DOES SOCIAL STATUS PREDICT INDIVIDUAL DIFFERENCES IN HYPOTHALAMIC-PITUITARY-ADRENOCORTICAL ACTIVITY?

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DOES SOCIAL STATUS PREDICT INDIVIDUAL DIFFERENCES IN HYPOTHALAMIC-PITUITARY-ADRENOCORTICAL ACTIVITY?

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Lack of control and threats to social standing, whether evoked by acute or chronic stressors or reflected in symptoms of depression, are salient correlates of hypothalamic-pituitary-adrenal (HPA) activity. These conditions are also reminiscent of low social status (subordination), which has long been associated with HPA activity in non-human primates. In humans, interpersonal dominance and socioeconomic indicators are often used interchangeably to describe social status, but have distinctly different referents. Here, we examined the relationship of these two status constructs with three indices of HPA functioning [Cortisol Awakening Response (CAR), diurnal decline in cortisol (slope), and cortisol Area Under the Curve (AUC)] in 488 employed, healthy volunteers (30-54 yrs; M=43; 53% F; 83% White). Measurements of salivary cortisol were taken on five occasions during three workdays and one non-workday. Cortisol indices were averaged over work days, and non-workday indices were analyzed separately. A trait measure of dominance was calculated using items from the NEO Personality Inventory-Revised, and socioeconomic status (SES) was indexed to participants' annual income, years of education, and occupational grade. Trait dominance and SES were entered separately as predictors of each HPA index in hierarchical linear regressions adjusted for age, sex, and race. A three variable composite of SES did not associate with cortisol, but an index restricted to income and occupation did. Both low trait dominance and low income and occupation derived SES associated with a larger workday CAR (β= -.13, p=.02 and β= -.17, p=.007) and flatter workday

diurnal slope (β= -.11, p=.03and β= -.16, p=.002), but were unrelated to AUC or any non-workday indices. Trait dominance and SES were only weakly correlated (r=.08, p=.09), and findings persisted when the two predictors were entered together in regression models. These results show two largely independent conceptualizations of social status in humans related to metrics of cortisol activity.

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1.0 INTRODUCTION

The hypothalamic-pituitary-adrenocortical (HPA) system is a neuroendocrine axis that is activated in circumstances that perturb homeostasis. Because psychological stress can also trigger an HPA response, this system has long been of interest to behavioral scientists, and its principal output, the glucocorticoid hormone, cortisol, is often referred to as a "stress" hormone. Although no normal values of cortisol have been established, prolonged activation of the HPA axis is associated with a number of deleterious health consequences (e.g. Glaser & Kiecolt-Glaser, 2005; Chrousos, 2000; Dekker et al., 2008; Delany, Dong, & Canalis, 1994; Lupien et al., 1998). Hence, the HPA system is widely considered a potential mechanism mediating effects of stress on long term health outcomes. Cortisol is secreted in pulses from the adrenal cortex when stimulated by the anterior pituitary hormone, adrenocoricotrophic hormone (ACTH), which is itself released into the circulation under hypothalamic control, stimulated by corticotropin-releasing hormone (CRH). The diverse biological actions of cortisol are mediated by intra-cellular glucocorticoid and mineralocorticoid receptors (GRs; MRs), which are present throughout the body. Termination of cortisol release is effected by feedback inhibition of CRH following activation of GRs located in the hippocampus and pituitary gland (see Young, Abelson & Lightman, 2004; Funder, 1997).

1.1 METRICS OF HPA ACTIVITY

HPA activity has been of interest behaviorally in relation both to acute environmental exposures (i.e. laboratory stressors) and chronic life circumstances, such as protracted psychological stress or psychopathologies of mood and affect (e.g., depression). In such studies, the primary (though not sole) index of HPA functioning has involved assessment of cortisol in circulation from blood, urine, or saliva. At present, sampling from plasma and saliva are preferred in laboratory investigations, and studies in naturalistic settings commonly rely on assessments from saliva. The latter is justified by a high and well-established correlation between salivary and plasma measurements ($r \geq .9$) and by the predominance of bioavailable (unbound) cortisol in saliva (Kirschbaum & Hellhammer, 1989; Kahn, Rubinow, Davis, Kling, & Post, 1988; Francis et al., 1987; Luthold, Marcondes, & Wajchenberg, 1985).

Time courses of cortisol assessment must take into account circadian variation, as cortisol release follows a clear diurnal pattern, with levels highest in the early morning hours, falling sharply in the several hours after awakening, and then continuing to decline at a more modest rate through the remainder of the day. Superimposed upon the diurnal rhythm is an acute rise in cortisol (commonly of about 50%) occasioned by awakening itself – referred to as the Cortisol Awakening Response (CAR) -- which peaks at about 30 minutes. Because disturbance of "normal" cortisol dynamics might reflect aspects of HPA dysregulation, the CAR and several indices of the diurnal rhythm have been employed in cortisol research. The latter include the slope of diurnal decline (Stone et al., 2001; Cohen, Doyle, & Baum, 2006; Sephton, Sapolsky, Kraemer, & Spiegel, 2000), and to assess total cortisol output, variously determined "area under the curve" (AUC) metrics (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003).

Because of the diurnal variation in cortisol, several measurements per day provide a more reliable estimate of total daily output and diurnal rhythm than single readings. Variance estimates from multilevel regression models suggest that at least four samples per day are necessary for estimating total daily cortisol output, and at least four to five samples are needed to estimate diurnal variation (Hruschka, Kohrt, & Worthman, 2005). The timing of measurements is also of great importance as time of day has been shown to account for up to 72% of variation in observed cortisol values across the day (Adam & Gunnar, 2001; Adam, 2006); thus, it is important to compare samples relative to the time of awakening, especially when the diurnal rhythm is of interest (Stewart & Seeman, 2000). Aside from within-day sampling, significant day-to-day variation argues against measurements restricted to a single day (Smyth et al., 1997). Results of some studies suggest that a reliable estimate of diurnal slope can be calculated from 3 days of sampling (Kraemer et al., 2006; Stone et al., 2001), although other studies indicate 3-4 days of sampling days necessary to estimate total cortisol output reliably and, for slope estimates, even more extensive sampling may be needed (Stewart & Seeman, 2000). Finally, cortisol samples taken on work and non-work days may differ, although only the morning awakening response (CAR) has yet been studied (with higher values obtained on work days; Kunz-Ebrecht, Kirschbaum, Marmot, & Steptoe, 2004; Schlotz, Hellhammer, Schulz & Stone, 2004). In sum, differences in cortisol sampling procedures may pose potential difficulties for interpretation when findings are inconsistent across studies.

1.2 ACUTE STRESS STUDIES

Cortisol has been studied extensively in relation to both acute and chronic stress. Studies of the former have made use of various types and combinations of stress-inducing stimuli. Other sources of variation among studies are differences in sampling methods (e.g., collection by blood or saliva), and as noted above, comparisons across studies may be impeded by differences in the frequency and timing of cortisol measurements. Among the diversity of stressors represented in acute stress studies, one test protocol -- the Trier Social Stress Task (TSST; Kirschbaum, Pirke, and Hellhammer, 1993) – has become increasingly prominent. This 10 minute stressor has components of public speaking and mental arithmetic, both performed in front of an audience and video camera. Although currently the TSST is the most commonly administered laboratory stimulus in the acute stress literature on cortisol and evokes a uniformly robust cortisol response, not all stressors similarly affect cortisol and some do not do so at all (e.g. Biondi & Picardi, 1999).

