cumulative PRD model (Table 2) and, thus, are not discussed here again. As noted earlier, adolescent and young adult PRD were moderately correlated ( $r = .43, p = .000$ ); as a result, these two indices were entered in models both individually, and also simultaneously in order to identify their unique contributions to adult cortisol patterns.

*Waking cortisol.* One again, waking cortisol levels were significantly lower for Blacks than for Whites, with levels being 17% lower on average for Black participants. When adolescent PRD and young adult PRD are entered separately in the model predicting waking cortisol, there are no main effects of PRD, but there are significant interactions with race. Both adolescent PRD and young adult PRD are related to significantly lower waking cortisol levels for Blacks (Black by adolescent PRD interaction  $b = -.16, SE = .064, p = .01$ ; Black by young adult PRD interaction  $b = -.15, SE = .074, p = .04$ ). When adolescent PRD and young adult PRD and their interactions with race are entered simultaneously in the model to examine their unique effects (See Table 3), there was a trend ( $p = .06$ ) for high adolescent PRD to be related to lower waking cortisol for Blacks, with levels being 13% lower for every 1 SD higher adolescent PRD. There were no significant unique effects of young adult PRD on waking cortisol (see Table 3).

*Cortisol awakening response.* When adolescent and young adult PRD are entered separately in the model, there were no main effects of adolescent PRD on the size of the cortisol awakening response ( $b = .008, SE = .03, p = .79$ ), nor was there a significant race by adolescent PRD interaction ( $b = -.023, SE = .06, p = .70$ ). For young adult PRD, there was no significant main effect on the cortisol awakening response ( $b = .006, SE = .05, p = .891$ ), but there was a significant race by PRD interaction ( $b = -.148, SE = .07, p = .045$ ), with Blacks

with high young adult PRD having a significantly larger cortisol awakening response. These results held up when both adolescent and young adult PRD were entered simultaneously in the model to estimate their unique effects (see Table 3). For Blacks and for young adult PRD only, the CAR was 15% larger for every SD higher PRD.

*Diurnal cortisol slope.* As in prior models, there was a trend ( $p < .10$ ) for Blacks to have flatter slopes than Whites (see Table 3). When entered in the model separately, both adolescent and young adult PRD showed significant main effects on diurnal cortisol slopes, (adolescent PRD  $b = .010$ ,  $SE = .004$ ,  $p = .008$ ; young adult PRD  $b = .012$ ,  $SE = .004$ ,  $p = .008$ ), with higher PRD during both age periods predicting flatter diurnal cortisol slopes. There were no significant race by PRD interactions either for adolescent or for young adult PRD. When entered simultaneously, both the adolescent and young adult PRD findings are reduced to a trend level (adolescent PRD  $b = .007$ ,  $SE = .004$ ,  $p = .075$ ; young adult PRD  $b = .008$ ,  $SE = .004$ ,  $p = .059$ ). Generally, these results suggest that perceived discrimination across both age periods contributes to flatter cortisol slopes for both Blacks and Whites.

*Average cortisol across the day.* Looking at average cortisol across the day, when adolescent and young adult PRD were entered simultaneously in the model, there were no significant main effects of race ( $b = 2.77$ ,  $SE = 1.90$ ,  $p = .15$ ) and no main effects of adolescent or young adult PRD (adolescent,  $b = 1.35$ ,  $SE = 1.16$ ,  $p = .25$ ; young adult;  $b = -1.06$ ,  $SE = 1.85$ ,  $p = .57$ ). There was, however, a significant race by PRD interaction for adolescent PRD ( $b = -4.88$ ,  $SE = 1.70$ ,  $p = .005$ ), but not young adult PRD ( $b = 2.39$ ,  $SE = 2.30$ ,  $p = .30$ ) in predicting lower average cortisol levels. Blacks with higher levels of perceived discrimination during adolescence had significantly lower average cortisol than both Blacks with low PRD and Whites with low or high PRD. The notably lower overall AUC for

Blacks with high adolescent perceived discrimination is depicted in Figure 1. When adolescent and young adult PRD are entered in the model separately, similar results are found: adolescent PRD is associated with significantly lower average cortisol across the day for Blacks ( $b = -4.05, SE = 1.59, p = .01$ ), but not Whites ( $b = 1.17, SE = 1.13, p = .31$ ). Young adult PRD and its interaction with Black race/ethnicity ( $b = .39, SE = 2.17, p = .858$ ) show no significant associations with average cortisol.

