Perceived Stigmatization, Ingroup Pride, and Immune and Endocrine Activity: Evidence From a Community Sample of Black and Latina Women

Kyle G. Ratner, May Ling Halim, and David M. Amodio

Abstract

Racial disparities are a major public health concern in the United States. The authors examined whether Black and Latina community members’ perceptions of stigmatization and personal feelings about their group relate to immune and endocrine markers associated with health risk, including the cytokine interleukin-6 (IL-6), which coordinates the immune response to infection, the anabolic hormone dehydroepiandrosterone (DHEA), which promotes cellular resilience, and the catabolic hormone cortisol, which releases metabolic stores in response to threat. Greater perceived stigmatization was associated with higher basal IL-6, whereas greater ingroup pride was related to elevated basal DHEA. These associations remained with adjustment for general perceived stress, experience with discrimination, age, and income. No significant perceived stigmatization or ingroup pride effects emerged for basal cortisol. These findings provide new evidence that perceived stigmatization and ingroup pride are linked to indicators of disease and resilience, respectively, highlighting mechanisms through which racial and ethnic stigmatization may contribute to health disparities.

Keywords
raced and ethnic health disparities, perceived stigmatization, DHEA, IL-6, ingroup pride, cortisol

In the United States, members of racial and ethnic minority groups are at a substantially greater risk for stress-related health problems than those in the White majority (American Cancer Society, 2011; Centers for Disease Control and Prevention, 2011). These group-based health disparities have traditionally been explained by societal inequalities, such as differences in socioeconomic status and access to quality health care (for a review, see Williams & Collins, 1995). More recently, however, several investigators have theorized that the psychological experience of belonging to a racial or ethnic group that is stigmatized by society may also contribute to these disparities (Clark, Anderson, Clark, & Williams, 1999; Halim, Yoshikawa, & Amodio, in press; Mays, Cochran, & Barnes, 2007; Pascoe & Smart Richman, 2009).

The current research investigated the direct relationships between the psychological experience of belonging to a racially stigmatized group and biological processes linked closely to disease and resilience. Specifically, we examined the relations among Black and Latina women’s perceptions of stigmatization, their personal feelings about their race/ethnicity, and baseline levels of three stress-related biomarkers—the immune protein interleukin-6 (IL-6) and the hormones dehydroepiandrosterone (DHEA) and cortisol—as part of a broader effort to illuminate the enduring racial disparities in health.

Stigmatization and Ingroup Pride

Racial and ethnic discrimination was legally prohibited in the United States nearly 50 years ago as a result of the 1964 Civil Rights Act; yet, members of American racial and ethnic minority groups continue to experience frequent instances of unfair treatment (Landrine & Klonoff, 1996; Panter, Daye, Allen, Wightman, & Deo, 2008; Pérez, Fortuna, & Alegria, 2008; Swim, Hyers, Cohen, Fitzgerald, & Bylsma, 2003). Even in the absence of direct experiences with discrimination, Black and Latino Americans are exposed to the media’s portrayal of their groups according to historically entrenched negative stereotypes (Dixon, 2008; Dixon & Linz, 2000), which contributes to the pervading belief among many minority group members that their group is viewed in a negative light by society at large. This belief describes the psychological experience of perceived stigmatization—a belief that is also referred to as low public
collective self-esteem or public regard (Luhtanen & Crocker, 1992; Major & O’Brien, 2005; Sellers, Smith, Shelton, Rowley, & Chavous, 1998). Members of racial and ethnic minority groups may experience this sense of being stigmatized independently of their personal experience with discrimination (Eccleston, 2008; Richman & Jonassaint, 2008), and therefore perceived stigmatization can affect all members of a group across many situations. Furthermore, because society’s low regard for one’s group is often experienced as stable and beyond one’s control, perceived stigmatization has been likened to a chronic stressor and, as such, has been examined for its potential associations with mental and physical health risks (Eccleston, 2008; Major & O’Brien, 2005).