To elucidate this variability in outcomes, Dickerson and Kemeny (2004) reported a meta-analysis of over 200 studies stratified by methodology and stressor characteristics. Results showed cortisol responses unrelated to task duration and cortisol levels unaffected by tasks involving exposure to noise or emotion induction. On the other hand, cortisol rose significantly in response to both public speaking (d = 0.20, p < .01) and cognitive challenges (d = 0.39, p < .01) and did so to an even larger degree when, as with the TSST, these two task types were administered in combination (d = .87, p < .01). Regarding properties of the experimental stimuli, cortisol responses were greatest when stressors were least controllable (d = .52, p < .01) or involved social evaluative components (e.g., monitored performance or negative social comparison; d = .67, p < .01). Thus, results of the meta-analysis highlight uncontrollability and

social evaluative threat (tasks involving public speaking or cognitive challenges including a social evaluative component) as those with the most appreciable influence on HPA response.

1.3 CHRONIC STRESS STUDIES

Chronic stressors are those that either persist or cause distress over an extended period of time. As in the acute stress literature, studies of chronic stressors and HPA activity have examined a wide variety of circumstances, employed diverse cortisol metrics, and generated mixed findings. For instance, some early studies found cortisol output lowered in the context of chronic stress (Bourne, Rose, & Mason, 1967; Friedman, Mason, & Hamburg, 1963; Yehuda, Teicher, Trestman, Levengood, & Siever, 1996; Yehuda et al., 1995; Miller, Chen, & Zhou, 2007; Vedhara et al., 2002; Seedat, Stein, Kennedy, & Hauger, 2003), while other studies have found either unaltered or heightened HPA activity among similarly exposed individuals (Lemieux & Coe, 1995; Pitman & Orr, 1990; B. B. Arnetz et al., 1987; Baum, Gatchel, & Schaeffer, 1983; Schaeffer & Baum, 1984; Kosten, Jacobs, & Mason, 1984; Yehuda et al., 1995).

Miller, Chen & Zhou (2007) conducted a meta-analytic review of 107 studies to determine whether variation in HPA findings could be traced to characteristics of the stressors or attributes of study samples. In this meta-analysis, stressors and sample characteristics were compared with respect to several indices of HPA activation. Chronic stress characteristics included time since stressor onset (in months), nature of the threat (physical, social, or traumatic), emotions elicited by the stressor (shame or loss), controllability of the stressor (controllable or uncontrollable), and psychiatric characteristics of the sample (inclusion of patients with PTSD, Major Depressive Disorder (MDD), and subjective distress). All categories

of stressors were associated with elevated cortisol levels in the afternoon and evening, but differed on other HPA metrics. Stressors involving physical harm, trauma, uncontrollability and loss were also characterized by diminished morning cortisol, reduced dexamethasone suppression (the extent to which cortisol release is suppressed following administration of a synthetic glucocorticoid), and, with the exception of loss, greater total daily output. A generally similar pattern of HPA activity was found for stressors rated as high in their potential to evoke subjective distress, whereas social threats (potential to diminish one's social standing or role, such as divorce or job loss) were found to occasion higher morning cortisol levels and unimpaired dexamethasone response.

1.4 DEPRESSION AND HPA ACTIVITY

Stress has long been implicated in risk for depression, and much research has focused on HPA activity in depressive disorders. Two indices of HPA activity predominate in the depression literature, the dexamethasone suppression test and the CAR. The former is the most frequently reported measure of HPA activity in depression. Most studies report individuals with depression to have a decreased sensitivity to dexamethasone or dexamethasone plus CRH administration (i.e., weaker suppression of cortisol release), and this association is stronger in more severe forms of depression (Nelson & Davis, 1997; Cowen, 2010; Kunzel et al., 2003; Zobel et al., 2001). Improvement of depressive symptoms tends to be accompanied by a normalization of response to dexamethasone challenges, and persistence of an impaired response has been found to predict relapse among remitted patients (Ribeiro, Tandon, Grunhaus, & Greden, 1993).

In contrast to dexamethasone challenge studies, literature on the awakening response in depressed patients has yielded mixed results, with some studies showing a greater awakening response (Bhagwagar, Hafizi, & Cowen, 2005; Pruessner, Hellhammer, Pruessner, & Lupien, 2003) and others a dampened response (Stetler & Miller, 2005; Huber, Issa, Schik, & Wolf, 2006). Also unlike dexamethasone challenges, where the cortisol response tends to normalize with symptom improvement, altered CAR has been shown to persist in depressed patients following remission of symptoms (Cowen, 2010; Bhagwagar, Hafizi, & Cowen, 2003). Recovered patients are also reported to have significantly higher morning cortisol levels than age- and gender-matched controls (Bhagwagar et al., 2003). The observation that normalization of HPA response to dexamethasone tends to accompany improvement of depressive symptoms and that failure to do so presages unfavorable clinical outcomes, in conjunction with limited evidence that familial risk for depression is associated with altered HPA activity (Mannie, Harmer, & Cowen, 2007), has prompted speculation that abnormalities of HPA function are, in part, causally related to the development of depression.

Because depression and exposure to psychological stressors are both associated with altered HPA activity, and stress is known to predict depressive symptomatology, it may be asked whether attributes of stressors that are most strongly related to HPA activity are analogous to the types of stressors that are most strongly associated with risk for depression. Particularly reminiscent of the stress literature are the psychological properties of depression-related life events, many of which tend to entail either loss of control (e.g. feelings of entrapment) or threats to actual or perceived social standing (e.g. humiliation). As reviewed earlier, acute stressors that are uncontrollable or incorporate social evaluative threats elicit an immediate rise in cortisol, and similarly characterized chronic stressors are associated with varying alterations in HPA activity.

Taken together, then, stressors that place an individual in a position of diminished control or that erode aspects of one's social position relative to others, are related to altered HPA activity in both acute (experimental) and chronic stress studies and to risk for depression.

One framework for understanding depression emphasizes social dominance in interpersonal behavior. It has been suggested that many symptoms of depression are analogous to the behaviors and cognitions of individuals occupying positions subordinate to the authority of higher ranked (dominant) others (e.g. Gilbert, 2000; Sloman, Gilbert, Hasey, 2003; Watson & Andrews, 2002; Gilbert & Allan, 1998). Hierarchical relationships in humans have been observed even in very young children, and humans preferentially process and remember relationships based on asymmetries of power (Strayer & Trudel, 1984; Zitek & Tiedens, 2012). Whereas dominant individuals are more active in interpersonal engagement, such that they speak first, talk more, and interrupt, initiate touch, make eye contact, and gesticulate more often, persons of lesser (subordinate) position talk less, avoid eye contact, keep more "closed" body postures, and defer to others when challenged (Hareli, Shomrat, & Hess, 2009; Hall, Coats, & LeBeau, 2005; Jayagopi, Hung, Gatica-Perez, & Yeo, 2009). Dominance relationships not only characterize human interpersonal behavior but are a common feature of sociality across mammalian taxa and, especially in non-human primates, comprise a primary axis of inter-individual behavior. As in humans, non-human primates form dominance relationships; these are defined by the outcomes of antagonistic encounters, wherein one animal (dominant) habitually defeats another (subordinate). Further, dyadic interactions among non-human primates can be aggregated across animals to describe a dominance hierarchy, which once established, is relatively stable and may be maintained less by physical aggression than by ritualized forms of agonism (Gilbert, 2000). Because symptoms of depression that are reminiscent of social

subordination have been related to altered HPA activity in humans, altered HPA function might also be expected to accompany social subordination in non-human primates.