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

In a reduced model examining race/ethnicity without the discrimination variables, strong effects of race are evident for waking cortisol, and for the diurnal cortisol slope. There were no significant main effects of race for the CAR. For waking cortisol, in the model without discrimination included, Black respondents have waking cortisol levels that are 20% lower than for Whites ( $b = -.226, SE = .07, p = .008$ ). In addition, Black participants had diurnal cortisol slopes that were 2.4% flatter per hour compared to White participants ( $b = .023, SE = .008, p = .005$ ). In the models above including cumulative discrimination, the size of these effects are reduced slightly, to 15% lower for Blacks for waking cortisol levels ( $b = -.157, SE = .078, p = .046$ ) and 1.5% flatter slopes, with the main effect of race on cortisol slope being reduced to marginal significance ( $b = -.015, SE = .008, p = .066$ ). Similar small reductions in effect size are found with the addition of the adolescent and young adult discrimination variables to the model (see Table 3). Thus, while this pattern of results suggests that discrimination accounts for some of the racial/ethnic disparities in diurnal cortisol rhythms, significant associations remain between Black race/ethnicity and lower morning levels and marginally significant associations remain between Black race/ethnicity and flatter diurnal cortisol rhythms remains after accounting for

developmental histories of perceived racial/ethnic discrimination from adolescence through young adulthood.

### **Discussion**

Our results suggest that developmental histories of PRD were characterized by a pattern of flatter diurnal cortisol slope and a lower area under the curve across the waking day. The pattern of flatter slopes with high PRD was present for both Blacks and Whites, and appeared to emerge from PRD experiences spanning both adolescence and young adulthood. The general lowering of the diurnal cortisol curve across the day associated with PRD was specific to Blacks reporting high PRD, and to experiences of discrimination occurring during adolescence, rather than experiences in young adulthood. Regardless of whether adolescent and young adult PRD were entered into the model simultaneously or separately, only *adolescent* levels of perceived discrimination predicted lower average cortisol levels across the day.

A pattern of lower average cortisol across the day is an indicator of hypocortisolism – a pattern of low and less dynamic cortisol levels that is thought to result from past chronic stress or trauma, and is associated with negative health outcomes (Fries et al., 2005; Heim et al., 2000). Several theories have suggested that patterns of low or attenuated cortisol levels may emerge under situations of chronic stress, after a period of over-activation of the HPA axis due to acute stress experiences. Susman and colleagues have referred to this as the attenuation hypothesis (Susman, 2006; Trickett, Noll, Susman, Shenk, & Putnam, 2010). Although no study to our knowledge has observed this pattern of attenuation emerging longitudinally, Miller and colleagues (Miller et al., 2007) reviewed extensive literature on cross-sectional studies of stress exposure and cortisol and found that

elevations in cortisol tended to occur when cortisol levels were measured shortly after the stressor, while a lowering of cortisol was found after a longer time delay post-stressor.

Somewhat in line with these hypotheses, recent (young adult) PRD experiences among blacks predicted higher, not lower, cortisol awakening responses. 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. Perhaps those reporting PRD in young adulthood are still actively mobilizing to cope with the anticipated discriminatory experiences.

Thus, our findings of more recent experiences of PRD (in young adulthood) being associated with cortisol activation, but earlier (adolescent) histories of PRD being associated with a lowering and flattening of cortisol activity, are consistent with attenuation models. That is, our findings are consistent with a model in which initially, the HPA activates in response to stressors, but over time, the system reduces responding and down-regulates, perhaps to protect the individual from chronic effects of elevated cortisol levels (Miller et al., 2007; Susman, 2006).