Early theories of stigmatization proposed that minority group members internalize public disregard, such that they develop a negative view of their group, and of themselves as a member of this group (Clark, 1965; Kardiner & Ovesey, 1951). However, empirical research has largely not supported this prediction. Rather, Black and Latino Americans tend to report high self-esteem and espouse racial and ethnic pride, despite the devalued status of their groups in American society (Crocker, Luhtanen, Blaine, & Broadnax, 1994; Gurin & Epps, 1975; Porter & Washington, 1979; Rowley, Sellers, Chavous, & Smith, 1998; Spencer-Rodgers & Collins, 2006; Wiley, Perkins, & Deaux, 2008). Findings such as these reveal a critical distinction between the perception of how the public views one’s group (i.e., public regard or public collective-esteem) and the personal attitude held toward one’s racial or ethnic group (i.e., private regard or private collective self-esteem; Luhtanen & Crocker, 1992; Sellers et al., 1998). This distinction has proven critical for research on the health implications of racial and ethnic stigmatization.

**Implications of Group-Based Attitudes for Health**

To date, research on the health implications of perceived stigmatization has focused primarily on psychological well-being. Although public collective self-esteem (public esteem) is not typically associated with explicit personal self-esteem, it has been linked to feelings of generalized psychological stress (Sellers & Shelton, 2003), as well as to physical health symptoms, such as headaches and heart palpitations (Rivas-Drake, Hughes, & Way, 2009). This pattern suggests that although people may react to stigma by bolstering their explicit views of the self and their ingroup the perception of being stigmatized may nevertheless take a toll on less controllable aspects of one’s health, such as generalized distress and physical illness. Therefore, when examining the relation between perceived stigmatization and low-level biological processes associated with health, we would expect that low public esteem would be associated with a biological profile of greater risk for illness.

In contrast to public esteem, private collective self-esteem (private esteem)—one’s personal view of his or her group—has been shown to reflect inner strength and resilience in the face of adversity (Crocker & Luhtanen, 1990). In line with these findings, more positive private esteem has been associated with better psychological well-being, as indicated by greater personal self-esteem and life satisfaction, along with lower depression (Crocker et al., 1994; Rowley et al., 1998). This body of research suggests that greater private esteem should be associated with biological processes involved in resilience to disease.

**Immune and Endocrine Processes Linked to Disease and Resilience**

Psychosocial factors can have profound effects on the immune system and related endocrine processes (Glaser & Kiecolt-Glaser, 2005; Kirschbaum & Hellhammer, 1989; Maier & Watkins, 1998; McEwen, 1998). For instance, stressful life experiences can influence levels of immune response molecules called cytokines. Cytokines coordinate a host of processes that are critical for combating infection and injury. Although there are several types of cytokines (e.g., tumor necrosis factor, interleukin-1), the present research focused specifically on IL-6, which is among the most widely studied and has been shown to be sensitive to social stress (Maier & Watkins, 1998; Sjögren, Leanderson, Kristenson, & Ermerudh, 2006; Slavich, Way, Eisenberger, & Taylor, 2010). IL-6 is secreted by lymphocytes and plays a critical role in elevating body temperature (i.e., fever) and signaling the release of other proteins from the liver that modulate inflammation (Van Snick, 1990). Although IL-6 is adaptive in the short term by mobilizing response to possible infection, high chronic levels are thought to be a risk factor for poor coronary health and cancer (Danesh et al., 2008; Hodge, Hurt, & Farrar, 1998).

Responses to psychosocial stressors have also been associated with endocrine activity. The hormone DHEA and its sulfated metabolite DHEAS are the most abundant steroidal hormones in humans. As an anabolic steroid—a class of hormones involved in the repair of cellular trauma (Epel, 2009; Mendes, Gray, Mendoza-Denton, Major, & Epel, 2007)—DHEA promotes the strength of organ systems and the integrity of the immune system, as well as resilience to the negative consequences of physical and social stressors (Kroboth, Salek, Pittenger, Fabian, & Frye, 1999). Higher basal DHEA levels have been associated with better coping against the duress of military training (Morgan, Rasmussen, Pietrzak, Coric, & Southwick, 2009), whereas lower basal DHEAS levels have been related to many negative health outcomes, including death from cardiovascular disease, chronic fatigue syndrome, and anxiety disorders (Barrett-Connor, Khaw, & Yen, 1986; Fava et al., 1989; Scott, Salahuddin, Cooney, Svec, & Dinan, 1999). Although DHEAS has been studied in relation to intergroup anxiety in White Americans (Mendes et al., 2007), no research, to our knowledge, has examined the relation between responses to stigmatization and DHEA among members of racial and ethnic minority groups.