1.5 SOCIAL STATUS AND HPA ACTIVITY IN NON-HUMAN PRIMATES

HPA activity has long been studied in relation to primate social dominance, although findings are not entirely consistent. Some studies have found subordinate monkeys to have higher baseline cortisol activity (e.g. Sapolsky 1989; Shively, Laber-Laird, & Anton, 1997; Sloman & Gilbert, 2000; Shively et al., 2005), while others report higher baseline cortisol in dominant animals (e.g. Ziegler, Scheffler, & Snowdon, 1995; Abbott, Saltzman, Shultz-Darken, & Smith, 1997; Saltzman, Schultz-Darken, Wegner, Wittwer, & Abbott, 1998; Cavigelli et al., 1999; Kimura, Shimizu, Hayashi, Ishikawa, & Ago, 2000; Ginther, Ziegler, & Snowdon, 2001), and still others find no differences as a function of social dominance (e.g. Suomi et al., 1989; Saltzman et al., 1991; Bercovitch & Clark, 1995; Smith & French, 1997; Morgan et al., 2000; Stavisky, Adams, Watson, & Kaplan, 2001). Although these studies vary on a variety of dimensions, differences in social structure may influence the relationship between dominance and cortisol in monkeys (Czoty et al., 2009; Abbott et al., 2003; Sapolsky, 1992). Normatively, in old world monkeys (which are most closely related to humans), subordinate animals tend to show higher cortisol levels than dominants (Abbott, 2003). In addition, among old world monkeys, subordinates exhibit increased cortisol activity, compared to dominants, across a variety of HPA metrics, including adrenal size (e.g. Kaplan et al., 2005; Shively & Kaplan, 1984), sensitivity to dexamethasone administration (e.g. Sapolsky, Alberts, & Altmann, 1997; Kaplan et al., 2010), and baseline serum cortisol levels (e.g. Sapolsky et al., 1997).

1.6 HUMAN SOCIAL STATUS

The closest apparent human parallels to primate dominance relationships and hierarchies, at least methodologically, are found in observational studies of children and sociometric rankings of dominance in classroom and playground settings (Hawley, 1999). Socially dominant toddlers, preschoolers, and kindergarteners employ agonistic strategies to obtain and control resources (e.g. toys, attention), much as socially dominant monkeys prevail in agonistic interactions for resource acquisition (Hawley, 1999; Russon & Waite, 1991; Strayer & Trudel, 1984). Adult dominance interactions have been examined in experimental social psychology as well, but primarily in order to investigate situational influences on competitive interactions rather than as stable characteristics of individuals (e.g. Burgoon & Dunbar, 2000; Wirth, Welsh & Schultheiss, 2006; Dunbar & Burgoon, 2005). Individual differences in enduring characteristics (or dispositional attributes), on the other hand, have been studied by trait researchers for many decades. One well-established model is the interpersonal circumplex, a framework that aligns personality characteristics of a social nature along two orthogonal axes, commonly termed control and affiliation (Wiggins & Broughton, 1991; Traupman et al., 2009). The axis of control is bounded by traits of dominance and submissiveness, whereas affiliation extends from hostility to friendliness (Traupman et al., 2009). Where individuals are positioned in the circumplex influences their social interactions by the manner in which they relate to others (more dominantly or submissively, hostile or friendly; Traupman et al., 2009). The two axes of the interpersonal circumplex are analogous to commonly studied components of primate social interactions, namely dominance and affiliation.

Another prominent theory of individual differences is the five factor model (FFM), which identifies five major trait dimensions: Extraversion, Neuroticism, Openness to Experience,

Agreeableness, and Conscientiousness (McCrae and Costa, 2010). Because measures of Extraversion have been aligned with dominance and measures of Agreeableness with affiliative dispositions, the circumplex and trait models appear reconcilable, at least with respect to trait representations of dominance and affiliation (McCrae & Costa, 1989; Trapnell & Wiggins, 1990). Thus, dominance has been variously conceptualized in humans as an enduring disposition to achieve positions of power in relation to others (Pratto, Sidanius, Stallworth, & Malle, 1994; Anderson & Summers, 2007), as an ability to prevail in resource acquisition (Hawley, 1999), and as enactment of behaviors aimed at controlling one's environment and influencing other people (Gray, Jackson, & McKinlay, 1991b).

In addition to the foregoing models of dominance rooted in individual differences, animal dominance hierarchies have often been compared to social stratification in human populations. Socioeconomic status (SES) is generally taken as an indicator of an individual's relative standing, or position, in society, as influenced by social and economic forces that engender differential access to material resources and their accumulation (Lynch & Kaplan, 2000; Adler et al., 1994). Most operational definitions of SES entail measurement of individual attributes such as income, education, and occupational grade (Lynch & Kaplan, 2000; Adler et al., 1994; Lupien, King, Meaney, & McEwen, 2001), and as these indicators are correlated but not fully overlapping, SES is often indexed as a composite of these three variables (Adler et al., 1994; Gallo & Matthews, 2003). In the behavioral sciences, socioeconomic differences have been of interest for their relationship to a range of life outcomes (see Adler et al., 1994; Marmot & Shipley, 1996). In addition, subjective measures of SES, or individuals' perceptions of their own social positions relative to others, overlap with objective socioeconomic indicators and often predict (sometimes independently) the same life outcomes associated with conventional

measures of SES (Adler, Epel, Castellazzo, & Ickovics, 2000; Manuck, Phillips, Gianaros, Flory, & Muldoon, 2010; Ostrove, Adler, Kuppermann, & Washington, 2000).

1.7 HUMAN SOCIAL STATUS AND HPA ACTIVITY

Given that both social dominance and SES have been conceptualized as human analogues of social status in monkeys, and that social status in monkeys has been related to HPA activity, it might be asked whether social dominance and/or SES are related to HPA activity in humans. Among investigations addressing this topic, far more studies examine HPA function in relation to SES than interpersonal dominance. Among the few studies relating social dominance to cortisol, outcomes are mixed, operational definitions of dominance vary, and no studies have indexed dominance with reference to the interpersonal circumplex. Among reported findings, lower ranked tennis players in one study (Booth, Shelley, Mazur, Tharp, & Kittok, 1989), but more dominant men (as indexed by peer rankings) in another, tended to have the highest cortisol levels (Decker, 2000), and still other studies find no relationship between dominance and cortisol (Hellhammer, Buchtal, Gutberlet, & Kirschbaum, 1997; Gray, Jackson, & McKinlay, 1991a). Further complicating interpretation, these investigations vary greatly by source (saliva or blood) and timing of cortisol samples. Indicators of dominance also differ appreciably, as for instance by rank on an athletic team (Booth et al., 1989), peer ratings (Decker, 2000; Hellhammer et al., 1997) or personality inventory (Gray et al., 1991a). In a study of adolescents, cortisol levels were found to vary by dominance ratings, but inconsistently across several activity domains (scholastic, peer, and sports hierarchies, and by sex; West, Sweeting, Young, & Kelly, 2010). Finally, studies of extraversion, a personality trait often linked to dominance (as described

above), have similarly shown this trait inconsistently correlated with HPA functioning. For example, low extraversion was associated with low afternoon plasma cortisol levels in one study (LeBlanc & Ducharme, 2005), but in another, proved unrelated to salivary cortisol levels collected throughout the day (Schommer, Kudielka, Hellhammer, & Kirschbaum, 1999). In sum, no clear conclusions regarding the relationship between interpersonal dominance and HPA functioning can be drawn from the available literature, owing in part to the small number of studies, wide range of cortisol sampling procedures and dominance metrics, and inconsistent or null findings.