It is worth noting that trends towards flatter slopes for Blacks appeared to be driven by lower waking levels of cortisol. Waking levels were significantly lower for Blacks compared to Whites, but particularly when Blacks reported high PRD. These findings suggest that flatter slopes may not be the result of cumulative stressors across the day affecting evening levels, but reflect a more fundamental alteration of the circadian rhythm of cortisol release evident in morning cortisol levels. These alterations were not accounted for by proximal sleep variables, such as hours of sleep the night before or time of waking that day, suggesting that a more entrained alteration of the HPA axis has taken place.



Our results suggest that the developmental timing of stress matters – although PRD during both the adolescent and young adult time periods had implications for cortisol functioning, it was adolescent PRD that predicted the most notable alterations in the HPA axis, in particular, the hypocortisolemic (low overall cortisol) pattern noted above. The stronger effects for adolescent PRD were not simply the result of a longer chronicity of exposure -- adolescent PRD significantly predicted lower average cortisol across the day, whereas cumulative PRD, which takes into account both adolescent and early adult exposures, did not. Our evidence suggests that while young adult experience matters also, adolescence may serve as a particularly sensitive period for effects of PRD on adult stress biology. Rapid changes in neurobiology, emotion, and critical developments in the development of identity are occurring during the adolescent time period. As noted earlier, time periods of rapid developmental and neurobiological change are also hypothesized to be time periods during which environmental events may have a larger impact, as such events become programmed or embedded in the developing systems (Hertzman, 1999). Thus, race-based stress experiences during adolescence may become embedded in the developing identities, emotions, and neurobiology of minority youth.

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 greater and earlier mortality (Kumari et al., 2009; Sephton, Sapolsky, Kraemer, & Spiegel, 2000; Steptoe, Kunz-Ebrecht, Brydon, & Wardle, 2004). 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, 1994; Fries et al., 2005; Heim et al., 2000; Yehuda, 1996).

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 later onset of depression and anxiety disorders in adolescents and young adults (Adam et al., 2010; Adam et al., In press; Vrshek-Schallhorn et al., 2013). Whether or not this pattern would remain for individuals into adulthood remains to be established.

Our finding of a lower CAR with later awakening time is in line with multiple past studies of the effect of time of waking on awakening responses (Edwards, Evans, Hucklebridge, & Clow, 2001; Federenko et al., 2004; Kudielka & Kirschbaum, 2003), further justifying the importance of including awakening time as a covariate in all studies examining predictors of the CAR. Although we did not find that hours of sleep mattered for waking cortisol, the CAR, or cortisol slopes in the current analyses, past research has found sleep hours to matter for cortisol rhythms (Zeiders, Doane, & Adam, 2011), and as a result we recommend its continued examination as a covariate in future studies of race-based stress and HPA-axis functioning.

It is of interest that not only was adolescent PRD more strongly associated with hypocortisolism in adulthood than was young adult PRD, but that this was the case only among Blacks. Blacks reported significantly higher *cumulative* PRD and significantly higher *young adult* PRD than their White counterparts, but they did not report significantly higher PRD in adolescence. Despite this, adolescent PRD emerged as the strongest predictor of a lowered average cortisol level among Blacks. Thus, our cortisol findings were not just due

to an increased quantity or extremity of PRD among Blacks, but rather increased sensitivity among Black participants to PRD during adolescence. This is in line with theoretical discussions about the importance of discriminatory experiences during adolescence, especially among racial/ethnic minority youth (Brown & Bigler, 2005; Eccles et al., 2006; Fisher, Wallace, & Fenton, 2000; Wong et al., 2003).

Adolescence is a developmental period in which the abilities to perceive and understand experiences of discrimination increase (Brown & Bigler, 2005; Eccles et al., 2006; Wong et al., 2003) and for racial/ethnic minority youth, the process of developing racial/ethnic identities emerge (Eccles et al., 2006; Phinney, 1989; Wong et al., 2003). Thus, our findings may reflect the differential import of adolescence in racial/ethnic identity development for Blacks compared with Whites, which in turn affects the perception of discriminatory events for Blacks vs. Whites, and hence their biological impact. Discrimination during adolescence may have a larger impact on Blacks adolescents because the historical mistreatment, prejudices, and oppression within US society could make experiences of discrimination particularly relevant and impactful among Blacks (Branscombe, Schmitt, & Harvey, 1999; Feagin et al., 1999) especially while they are actively grappling with the process of racial/ethnic identity formation.