In the field of social psychology, research on the psychosocial effects on endocrine function has focused primarily on the hormone cortisol (e.g., Amodio, 2009; Dickerson & Kemeny, 2004; Page-Gould, Mendes, & Major, 2010). Cortisol is the
primary catabolic stress hormone released by the adrenal gland in response to threat—including the threat of social evaluation (Dickerson & Kemeny, 2004). By breaking down cellular structures to release energy, cortisol provides an organism with a metabolic boost to respond adaptively to threat, such as with a fight-or-flight response (Cannon, 1932; McEwen, 1998). Additionally, in the short term, cortisol is a potent suppressor of IL-6 production and inflammation. At sustained high levels, however, cortisol no longer has anti-inflammatory effects (Miller, Cohen, & Ritchey, 2002). Because of cortisol’s catabolic effects and influence on the immune system, chronically elevated activity may pose a health risk (McEwen, 1998).

Cortisol levels typically peak during early morning hours and decline from the afternoon until reaching the lowest point in the evening (Kirschbaum & Hellhammer, 1989; Weitzman et al., 1971). However, people who experience chronic stress demonstrate a blunted increase in waking cortisol and a less steep decline in evening cortisol, which forms a flatter diurnal slope (Adam & Kumari, 2009; Friedman, Karlamangla, Almeida, & Seeman, 2012). Consistent with the notion that stigmatized minorities experience stressful lives, there is evidence that Black individuals generally exhibit a flatter diurnal slope than non-Latino Whites (Cohen et al., 2006; DeSantis et al., 2007). Other work examining the situational manipulations of cortisol reactivity has found greater reactivity among minority individuals in response to social evaluative threat and manipulated discrimination stress (Page-Gould et al., 2010; Townsend, Major, Gangi, & Mendes, 2011). However, a direct link between perceived discrimination and less healthy basal cortisol levels is not well established, with one study finding no associations (Cohen et al., 2006), and another study finding an association only in White participants (Fuller-Rowell, Doan, & Eccles, 2012).

**Study Overview**

The present research examined responses to ethnic/racial stigmatization and ingroup pride (i.e., measures of public and private esteem) as they relate to baseline resting levels of salivary IL-6, DHEA, and cortisol in a New York City community sample of Black and Latina women. These biomarkers were examined because of their established links to risk for disease and resilience and because they can each be reliably assessed noninvasively from saliva (Granger, Schwartz, Booth, Curran, & Zakaria, 1999; Kirschbaum & Hellhammer, 1989; Sjögren et al., 2006). All analyses were adjusted for general perceived stress, experience with discrimination, age, and income.

Two broad hypotheses are suggested by existing research. As noted above, low public esteem has been linked to physical symptoms associated with illness, and thus we expected that it would be associated with higher basal levels of IL-6. By comparison, high private esteem—one’s personal attitude toward one’s social group—has been described as a protective factor in the context of stigmatization, which suggests that it may relate to higher baseline DHEA. Additionally, given the hypothesized relation between perceived stigmatization and chronic stress in past research, it is possible that perceived stigmatization may relate to baseline cortisol levels. However, because the functions of cortisol are very complex, and because our research was not optimally designed for assessing diurnal cortisol variation, which has been linked to generalized stress, predictions for cortisol effects were tentative.