The literature on SES and HPA activity is much larger than that for dominance, although studies here also differ considerably in measurement and sample characteristics. Despite these differences, studies using composite measures incorporating at least two socioeconomic indicators (e.g. current or lifetime income, educational attainment, occupational grade, or accumulated wealth) have commonly found low SES associated with altered HPA activity, as evidenced by higher total salivary cortisol output (Cohen et al., 2006), a flatter diurnal rhythm (Kumari et al., 2010; Agbedia et al., 2011; Hajat et al., 2010; S. Cohen, Schwartz, et al., 2006), lower total morning and afternoon cortisol (Hajat et al., 2010; Brandtstädter, Baltes-Götz, Kirschbaum, & Hellhammer, 1991), and higher evening levels (in women; Gustafsson, Janlert, Theorell, & Hammarstrom, 2010). Similar results have been reported in some studies relying on a single SES indicator, where low SES was associated with (1) higher total cortisol output, irrespective of sampling method (Chen, Cohen & Miller, 2010; Miller et al., 2009; Li, Power, Kelly, Kirschbaum, & Hertzman, 2007; Arentz et al., 1991; Evans & English, 2002; Evans & Kim, 2007, (2) a flatter diurnal rhythm (Ranjit, Young, & Kaplan, 2005), and (3) lower total waking and afternoon cortisol levels (Dowd et al., 2011; Chen et al., 2010). Nonetheless, other

studies using single socioeconomic indicators have reported opposite (Dowd et al., 2011; Lupien, King, Meaney, & McEwen, 2000; Li et al., 2007) or null findings (Agbedia et al., 2011; Rosmond & Bjorntorp, 2000; Miller et al., 2009; Dowd et al., 2011; Steptoe, Cropley, Griffith, & Kirschbaum, 2000; West et al., 2010) across a range of cortisol metrics. Also, studies of the awakening response have yielded mixed and largely negative results. Although a few investigations have shown single socioeconomic indicators to covary inversely with CAR (Kunz-Ebrecht, Kirschbaum, Marmot, & Steptoe, 2004; Wright & Steptoe, 2005; Gustafsson et al., 2010), one found the opposite (Ranjit et al., 2005) and all others reported null effects (Wright & Steptoe, 2005; Steptoe, Brydon, & Kunz-Ebrecht, 2005; Steptoe et al., 2003; Miller et al., 2009; Agbedia et al., 2011; Kumari et al., 2010).

While the literature linking SES to HPA function is large and diverse, an association of low SES with possible indicators of HPA dysregulation (increased total and bedtime cortisol levels, decreased diurnal rhythm, lower morning and afternoon levels) seems somewhat consistent across studies using composite socioeconomic indices and is supported (albeit with mixed results) in studies of single socioeconomic variables. Nonetheless, effect sizes are small, at best, and variable. Estimating any true strength of association with SES is also impeded by probable deficiencies in cortisol sampling procedures. Thus, while protocols entailing multiple collections over multiple (e.g. 3-4) days, depending on the metric of interest, have been recommended for observational studies (Stewart & Seeman, 2000), the great majority of studies cited above (23 of 30) sampled cortisol on a single day and most (19) collected fewer than 5 samples per participant. Because of the varied number of samples obtained, studies of diurnal rhythm also differ widely in the duration of intervals between successive measurements. Altogether, these considerations suggest that, despite several positive findings, associations of

SES with variation in HPA dynamics have been imperfectly studied to date and, in future work, would benefit from application of more intensive measurement protocols.

1.8 SUMMARY

To summarize the foregoing discussion, stressors that are uncontrollable or socially threatening exert the most appreciable influence on HPA functioning, both acutely and in relation to chronic adversities. Analogous states are prominent in depression, a disorder in which altered HPA activity figures significantly. Diminished control and lower social standing are also states reminiscent of positions of subordination, and in species of non-human primates most closely related to humans social subordination has been associated consistently with HPA activity. These findings invite speculation that an analogous status construct might be related to HPA function in humans, either as personal attributes or in socioeconomic stratification.

Unfortunately, very few studies have examined the relationship of interpersonal dominance to HPA functioning, and the wide range of cortisol sampling protocols reported and the diversity of dominance metrics used make this small literature difficult to interpret. The literature linking SES to HPA activity is much larger, although here too studies differ widely in measurement and sample characteristics**.** In addition, many of these studies report no association between SES and HPA functioning and among those which do, the strength and direction of this association varies greatly. Altogether, these mixed results may be partially explained by methodological inconsistencies in cortisol sampling protocols and dominance metrics.

The preceding review suggests that a more thorough investigation of the relationship between social status and HPA function in humans is warranted. Accordingly, the purpose of the

present study is to reexamine relationships of the two conceptualizations of status with HPA functioning. We hypothesize that relative to participants higher in status [conceptualized as both trait dominance (hypothesis 1) and SES (hypothesis 2)] individuals of lower status will exhibit an altered awaking response; a more blunted diurnal slope; and higher total cortisol output across the day.

2.0 RESEARCH DESIGN AND METHODS

2.1 PARTICIPANTS

Data from a total of 501 participants were obtained from the Adult Health and Behavior study-phase 2 (AHAB-2) project, which provides a registry of behavioral and biological measurements collected on healthy mid-life male and female community volunteers (30-54 years of age) over the course of six study visits. AHAB-2 participants were recruited via mass mailings to southwestern Pennsylvania communities (primarily Allegheny County). All participants were proficient in English, worked a minimum of 25 hours per week, and had completed at least 8 years of schooling. Study exclusions included: (1) medical or psychiatric diagnoses, including cardiovascular, lung, liver or kidney diseases, insulin treatment, cancer, severe hypertension, and psychotic or other chronic medical conditions; (2) use of psychotropic, antihypertensive, antianginal, antiarrhythmic, or glucocorticoid medications; (3) consumption of more than 35 alcoholic drinks per week, (4) inability to participate in a magnetic resonance imaging scan; and (4) nightshift work. The AHAB-2 project collected variables related to subjects' demographic and physical characteristics, medical history, stress exposure, childhood environment, social functioning, personality, psychopathology, lifestyle, cognitive performance, and cardiovascular, central nervous system, immune and neuroendocrine functioning.

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2.2 MATERIALS

2.2.1 Trait dominance

Trait dominance was measured using items of the NEO Personality Inventory -Revised (NEO-PI-R; Costa & McCrae, 1992) to construct the axes of the Interpersonal Circumplex (IPC) for derivation of dominance scores (Trapnell & Wiggins, 1990; Wiggins & Trobst, 1997). The NEO-PI-R is a 240-item measure of FFM personality traits (Extraversion, Agreeableness, Conscientiousness, Neuroticism and Openness to Experience), which has shown high internal consistency, test-retest reliability, and convergent and discriminant validity (McCrae & Costa, 2010). Previous research has found IPC octant scales derived from the NEO-PI-R to conform closely to circumplex structure in four separate analyses, and to show convergent and discriminant validity in the form of expected correlation patterns between self-reports and spouse ratings (Traupman et al., 2009). The IPC is comprised of two major dimensions, control (dominance versus submissiveness) and affiliation (friendliness versus hostility), and each octant reflects a blend of these dimensions. Agreeableness and extraversion traits from the FFM are considered blends of the IPC dimensions of affiliation and control. In order to obtain these two IPC dimensions, items from the agreeableness and extraversion domains of the NEO-PI-R were used to obtain IPC octants. Six NEO-PI-R items comprise each octant, chosen from previous studies based on item content (internal consistencies of these 6-item octant scales have shown a median *a* value of .68; Traupman et al., 2009). Octant scales are then combined in order to obtain scores for dominance and affiliation.

2.2.2 Socioeconomic status (SES)

Socioeconomic status was obtained using a variety of metrics, which were derived from the MacArthur Network on SES and Health. For the purposes of the present analysis, we utilized income, education, wealth, and occupational grade as measures of objective SES, and considered subjective SES (MacArthur SES Ladder) separately.