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 objective measures of compliance with the requested timing of cortisol samples. Although our diurnal cortisol measurement including a fairly minimal

protocol, in terms of number of samples per day, similar protocols have been widely utilized in cortisol research (Adam et al., 2006; Adam & Kumari, 2009) and our measurement of cortisol over a full 7 days helps to increase our reliability of cortisol measurement (Hellhammer et al., 2007).

Despite these limitations, this study reflects the first prospective examination of PRD measured 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, and our PRD measures partially accounted for the Black-White difference in the slope of diurnal cortisol rhythm that was present prior to the entering the PRD variables in the model, suggesting that cumulative histories of discrimination may indeed be one factor accounting for differences in cortisol slopes found in other studies. However, to fully explain racial/ethnic differences in cortisol slopes, it may be that even earlier PRD experiences – during early childhood or even during the prenatal period (maternal PRD exposure) need to be considered, as well as additional psychosocial and sociocultural factors, such as non-racial stressors from employment and/or family and relevant neighborhood characteristics.

Overall, we find that PRD during both adolescence and young adulthood matter for adult cortisol functioning, but that adolescence may be a sensitive period for the impact of perceived discrimination on overall levels of cortisol for Blacks, contributing to a hypocortisolemic pattern of lower average cortisol which has been associated with

numerous health risks. The extent to which PRD, by way of its alterations in HPA axis functioning, contributes to disparities in mental health, physical health, and academic attainment, remains to be established in future work.

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Table 1.

	Full Sample		Black Sample		White Sample		Comparison	
	Mean	SD	Mean	SD	Mean	SD	<i>t</i>	<i>p</i>
Waking cortisol level (µg/dl)	0.255	0.108	0.234	0.105	0.275	0.108	2.108	0.037
Wake + 30 cortisol (µg/dl)	0.363	0.130	0.346	0.148	0.379	0.110	1.410	0.161
Bedtime cortisol (µg/dl)	0.067	0.070	0.084	0.089	0.051	0.042	-2.673	0.009
Wake time (in decimal-hours)	7.349	1.355	7.663	1.530	7.064	1.111	-2.469	0.015
Total hours of sleep	6.249	1.092	6.143	1.150	6.345	1.038	1.012	0.314
Time between first and third samples	15.936	1.219	16.036	1.477	15.845	0.929	-0.855	0.394
Female (percent)	0.592	0.494	0.614	0.065	0.571	0.499	-0.471	0.639
Income (in 000's of \$)	50.813	20.087	46.404	23.323	54.802	15.781	2.329	0.022
Age	32.364	0.428	32.355	32.371	32.371	0.467	0.205	0.838
Birth control use (percent)	0.108	0.312	0.105	0.310	0.111	0.317	0.102	0.919
Marijuana use (percent)	0.050	0.219	0.053	0.225	0.048	0.215	-0.125	0.901
Cumulative PRD	0.000	1.000	0.373	1.132	-0.337	0.720	-4.141	0.000
Adolescent PRD	0.000	1.000	0.150	1.132	-0.136	0.850	-1.571	0.119
Young Adult PRD	0.000	1.000	0.448	1.104	-0.405	0.086	-5.144	0.000
N	120		57		63			