**Method**

**Participants**

Sixty Black and Latina female community members were recruited from a job skills training program that serves women in New York City (Age: 18–44 years, \( M = 29 \); median income $20,000–30,000). They received $40 for completing the study. Data from participants who were pregnant (\( n = 1 \)), breastfeeding (\( n = 1 \)), had experienced menopause (\( n = 8 \)), or were taking oral contraception (\( n = 3 \)) were excluded from analysis because of the potential effect of these conditions on hormone and cytokine levels (e.g., Gameiro, Romão, & Castelino-Branco, 2010; Labrie, Martel, & Balser, 2011). Data from participants with missing data (\( n = 3 \)) or outlying IL-6 and DHEA scores (+/− 3 SDs; \( n = 3 \)) were excluded from analysis. These exclusions yielded a sample of 41 participants (self-identified: 27 Black, 11 Latina, 3 Black–Latina). Although this sample was small in comparison with large epidemiological surveys, it allowed for an initial test of our hypotheses in this unique sample.

**Procedure**

After providing consent and receiving instructions, participants completed a set of questionnaires privately on individual computers in a computer lab with approximately 25 individual workstations. Participants then provided duplicate saliva samples (.5 ml) via passive drool. All sessions were conducted in the late afternoon.

**Measures**

Public esteem and private esteem were measured using their respective subscales of the Collective Self-Esteem Scale (Luhtanen & Crocker, 1992). The public esteem subscale assesses the perception of how society perceives one’s racial or ethnic group (e.g., *In general, others think my group is unworthy*). The private esteem subscale measures personal feelings about one’s group (e.g., *In general, I’m glad to be a member of my group*). Although each item referred to a general “group,” participants were instructed to respond with regard to their own racial or ethnic group, which they had self-identified and indicated on a previous questionnaire. Each subscale included 4 items, scored on a 1–7 scale and coded so that higher scores reflected greater public esteem (\( M = 3.92, SD = 1.28; \alpha = .54 \)) and greater private esteem (\( M = 5.84, SD = 1.13; \alpha = .70 \)). A set of adjustment variables were also assessed. These included personal experience with discrimination, generalized
perceived stress, age, and reported income. Personal experience with discrimination was assessed with the 9-item Everyday Discrimination scale (Williams, Yu, Jackson, & Anderson, 1997; \( \alpha = .85; M = 1.19, SD = .70 \)). Participants were asked to respond to the items according to the prompt, How often do you experience the following events because of your race or ethnicity? Example items included, You are treated with less respect than other people and You are called names or insulted. Response options were: 0 = never, 1 = rarely, 2 = sometimes, 3 = often, with higher scores indicting greater personal experience with discrimination. The Perceived Stress scale (Cohen, Kamarck, & Mermelstein, 1983; \( \alpha = .85 \)) included 14 items (e.g., In the last month, how often have you felt nervous and ‘stressed’?), on a 0–4 scale, with higher numbers reflecting greater perceived stress (\( M = 2.03, SD = .53 \)).

Upon their collection, saliva samples were stored in a cooler and then later transferred to a −80°C freezer. Samples were then shipped and enzyme-immunoassayed in a single batch for DHEA, IL-6, and cortisol concentrations by Salimetrics Labs (University Park, PA). Mean concentrations from the duplicate samples were used as dependent measures.

### Results

Zero-order correlations between all variables, along with their means and SDs, are presented in Table 1 for descriptive purposes. However, the hypothesized associations were tested using linear regressions, in which critical adjustment variables were included, and therefore, the zero-order correlations should be interpreted with caution. In the regression analysis for each biomarker outcome variable, the predictors included public esteem, private esteem, personal experience with discrimination (i.e., everyday discrimination), general perceived stress, age, and income.

#### IL-6

Higher basal levels of IL-6 were expected to relate to lower public esteem. Indeed, this analysis produced a significant negative association between public esteem and basal IL-6, \( \beta = -.46, t(34) = 2.85, p = .007 \) (Figure 1A). This analysis also produced a marginal effect for income, \( \beta = -.32, t(34) = 1.90, p = .07 \), indicating that IL-6 was higher among lower income participants, independent of its relation to public esteem. Private esteem was not related to IL-6 levels, \( \beta = .22, t(34) = 1.37, p = .18 \). No other effects reached significance, \( ps > .77 \).