Income. Pre-tax income was assessed as annual household earnings on a 15-point scale. This scale ranges from < $10,000/year (or $0-833/month) to > $185,000/year (or more than $15,417/month). Thirteen income brackets between these values were spaced in $15,000 increments. Income was adjusted by household size by the formula: $\frac{m}{\sqrt{h}}$ where *m* is the midpoint of the bracketed range of annual earnings (the highest annual earning midpoint was scaled to 25% above $185,000), and *h* is the number of occupants in the household (see Gianaros, Marsland, Sheu, Erickson, & Verstynen, 2012).

Education. Education was assessed as years of schooling on a scale of 1 to 24+ years.

Wealth. Wealth was calculated as the dollar value held in all reported bank accounts, stocks or bonds, minus any existing debt, each measured on bracketed 9-point scales and converted to an estimated dollar amount.

Occupation. Participant occupation was graded according to the Hollingshead index of occupations (Hollingshead, 1958): 1 (laborers, service workers), 2 (unskilled workers), 3 (machine operators and semiskilled workers), 4 (skilled manual workers, craftsmen), 5 (clerical and sales workers), 6 (technicians, semiprofessionals), 7 (managers, minor professionals), 8 (administrators, lesser professionals), and 9 (higher executives, major professionals).

MacArthur Scale of Subjective Social Status. Subjective SES is defined as an individual's perception of their social position relative to others, and lower perceived status has been found to be associated with greater cortisol reactivity to stress (Adler et al., 2000), and higher cortisol awakening response (Wright & Steptoe, 2005), even after adjusting for correlated variation in objective SES. Subjective SES was assessed using the MacArthur Scale of Subjective Social Status (http://www.macses.ucsf.edu/). This measure depicts relative social standing as the ten rungs of a pictured "social ladder" on which participants are asked to place an "x" on the rung that best depicts where they stand in relationship to others in the U.S. population by reference to income, education, and occupational prestige.

2.2.3 Cortisol

Salivary cortisol was collected at home by participants on four monitoring days (3 work days, and 1 non-work day). On each day, participants were prompted by an electronic diary to collect 5 saliva samples, the first immediately upon awakening, at 30 minutes after awakening, 4 and 9 hours after awakening, and at bedtime. For each sample, participants were instructed to chew on a cotton swab for 2 minutes while they answered questions on the electronic diary. Participants were asked to place the cotton swab into a salivette and record a six-digit code that appeared on the electronic diary onto the salivette tube in order to ensure compliance. Each completed cortisol sample was placed into a plastic bag marked "used" and stored in the participant's refrigerator until their next visit to the lab. Cortisol, expressed as nmol/L, was assayed in duplicate using a time-resolved fluorescence immunoassay with a cortisol-biotin conjugate as a tracer with a sensitivity of 0.43 nmol/L in the laboratory of Clemens Kirschbaum [Dresden,

Germany]. The intra-assay coefficient of variability was < 10 percent, and inter-assay coefficient of variability was <12 percent.

2.3 PROCEDURE

Participants in the AHAB-2 study attended six sessions, each lasting 3-4 hours at either the Behavioral Physiology Laboratory (Manuck, director) or the Behavioral Medicine Research Group (Kamarck, director) within a period of generally one to two months. At the first visit (Behavioral Physiology Laboratory), written informed consent, basic demographic and anthropomorphic measurements were obtained, as well as a fasting blood draw, followed by several standardized tasks of psychological, behavioral, and cognitive functioning. Following the second session (Behavioral Medicine Research Group), participants engaged in multiple days of ambulatory blood pressure monitoring, physical activity monitoring and cortisol assessment prompted by an electronic diary device (instructions were given at visit 2). Participants returned their cortisol collection packets and other ambulatory monitoring devices at visit 3, and completed additional questionnaires during visits 3 (Behavioral Medicine Research Group) and 4, including the NEO-PI-R (Behavioral Physiology Laboratory). At visit 5 (Magnetic Resonance Research Center), participants engaged in a functional magnetic resonance imaging protocol, and at visit 6 (Behavioral Physiology Laboratory and Ultrasound Research Laboratory) a carotid artery ultrasound, as well as additional questionnaires.

3.0 ANALYSES

3.1 DATA REDUCTION

3.1.1 SES

The distribution of wealth was highly skewed, with a median value of $2,750. Because standard transformations failed to satisfactorily normalize this variable, wealth was not included in subsequent analyses. A principal components analysis confirmed that each objective measure of SES (income, education, and occupational grade) aligned dimensionally on a single factor with an eigen value of 1.64, explaining 54.76% of the variance, with the following factor loadings: income (.79), years of education (.64), occupational grade (.77). These three indicators were standardized and averaged to obtain a unit-weighted composite score of objective SES.

3.1.2 Cortisol

Cortisol values were log transformed, and these values were used in the calculation of diurnal slope. Raw cortisol values were used for percent increase in cortisol from waking to 30 minutes, and total cortisol output.

Cortisol Awakening Response (CAR). CAR was calculated as percent change from baseline to 30 minutes after awakening. Cortisol samples not taken within 10 minutes of the 30 minute post-awakening instruction were excluded.

Diurnal slope. Diurnal slope was calculated by fitting a regression line for each participant where successive cortisol measurements are predicted from hours since awakening (e.g. Matthews, Schwartz, Cohen & Seeman, 2006) to yield the linear equation: $\hat{Y} = a + bx$,

where *a* denotes the intercept and *b* the slope of the diurnal rhythm. The second cortisol sample was excluded from the calculation of diurnal slope and AUC in order to minimize the influence of the awakening response on these estimations.

Area under the curve (AUC). AUC was calculated as described by Pruessner et al., (2003) as follows:

$$\sum_{i=0-3} \frac{(c_i + c_{i+1})(t_i + t_{i+1})}{2}$$

where *c* refers to a cortisol value, *t* to the number of hours since awakening, and *i* the sample collection number (the second cortisol sample was excluded as described above).

Cortisol exclusions and outliers. On examining preliminary distributions, a threshold value of 60 nmol/L was set to exclude cortisol outliers falling above this value. Additionally, cortisol values falling below .3 nmol/L were excluded, as they fall below estimated lowest reliable detection levels for cortisol. Only participants with complete cortisol data for at least two days of collection were included in our analyses.

3.1.3 Preliminary analyses

Distributions for all study variables were examined in order to verify assumptions of normality. In addition, zero-order correlations were calculated in order to examine simple bivariate associations between study variables.

3.1.4 Primary analyses

Hierarchical regression analysis was used in order to determine whether trait dominance or SES indicators account for significant variability in CAR, diurnal slope or AUC. In order to test hypothesis 1, traditional demographic covariates which could likely be associated with status or HPA function (age, sex, and race) were entered in step 1, followed by trait dominance in step 2 for each of the three cortisol indices. For hypothesis 2, demographics were entered in step 1, followed by an SES parameter (objective SES composite, subjective SES) in step 2 for each of the three cortisol indices.

Because elevated total daily cortisol output and awakening response have been documented in current smokers compared to former and never smokers (e.g. Badrick, Kirschbaum, & Kumari, 2007), the association between smoking status and our study variables (trait dominance, SES, and all cortisol indices) were examined in order to determine whether smoking status should be included as a covariate to be entered along with demographic variables in step 1 of each analysis.

Additional analyses examined whether an interaction exists between sex and all status variables (trait dominance, objective and subjective SES) for each of the cortisol indices using hierarchical linear regression analyses entering the main effects of sex and a status variable at

step 2 and the product of these variables at step 3. Finally, if hypothesis 1 and 2 are both significant for a given cortisol index, additional hierarchical linear regression analyses will test whether these constructs reflect the same or different associations by entering them simultaneously at step 2.

Because preliminary analyses showed day-specific effects of status and cortisol indices, mixed-design analyses of variance with status (either dominance or SES) as a continuous between-subjects variable and day type (workday versus non-workday) as a categorical within-subjects variable were conducted in order to examine possible interactions of status by day type for each cortisol index. In order to accommodate the continuous between subjects factor (status), these analyses were run in the context of a general linear model.