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

Fixed Effect	Coefficient	SE	<i>t</i>	<i>P</i>	Interpretation
Model for waking cortisol level, $\pi_0$					
Average waking cortisol level, $\beta_{00}$					
Intercept, $\gamma_{000}$	-1.478	0.060	-24.496	<0.001	Waking level=0.23 $\mu\text{g}/\text{dl}$ *
Female, $\gamma_{001}$	0.039	0.068	0.568	0.571	n.s.
Black, $\gamma_{002}$	-0.157	0.078	-2.017	0.046	-15% for Black respondents
Income, $\gamma_{003}$	-0.003	0.002	-1.765	0.080	-0.3% for every \$1000 in family income**
Wake, $\gamma_{004}$	-0.020	0.031	-0.645	0.520	n.s.
Cumulative PRD, $\gamma_{005}$	-0.030	0.046	-0.653	0.515	n.s.
Black * Cumulative PRD, $\gamma_{006}$	-0.142	0.063	-2.272	0.025	-13% for every +1SD for Black respondents
Wakeup time, $\beta_{01}$					
Intercept, $\gamma_{010}$	-0.002	0.023	-0.079	0.937	n.s.
Model for cortisol awakening response, $\pi_1$					
Average cortisol awakening response, $\beta_{10}$					
Intercept, $\gamma_{100}$	0.436	0.051	8.556	<0.001	+55% CAR for males
Female, $\gamma_{101}$	0.155	0.056	2.779	0.006	+17% larger CAR for females
Black, $\gamma_{102}$	0.011	0.064	0.171	0.864	n.s.
Income, $\gamma_{103}$	0.001	0.002	0.462	0.645	n.s.
Wake, $\gamma_{104}$	0.016	0.023	0.693	0.490	n.s.
Cumulative PRD, $\gamma_{105}$	0.027	0.032	0.839	0.403	n.s.
Wakeup time, $\beta_{11}$					
Intercept, $\gamma_{110}$	-0.105	0.020	-5.205	<0.001	-10% for every hour later waking
Model for time since waking, $\pi_2$					
Average effect of time since waking, $\beta_{20}$					
Intercept, $\gamma_{200}$	-0.303	0.028	-10.866	<0.001	-26% per hour at waking
Female, $\gamma_{201}$	0.014	0.008	1.740	0.085	1% flatter for females
Black, $\gamma_{202}$	0.015	0.008	1.854	0.066	2% flatter for Black participants
Income, $\gamma_{203}$	0.000	0.000	-0.227	0.821	n.s.
Wake, $\gamma_{204}$	0.010	0.004	2.640	0.009	1% flatter per hour later average waking
Cumulative PRD, $\gamma_{205}$	0.013	0.004	3.226	0.002	1% flatter per +1SD
Wakeup time, $\beta_{21}$					
Intercept, $\gamma_{210}$	-0.005	0.011	-0.423	0.672	n.s.

Model for time since waking squared,  $\pi_3$

Intercept, $\beta_{30}$					
Intercept, $\gamma_{300}$	0.011	0.002	6.678	<0.001	1% deceleration in slope per hour after waking
Wakeup time, $\beta_{31}$					
Intercept, $\gamma_{310}$	0.000	0.001	0.607	0.544	n.s.

Note. 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 midday values, slopes, and CAR were fixed at level 2 and 3, while all other coefficients are set as random. Random effects for the level 2 intercept was significant at the  $P < .05$  level [ $r_0 = 0.136$ ,  $\chi^2(597) = 690.530$ ,  $P = .005$ ]. Random effects for all of the four level 3 midday intercepts were significant at the  $P < 0.05$  in the full model [ $u_{00} = 0.332$ ,  $\chi^2(109) = 386.404$ ,  $P < 0.001$ ;  $u_{01} = 0.172$ ,  $\chi^2(110) = 276.070$ ,  $P < 0.001$ ;  $u_{20} = 0.158$ ,  $\chi^2(110) = 168.070$ ,  $P < 0.001$ ;  $u_{30} = 0.010$ ,  $\chi^2(115) = 261.693$ ,  $P < 0.001$ ].

\* 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:  $B_{\%change} = [\exp(B_{raw})] - 1$