#### DHEA

Higher basal DHEA was expected to relate to greater private esteem. Supporting this prediction, private esteem for one’s group was positively associated with higher baseline DHEA, \( \beta = .35, t(34) = 2.79, p = .009 \) (Figure 1B). In line with substantial evidence that has shown that DHEA declines with age (see Kroboth et al., 1999, for a review), our analysis indicated that older participants exhibited lower concentrations of DHEA, \( \beta = -.34, t(34) = 2.30, p = .03 \). Independent of these effects, greater income was related to higher DHEA concentrations, \( \beta = .28, t(34) = 2.14, p = .04 \). No other effects reached significance, \( ps > .14 \).

#### Cortisol

For the analysis of cortisol, we excluded four additional participants who had extreme outlying cortisol concentration values (+/- 3 SD). In the regression analysis neither public esteem, \( \beta = .27, t(30) = 1.60, p = .12 \), nor private esteem, \( \beta = .03, t(30) = .16, p = .88 \), significantly predicted cortisol concentrations. These null effects were not surprising, given that we only collected saliva in the midafternoon in this study, and basal cortisol samples collected at this time have not previously
been related to social stressors. It is notable, however, that a significant effect emerged for income, $\beta = .37$, $t(30) = 2.08$, $p = .05$, such that higher income was associated with higher levels of baseline cortisol. A marginal effect also emerged for general perceived stress, $\beta = .41$, $t(30) = 1.95$, $p = .06$. While the finding that general stress related to a baseline afternoon measure of cortisol is novel, this marginal effect is consistent with the broader literature linking higher cortisol levels assessed later in the day to general life stress. Thus, it is possible that this effect provides validation of the cortisol measure in the context of the observed null effects for public and private esteem.

**Discussion**

Past research suggests that the perception of being devalued in society on the basis of race or ethnicity can take a toll on one’s mental and physical health (Rivas-Drake et al., 2009; Sellers & Shelton, 2003). Here, we provide new evidence that the perception of social stigmatization relates to activity of the immune system. Specifically, Black and Latina women who viewed their group as being more devalued by society exhibited elevated baseline levels of the cytokine IL-6, an immune-system protein that promotes fever and inflammation in response to infection or injury. Because high basal IL-6 levels have been linked to risk for a host of illnesses, including cardiovascular disease and cancer (Danesh et al., 2008; Hodge et al., 1998), this result suggests that the perception of being stigmatized may pose a significant health risk.

Our results also revealed that participants holding more positive personal attitudes about their ethnic or racial group exhibited higher baseline levels of DHEA, an anabolic hormone that is associated with resilience to disease. This finding builds on research showing that a positive view of one’s group relates to higher self-esteem in the face of potential stigma and discrimination (Crocker et al., 1994; Rowley et al., 1998), and our finding highlights a biological pathway through which high private esteem promotes good health. Furthermore, the present research helps to forge an important connection between intergroup processes and DHEA, a hormone widely recognized for its prominent role in endocrine function and as a biological buffer against stress, but understudied in social contexts (see also Mendes et al., 2007, who examined DHEAS in White majority-group members during interracial interactions). This finding is particularly noteworthy given that, to date, research on the relation between race and hormones has predominantly focused on cortisol (e.g., Cohen et al., 2006; Fuller-Rowell et al., 2011).

The results observed for public and private esteem on IL-6 and DHEA, respectively, emerged beyond any effects of personal experience with discrimination, general perceived stress, age, or income. Thus, these effects could not be attributed to participants’ general sensitivity to stressful experiences or to differences in their personal experiences with discrimination. Taken together, these findings highlight the importance of considering the psychological experiences of feeling devalued by society on the basis of one’s social group and of feeling good about one’s own racial or ethnic group as critical factors in biological health processes.