4.0 RESULTS

4.1 PARTICIPANT CHARACTERISTICS

Summary statistics for demographic characteristics, status, and cortisol-related variables are listed in Table 1. After removing cortisol outliers (>60 nmol/L, <.3 nmol/L) and excluding data in which cortisol samples could not be matched to sampling times, or awakening cortisol values were missing, 494 of the initial 501 participants in our sample remained. An additional 21 participants were excluded because they were missing complete cortisol data for at least two days of collection, yielding a final sample of 473 for our analyses. Participants were 43 years of age on average, 53% female, 83% white, and 16% were current smokers. Average annual income adjusted for the number of individuals in the household was $49,892, and participants had completed, on average, 17 years of education. Mean occupation code was 6.35, which corresponds to the technical/semiprofessional grade of employment according to the Hollingshead index. The mean subjective SES score in this sample was 6.12 (± 1.50) and mean dominance score was 1.82 (± 11.54). Mean increase in cortisol levels following awakening was 60% (± .60) on averaged work days and 39% (± .75) on the non-work day. Diurnal slope in cortisol levels showed a mean decrease of 0.04 nmol/L/hour across both the averaged work days and non-work day, and total cortisol output assessed by AUC was somewhat higher for the mean of work days (7.44 ± 2.12 nmol/L) than the single non-work day (7.01 ± 2.63 nmol/L).

4.2 COMPARISONS OF INDIVIDUAL DAYS OF CORTISOL MEASUREMENT

Table 2 presents between-day correlations of cortisol levels at each time of measurement, and table 3 displays correlations among cortisol indices (CAR, slope, AUC), also on each of the sampling days. For than the majority of cortisol samples, cortisol levels on the non-work day correlated less strongly with samples taken on the three workdays (average r= .22) than workday samples correlated with each other (average r= .33). Correlations were strongest across all four days for AUC (average r= .46) followed by slope (average r=.35) and then CAR (average r = .22), and like cortisol levels, most indices calculated on the non-work day correlated less strongly with indices calculated on each of the three workdays (average r= .29) than indices on workdays correlated with one another (average r= .38).

Additionally, a series of repeated measures Analyses of Variance (ANOVA) was conducted to examine whether mean cortisol levels differed significantly between days. As presented in table 4, cortisol levels differed between days at awakening, 30 minutes after awakening, and 9 hours after awakening (but not at 4 hours post-awakening or bedtime) exhibiting a main effect of days. Post hoc comparisons showed that for each sampling time, values obtained on the single non-workday were lower than each workday, and the latter values did not differ from each other. Another series of ANOVAs tested for differences in the actual time (in 24-hour clock) that samples were collected and found collection times to differ across days for all five samples (table 5). Post hoc comparisons here showed that at least one workday differed from the other workdays at each sampling time, although the differences were trivial (less than 10 minutes for each). In contrast, for all but the bedtime sample, non-workday time of collection was significantly later than each of the workdays by over an hour. The latter finding suggests that the lower cortisol levels described above (table 4) on the non-workday might reflect

a right-shifted displacement of cortisol measurements along the diurnal curve. Thus, the lower non-workday cortisol levels, relative to workday values, may be confounded by a later waking time on the non-workday. Indeed, differences between average workday cortisol levels and non-workday levels correlated inversely with differences in time of collection for all but the awakening sample [awakening (r = -.06, *p*= .25), 30 minutes (r= -.28, *p*=.00), 4 hours (r= -.18, *p*=00), 9 hours (r= -.10, *p*= .03), bedtime (r=.16, *p*=.001)].

A final series of repeated measures ANOVAs was performed to examine whether cortisol indices (CAR, diurnal slope, AUC) differed across days (table 6). Mean CAR and AUC, but not slope, varied significantly between days of measurement. Post-hoc comparisons showed the mean CAR to be smaller on the single non-workday relative to each workday, while workdays did not differ from each other. Post-hoc comparisons of AUC, on the other hand, found the single non-workday to differ from two of the three workdays, two of which differed from each other. Because cortisol indices and the levels from which they were derived differed in general between the non-workday and workdays, workday indices were averaged and non-workday indices were treated separately in subsequent analyses testing the main study hypotheses.

4.3 CORRELATIONS AND MEAN COMPARISONS

Before proceeding to tests of study hypotheses in linear regression, we examined zero order correlations and mean comparisons between principal study variables.

4.3.1 Demographic and status variables

Older age was associated with higher income and lower education, but with no other SES indicator or dominance (table 7). As shown in table 8, relative to females, males had higher composite SES, income, and years of education (but did not differ in occupational status) reported higher subjective SES, and obtained marginally lower (p = .05) dominance scores. There were significant race differences in mean levels of all SES measures, but not in dominance scores (presented in table 9) such that white participants had higher composite SES, income, occupational status, and years of education, and reported greater subjective SES, relative to non-white participants. Finally, current smokers had lower composite SES, income, occupational status and subjective SES than non-smokers, but did not differ in years of education or levels of dominance (table 10).

4.3.2 Cortisol indices

As shown in table 11, a higher CAR was associated with a flatter diurnal slope and smaller AUC on averaged workdays and the single non-workday. On the other hand, a flatter diurnal slope was associated with a smaller AUC on the non-workday, but did not associate with AUC derived from average workdays. In addition, all bivariate correlations between indices derived from the averaged workdays and those derived from the single non-workday were significant: CAR (r=.23, p<.001), slope (r=.46, p<.001), AUC (r=.49, p<.001).

4.3.3 Demographic and cortisol indices

As shown in table 12, older age was associated with a flatter average workday diurnal slope and with a higher non-workday CAR, but was unrelated to AUC, non-workday slope, or workday CAR. Also, males had a marginally smaller workday CAR (p= .06) and significantly steeper workday diurnal slope than females, but did not differ on AUC or non-workday CAR or slope (table 13). In addition, white participants had steeper diurnal slopes on both the mean workday and non-workday measurements and greater non-workday CAR, relative to non-white participants, participant race was unrelated to either workday or non-workday AUC (table 14). Finally, non-smokers had steeper non-workday diurnal slopes than current smokers, but did not differ on any other cortisol index (table 15).

4.3.4 Status and cortisol indices

Relationships among status variables are presented in table 16, and status and cortisol relationships are presented in table 17. Higher dominance was associated positively with income and subjective ratings of SES, while subjective SES and all objective SES indicators (education, income, occupation, and composites) were positively correlated with one another (table 16). As presented in table 17, dominance covaried inversely with average workday CAR, as did composite SES, and income and occupational grade separately; these objective SES indicators were associated also with a steeper average workday diurnal slope. Interestingly, in contrast to income and occupation, years of education did not correlate significantly with any workday or non-workday cortisol indices. Also, greater subjective SES was associated with a steeper average workday diurnal slope, but with no other cortisol index. No other significant relationships were

observed. At the level of bivariate correlations, then, predicted relationships between some indicators of status (dominance, composite SES, income, and occupation) were associated with two of the three cortisol indices (CAR and slope), and subjective SES with one (slope). Hierarchical linear regression analyses were next conducted to further examine these relationships adjusting for the influence of demographic covariates.