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

Fixed Effect	Coefficient	SE	<i>t</i>	<i>P</i>	Interpretation
Model for waking cortisol level, $\pi_0$					
Average waking cortisol level, $\beta_{00}$					
Intercept, $\gamma_{000}$	-1.454	0.062	-23.464	<0.001	Waking level=0.23 $\mu\text{g}/\text{dl}$ *
Female, $\gamma_{001}$	0.024	0.067	0.361	0.718	n.s.
Black, $\gamma_{002}$	-0.189	0.087	-2.165	0.033	-17% for Black respondents
Income, $\gamma_{003}$	-0.003	0.002	-1.753	0.082	-3% for every \$10,000 in family income**
Wake, $\gamma_{004}$	-0.008	0.032	-0.252	0.802	n.s.
Adolescent PRD, $\gamma_{005}$	-0.032	0.055	-0.585	0.560	n.s.
Young Adult PRD, $\gamma_{006}$	0.026	0.056	0.469	0.640	n.s.
Black * Adolescent PRD, $\gamma_{007}$	-0.138	0.072	-1.911	0.059	-13% for every +1SD for Black respondents
Black * Young Adult PRD, $\gamma_{008}$	-0.087	0.075	-1.159	0.249	n.s.
Wakeup time, $\beta_{01}$					
Intercept, $\gamma_{010}$	-0.002	0.023	-0.08	0.937	n.s.
Model for cortisol awakening response, $\pi_1$					
Average cortisol awakening response, $\beta_{10}$					
Intercept, $\gamma_{100}$	0.439	0.053	8.332	<0.001	+55% CAR for males
Female, $\gamma_{101}$	0.155	0.055	2.831	0.006	+17% larger CAR for female
Black, $\gamma_{102}$	-0.003	0.065	-0.053	0.958	n.s.
Income, $\gamma_{103}$	0.001	0.001	0.36	0.720	n.s.
Wake, $\gamma_{104}$	0.019	0.024	0.808	0.421	n.s.
PRD34, $\gamma_{105}$	0.010	0.037	0.271	0.787	n.s.
PRD567, $\gamma_{106}$	-0.034	0.046	-0.737	0.463	n.s.
Black * Adolescent PRD, $\gamma_{107}$	-0.076	0.068	-1.123	0.264	n.s.
Black * Young Adult PRD, $\gamma_{108}$	0.142	0.069	2.071	0.041	+15% for every +1SD for Black respondents
Wakeup time, $\beta_{11}$					
Intercept, $\gamma_{110}$	-0.104	0.020	-5.177	<0.001	-8% for every hour later waking
Model for time since waking, $\pi_2$					
Average effect of time since waking, $\beta_{20}$					
Intercept, $\gamma_{200}$	-0.302	0.028	-10.789	<0.001	-26% for every hour at midday
Female, $\gamma_{201}$	0.014	0.008	1.741	0.084	1% flatter for females
Black, $\gamma_{202}$	0.015	0.009	1.677	0.096	1% flatter for Black participants
Income, $\gamma_{203}$	0.000	0.000	-0.168	0.867	n.s.



Wake $\gamma_{204}$	0.010	0.004	2.506	0.014	1% flatter per hour later average waking
Adolescent PRD, $\gamma_{205}$	0.007	0.004	1.794	0.075	1% flatter per +1SD
Young Adult PRD, $\gamma_{206}$	0.008	0.004	1.904	0.059	1% flatter per +1SD
Wakeup time, $\beta_{21}$					
Intercept, $\gamma_{210}$	-0.006	0.012	-0.482	0.63	-1% for every hour later waking
Model for time since waking squared, $\pi_3$					
Intercept, $\beta_{30}$					
Intercept, $\gamma_{300}$	0.011	0.002	6.596	<0.001	1% deceleration in slope per hour after waking
Wakeup time, $\beta_{31}$					
Intercept, $\gamma_{310}$	0.000	0.001	0.662	0.508	n.s.

Note. 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 midday values, slopes, and CAR were fixed at level 2 and 3, while all other coefficients are set as random. Random effects for the level 2 intercept was significant at the  $P < .05$  level [ $r_0 = 0.135$ ,  $\chi^2(597) = 690.462$ ,  $P = .005$ ]. Random effects for all of the four level 3 midday intercepts were significant at the  $P < 0.05$  in the full model [ $u_{00} = 0.307$ ,  $\chi^2(107) = 376.586$ ,  $P < 0.001$ ;  $u_{01} = 0.172$ ,  $\chi^2(109) = 276.589$ ,  $P < 0.001$ ;  $u_{20} = 0.154$ ,  $\chi^2(109) = 166.047$ ,  $P < 0.001$ ;  $u_{30} = 0.010$ ,  $\chi^2(115) = 261.988$ ,  $P < 0.001$ ].

\* 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:  $B_{\%change} = [\exp(B_{raw})] - 1$

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