Neither public nor private esteem for one’s ethnic group was associated with baseline cortisol concentrations. However, the procedure used in the present study—a single afternoon sample
rather than an assessment of diurnal change (e.g., Cohen et al., 2006)—was not optimized for cortisol analysis. That said, it is possible that cortisol is not directly associated with the global psychological perception that one belongs to a stigmatized group. Future research with larger participant samples and more sensitive assessment procedures will be needed to further examine the relation between social stigmatization and cortisol.

It is notable that participants’ direct experience with racism was not associated with any of the biological outcome variables. There are several potential explanations for why effects emerged for perceived stigmatization, but not personal experiences with discrimination. One possibility is that instances of personal discrimination do not loom as large as a general sense of stigmatization. Research has shown that people report more discrimination against their group than they directly experience themselves (Armena & Hunt, 2009; Taylor, Wright, Moghadam, & Lalonde, 1990). It could also be that stigmatization is more difficult to discount. It has been shown that attributing particular instances of negative treatment to discrimination can have protective effects (Crocker, Voeikl, Testa, & Major, 1991; Krieger & Sidney, 1996; Rowley et al., 1998). However, perceived stigmatization requires a global assessment about the reputation of one’s group, and past research has found that external attributions that are negative and global are detrimental to well-being (Branscombe, Schmitt, & Harvey, 1999; Leonardelli & Tormala, 2002; Weiner, 1985). For these reasons, stigmatization may constitute a more pervasive psychosocial stressor than personal experiences with discrimination. An important goal of future research will be to identify the respective effects of perceived stigmatization and personal experience with discrimination on health risk.

**Limitations**

While providing an initial exploration of biological correlates of social stigmatization, our findings must be interpreted in the context of several limitations. First, the participant sample included only female community members. Although the inclusion of a community sample with a wide age range facilitates the generalization of our findings to the broader population of interest, the extent to which these findings generalize to men is less certain.

A second limitation was that our procedure included a saliva sample from a single time point. This was due to restricted access to our community participants. Thus, although we were able to find interesting and theoretically consistent associations, we were not able to fully characterize fluctuations in psychological and physiological responses throughout the day and consistency across days. This was especially a limitation for assessing cortisol effects, as previous research linking cortisol to both life stress and participant race assessed diurnal variation (Cohen et al., 2006). Nevertheless, an association between perceived stress and cortisol emerged in our study, suggesting that the single afternoon salivary assessment might be sensitive to baseline effects.

Third, the sample size was small, particularly in comparison to large epidemiological studies. Due to limited access to the community sample, we could only recruit a relatively small number of participants and assess a restricted set of variables. Nevertheless, the observed relationships were robust and not driven by outliers, as evident in Figure 1.

It is also notable that some psychological factors that were not assessed, such as depression and physical ailments, may be associated with both perceived stigmatization and the endocrine and immune processes examined here. Although we did not assess these variables in our study, they are known to be strongly associated with generalized perceived stress, a variable that was measured and included as a covariate in our analyses. Thus, theoretically, the effects of these unmeasured variables would likely be mediated through increased general perceived stress. Because our effects emerged after adjusting for general perceived stress, it is unlikely that they merely reflect the role of these unmeasured factors. Larger scale studies may be conducted in the future to include a broader range of relevant psychosocial and health variables while also increasing statistical power. It will also be important for these studies to examine effects on actual health outcomes, such as incidence of cancer, heart disease, as well as resistance to and recovery from infection.

**Implications for Public Health and Social Psychology**

From a public health standpoint, our results suggest that, in addition to improving social-economic factors among disadvantaged racial and ethnic groups, strategies for reducing health disparities should address the psychological effects of stigmatization. An important short-term intervention would be to develop strategies that targetstigmatization could use to deflect or diminish its influence. Our research suggests that valuing one’s racial and ethnic group is associated with a healthy endocrine profile. Therefore, bolstering racial and ethnic pride and the appreciation of cultural history and achievements within minority communities might buffer against negativity expressed from the greater society (see also, Baldwin, 1984; Cross, 1991; Du Bois, 1903; Sellers et al., 1998). Of course, the cause of stigmatization effects is the social system, and therefore, a longer-term intervention would require broad programs of education and social action campaigns that encourage intercultural understanding, an appreciation of the historical and political bases of group disparities, and the proscription of stereotypical portrayals of minority groups in the media and popular culture.