4.4 EFFECTS OF STATUS ON CAR, SLOPE, AND AUC

4.4.1 Trait dominance

A series of hierarchical linear regressions was performed to determine the effect of trait dominance on each index of cortisol activity after adjustment for age, sex and race (tables 18-20). When entered simultaneously, covariates accounted for 1-3% of variance in all analyses, with the single exception that covariates accounted for 16% of variance in average workday slope. Dominance scores predicted variability in both workday CAR ($F_{1, 297} = 5.27$, $p = .02$) and workday diurnal slope ($F_{1, 366} = 4.85$, $p = .03$), but not workday AUC ($F_{1, 312} = 1.89$, $p = .17$). More specifically, higher dominance scores predicted a smaller workday CAR and steeper mean workday slope, such that a one standard deviation elevation in dominance reflected an 8% lower CAR and a .001 nmol/L/hour greater diurnal slope. Although dominance predicted variability in workday CAR and slope measurements, the magnitude of these effects was small, accounting for 2 and 1% of the variance (respectively) after covariates, and in contrast to workday measurements, dominance scores were unrelated to any of the three non-workday indices: CAR ($F_{1, 374} = .33$, $p = .57$), slope ($F_{1, 385} = .04$, $p = .85$), or AUC ($F_{1, 379} = .56$, $p = .45$). Mixed-design

analysis of variance showed no interaction between dominance and day type for either CAR [*F* (195, 61) = .83, *p*= .82] or slope [*F* (236, 93) = .78, *p*= .92]. In addition, no significant interactions of dominance by age, sex, or race were observed.

4.4.2 Objective SES

Like dominance analyses, hierarchical linear regressions were used to examine the effect of objective SES on each of the cortisol indices (tables 21-23). Because smoking status was correlated with objective SES (and each of its components separately), smoking was included as an additional covariate along with age, sex, and race, in Step 1 of each of these analyses. Covariates accounted for 2-4% of the variance in all analyses with the exception of average workday slope, where covariates accounted for 17% of variance. SES contributed significantly to the prediction of workday CAR ($F_{1, 296}$ = 4.37, *p*=.04), but none of the other workday or non-workday indices: Workday slope ($F_{1, 366}$ = 2.99, *p*=.09), workday AUC ($F_{1, 312}$ = .06, *p*=.81), non-workday CAR ($F_{1, 373}$ = .24, *p*= .39), non-workday slope ($F_{1, 385}$ = .75, *p*=.39), non-workday AUC ($F_{1, 379}$ = .37, *p*=56). Similar to dominance, a higher composite SES was associated with a smaller workday CAR, with a one standard deviation elevation in SES corresponding to an 8% lower cortisol response to awakening. Mixed-design analysis of variance examining possible SES by day type (workday versus non-workday) interactions showed a significant interaction for CAR [*F* (162, 95) = 1.37, *p*= .05], and marginally significant interaction for slope [*F* (187, 144) = 1.24, *p*= .09]. No significant interactions of SES by age, sex, or race were observed.

4.4.3 Secondary analysis of objective SES

Bivariate correlations between individual SES indicators and each of the cortisol indices show that, on average, correlations with income (median r= .11, range = .04 - .15) and occupational grade (median r = .16, range .04 - .28) were higher than those for years of education (median r = .06, range = .05 - .06). In addition, education had shown a lower factor loading (r= .64) than income (r= .79) or occupation (r= .77) in the principal components analysis supporting the composite SES measure, and education correlated less strongly with income (r=.28) and occupation (r= .25) than these two were with each other (r= .43). For this reason, analyses were repeated based on a composite of income and occupation alone (i.e. excluding years of education; tables 24-26).

Objective SES assessed as a mean of the standardized distribution of income and occupation (SES I/O) predicted variability in both workday CAR ($F_{1, 296}$ = 7.44, p = .007) and workday diurnal slope ($F_{1, 366}$ = 10.11, p = .002), but not workday AUC ($F_{1, 312}$ = .35, p = .56). More specifically, higher SES I/O was associated with a smaller workday CAR and steeper workday diurnal slope, such that a one standard deviation higher SES I/O predicted a 10% lower cortisol response to awakening and a .002 nmol/L/hour greater diurnal slope. Conversely, in none of the non-workday analyses was SES I/O related to cortisol: Non-workday CAR ($F_{1, 373}$ = .43, p = .51), slope: ($F_{1, 385}$ = .24, p =.62), AUC: ($F_{1, 379}$ = .04, p = .84). Mixed-design analysis of variance examining possible interactions between SES I/O and day type (workday versus non-workday) found a marginally significant interaction for CAR [F (124, 133) = 1.31, p= .06] and significant interaction for slope [F (136, 195) = 1.38, p= .02]. No significant interactions of SES I/O by age, sex, or race were observed.

4.4.4 Subjective SES

A parallel series of hierarchical linear regressions was conducted to determine the effect of subjective SES on each index of cortisol activity (tables 27-29). As with objective SES, persons of lower subjective SES were more likely to be smokers, so that smoking status was included as a covariate with age, sex, and race entered at Step 1. These covariates accounted for 1-4% of the variance in all analyses, again with the exception of average workday slope, where covariates accounted for 17% of the variance. In none of these analyses did subjective SES contribute significantly to the prediction of cortisol activity (tables 22-24): workday CAR ($F_{1, 296}$ = .07, *p*= .07), workday slope ($F_{1, 366}$ = .38, *p*= .54), workday AUC ($F_{1, 312}$ = .13, *p*= .72), non-workday CAR ($F_{1, 373}$ = .41, *p*= .52), non-workday slope ($F_{1, 385}$ = 1.00, *p*=.32), non-workday AUC ($F_{1, 379}$ = 1.65, *p*=.20). On the other hand, there was a significant sex by subjective SES interaction on average workday slope ($F_{1, 367}$ = 6.43, *p*= .01, β= .38), such that males higher in subjective SES had a steeper workday slope ($F_{1, 166}$ = 4.08, *p*= .045, β-.15), whereas the simple slope in women was not significant ($F_{1, 196}$ = 1.65, *p*= .20, β= .09). No other significant interactions of age or race by status were observed.

4.4.5 Adjusting for wake time

The foregoing analyses showed higher dominance scores and SES I/O associated with a smaller workday CAR and steeper diurnal slope. In follow-up analyses of individual cortisol measurements (e.g. at awakening, +4 and +9 hours, and at bedtime), dominance and SES I/O both covaried positively with awakening cortisol levels only (*β's*= .09; .15, *p's*=.04; .002). Because higher dominance and SES I/O predicted higher initial cortisol levels but not levels

collected at later times, and because a later awakening time associates with lower cortisol levels (r = .13, *p*=.01), it is conceivable that the effects of these indicators of status on CAR and slope are driven by time of awakening. Indeed, higher dominance was associated with a later wake time (r = .09, *p*=.05), hence, wake time was added as a covariate examining the effects of dominance on CAR and slope. On the other hand, higher SES I/O was associated with an earlier wake time (r= -.13, *p*=.01), and although this opposite relationship does not subject SES I/O to the same logic of confounding by wake time as with dominance, we nonetheless extended the re-analysis of SES I/O relationships with CAR and slope to include wake time as a covariate. In all analyses, adjusting for time of awakening collection did not affect significant results (tables 30-31).

4.4.6 Independent effects of dominance and SES

Principal findings of the foregoing analyses were that higher trait dominance and higher SES I/O each associated with a smaller CAR and steeper diurnal slope. In addition, dominance correlated significantly with income, and did so at trend level with the composite SES I/0 (table 16). To determine if dominance and SES I/O were independent predictors of CAR and diurnal slope, we performed a final regression analysis in which, after entry of covariates, dominance and SES were entered simultaneously. In analyses of both CAR and slope, SES I/O remained a significant predictor, and dominance approached significance (*p*'s = .06 and .07, respectively. Though significance eroded slightly, there was little change in effect size ($\Delta\beta$'s= -.01 - .02) for either variable (table 32).