Our findings also have implications for social psychological theories of group identity and health. Most importantly, our findings suggest that perceived stigmatization and ingroup pride may influence health risk through separate psychobiological pathways. That is, perceived stigmatization appears to be associated with psychosocial stress and biological risk of disease, whereas ingroup pride is associated with biological processes that promote resilience. However, it will also be critical for subsequent work to consider the moderating role...
of other components of racial and ethnic identity (Cross, 1991; Luhtanen & Crocker, 1992; Parham & Helms, 1981; Sellers et al., 1998). For instance, the importance of one’s group identity (i.e., centrality) can influence public and private esteem. Individuals often cope with identity threat by deemphasizing the importance of their group. However, when deidentification is not possible, they may respond by embracing their group with increased positive attitudes (Branscombe et al., 1999; Rowley et al., 1998). Thus, centrality may prove to be a moderator of the relative impact of perceived stigmatization and ingroup pride on health-related physiological functioning.

Conclusion

Our findings join the growing body of evidence indicating that enduring racial disparities in health may reflect the impact of perceived stigmatization and ingroup pride on biological systems associated with disease and resilience. The illumination of a psychobiological pathway through which stigmatization “gets under the skin” is critical for advancing theoretical models of the social–biological health interface as well as for informing new social policies that recognize psychobiological contributions to race and ethnicity-related health disparities.

Acknowledgements

The authors thank Carly Erskine and Michele Dorsainvil for their assistance with data collection, Claudia Farb for her help with saliva sample storage, and members of the NYU Social Neuroscience Laboratory for their feedback on earlier versions of this manuscript.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclose receipt of the following financial support for the research and/or authorship of this article: This research was supported by awards from the New York University Research Challenge Fund and National Science Foundation (NSF) (BCS 0847350) to David Amodio and a NSF Graduate Research Fellowship to Kyle Ratner.

Notes

1. DHEAS is sometimes assessed as an indicator of DHEA instead of DHEA directly. The rationale for measuring DHEAS is often that it is more robust to diurnal fluctuation (Kroboth et al., 1999). However, DHEA levels after the postawakening period are relatively stable, especially in comparison to diurnal fluctuations of cortisol (Hucklebridge, Hussain, Evans, & Clow, 2005). Moreover, previous research suggests that a single daytime baseline measure can relate inversely to psychosocial stress (Morgan et al., 2009) and coronary disease (Herrington et al., 1990).

2. When hormones are assayed from saliva instead of blood plasma, it is recommended that DHEA, and not DHEAS is measured, since DHEA is more robust to artifacts resulting from blood contamination and differences in saliva flow rate (Kivlighan et al., 2004; Vining, McGinley, & Symons, 1983).

3. Although it is often informative to examine effects of the basal cortisol-DHEA or cortisol-DHEAS ratios given the opposing influences of these hormones, our data were not suited for such an analysis. Cortisol-DHEA/DHEAS ratios are typically calculated from waking or bedtime hormone levels (e.g., Michael, Jenaway, Paykel, & Herbert, 2000). Moreover, a lack of an association between distress and baseline DHEAS-cortisol ratio has been reported when cortisol and DHEAS are measured in the late afternoon (Goodyer, Herbert, & Altham, 1998; Morgan et al., 2004). No significant cortisol-DHEA ratio effects emerged in our data.

References


**Bios**

**Kyle G. Ratner** is pursuing his PhD in social psychology at New York University. He is broadly interested in the interplay between social influences and biological systems, with a special emphasis on intergroup bias and stigmatization.

**May Ling Halim** is also pursuing her PhD in social psychology at New York University. Her research focuses on social identification in children and adults and links between social identification and health.

**David M. Amodio** is an associate professor of psychology and neural science at New York University, where he directs the NYU Social Neuroscience Laboratory. His research investigates the psychological and physiological mechanisms of intergroup relations and self-regulation.