5.0 DISCSUSSION

5.1 SUMMARY

The present study examined the relationship between constructs of status and HPA functioning. Rationale for this study derives from evidence suggesting that conditions of diminished control and threats to social standing, whether evoked by acute or chronic stressors or reflected in symptoms of depression, are salient correlates of HPA activity. Further, these psychological factors are reminiscent of social subordination, which has long been associated with heightened HPA activity in non-human primates, principally old world monkeys. Here, we hypothesized that analogous status constructs, either trait dominance, as a dispositional attribute related to personality (hypothesis 1), or socioeconomic indicators, as reflecting social stratification (hypothesis 2), might be related to HPA functioning in humans. Results of our analyses supported these hypotheses for two of three HPA indices, the cortisol awakening response and the slope of cortisol decline over the waking day. Specifically, higher levels of trait dominance and higher SES were each associated with both an attenuated awakening response and steeper diurnal slope on workdays, but did not extend to measurements obtained on a single non-workday.

These findings confirm a relationship of status and cortisol activity in humans, and for two conceptualizations of status, interpersonal dominance and SES. Regarding the former,

previous work has defined interpersonal dominance operationally in a variety of ways (e.g. peer status, athletic ranking, and personality ratings of extraversion), but with few studies overall and little commonality of cortisol measurement among studies. Further, results of these previous investigations were mixed, making clear conclusions about the relationship between interpersonal dominance and cortisol activity difficult to establish. No studies to our knowledge have examined the relationship between trait dominance specifically and cortisol activity, although as noted, a few examined extraversion, a trait linked to dominance. One of the three previous studies of extraversion in relation to salivary cortisol output reported no association with CAR and another reported no association with slope, though both of these had relatively small samples (n = 33 and 81, respectively; Munafo et al., 2006; Schommer, Kudielka, Hellhammer & Kirschbaum, 1999). On the other hand, a more recent study with a much larger sample (n=381) found higher extraversion associated with a smaller CAR, a result which our findings corroborate (van Santen,Vreeburg,Van der Does, Spinhoven, Zitman, & Penninx, 201). Recall that trait dominance was derived here using items from the extraversion and agreeableness scales of the NEO personality inventory to construct axes of the interpersonal circumplex. As described above, the interpersonal circumplex (IPC) aligns interpersonal behavior on two orthogonal axes – control (dominance vs. submissiveness) and affiliation (hostility vs. friendliness). As extraversion and agreeableness are considered blends of the IPC dimensions of affiliation and control, items from each can be used to construct a circumplex from which dominance scores are derived. Thus, we might expect extraversion to similarly associate with cortisol in our study. Indeed, a secondary analysis indicates that higher scores on extraversion were associated with a smaller CAR (R^2 = .06, β = -.12, p =.046) and steeper workday diurnal slope (R^2 = .18, β = -.14, p =.004).

In contrast to interpersonal dominance, a large and diverse number of studies have investigated the relationship between SES and cortisol activity. Though these studies differ considerably in their indices of SES, sample characteristics, and measurement of cortisol, consistencies across studies using a composite measure of SES suggest that, compared to individuals with higher SES, lower SES is associated with a larger CAR (Cohen et al., 2006) flatter diurnal slope (Kumari et al., 2010; Agbedia et al., 2011; Hajat, et al., 2010; Cohen, et al., 2006) and greater total cortisol output (Gustafsson, et al., 2010). Our results using SES as a composite of income and occupation are consistent with these findings for CAR and slope; however, we did not find an association with total cortisol output (AUC). That education in our sample dilutes the associations between SES and cortisol was unexpected since education is often included in literatures predicting SES-related outcomes. A satisfactory explanation for why education may not be as strongly associated with cortisol is not readily apparent, although as described above, education showed a lower factor loading than income and occupation in a principal components analysis of these variables, and was correlated less strongly with income and occupational grade than these two were with each other.

5.2 STATUS

Trait dominance and SES are often used interchangeably to describe social status in humans, but have previously not been examined together in the same sample. Interestingly, in the current investigation, trait dominance and SES based on income and occupation, were not themselves strongly correlated ($r = .08$, $p = .09$), and when entered simultaneously into statistical models

predicating CAR and slope, effect sizes and significance levels were largely unchanged. Despite the minimal overlap of these two status constructs, our results showed a consistent association of each of these two variables with indices of cortisol activity. That they were related in a similar fashion to cortisol activity suggests these status constructs may possess some common attribute that, although differently expressed in social dominance and socioeconomic status, associated similarly with HPA activity. One characteristic shared by low dominance and low SES is lack of power. Power is typically conceptualized as actual or perceived control over resources, information, decision making ability, or influence over others (Anderson, John & Keltner, 2012). Asymmetries of power occur across a variety of contexts, including interpersonal interactions and socioeconomic stratification, though a lack of power in one context does not imply a lack in power in others. For instance, a study that stratified participants by control over resources found trait dominance to predict personal ratings of power beyond the effect of assigned control over resources (Anderson, John & Keltner, 2012). Thus, an individual low on trait dominance may experience a lack of power in interpersonal situations but not in access to socioeconomic resources, and conversely, an individual low on SES may experience a lack of power in their access to material resources, but not in their interpersonal interactions. In both cases, the lack of power may be what is reflected in an altered HPA profile.

The association between lower power and altered HPA activity may reflect a biological correlate of withdrawal or disengagement in subordinate individuals. In this regard, the HPA system has been contrasted with a second neuroendocrine axis, the sympatho-adreno-medullary (SAM) system, in relation to the types of stress reactions with which each is associated (Henry, 1992). In contrast to SAM activation, which subserves preparation for active coping (e.g. fight or flight responses), HPA activation appears to accompany behaviors indicative of

disengagement or withdrawal from adverse circumstances. These associations have been documented in a number of species (see Koolhaas et al., 1999). For example, when rats are presented with a noxious stimulus (e.g. an electrified prod mounted inside their cage), active coping behavior (burying the prod using cage bedding) is accompanied by high levels of SAM output – norepinephrine, and lower levels of corticosterone (cortisol analogue), whereas withdrawal behavior (freezing) is accompanied by lower levels of norepinephrine and higher corticosterone levels (De Boer, 1990). Additionally, the HPA system has been causally linked to withdrawal behavior, with both adrenalectomy and administration of a corticosterone inhibitor in rats resulting in a reduction in fear-induced freezing behavior (Bohus, Hellhammer, Florin and Weiner, 1987; Roozendaal, Bohus & McGaugh, 1996). Finally, active coping versus passive withdrawal and relative SAM and HPA activity also appear to vary by social rank (see Koolhaas et al., 1999). For example, in hens, SAM activation and physical resistance in response to restraint stress is highest in higher-ranked hens, and HPA activity is highest in lower-ranked hens (Korte, Beuving, Ruesink, & Blokhuis, 1997; Korte, Ruesink, & Blokhuis, 1999). Functionally, withdrawal behavior has been speculated to be advantageous in circumstances where avenues for active coping are not available (more often experienced by lower-ranked individuals) in order to minimize further exposure to the adverse stimulus (Henry, 1992; Koolhaas et al., 1999).

5.3 DIURNAL SLOPE

Cortisol release is under the control of pacemaker cells in the suprachiasmatic nucleus which project to the hypothalamic paraventricular nucleus and initiate the signaling cascade of corticotrophin releasing hormone (CRH) from the hypothalamus, adrenocorticotrophic hormone

(ACTH) release from the pituitary, and finally, cortisol release from the adrenal cortex. In a 24 hour period cortisol release follows a clear diurnal pattern, with levels highest in the early morning hours, falling sharply in the several hours after awakening, and then continuing to decline at a more modest rate through the remainder of the day. A flatter slope in cortisol levels across the day has been associated with greater exposure to chronic stress (Miller et al., 2002), decreased life expectancy among cancer patients (Sephton, et al., 2000), coronary artery calcification (Matthews et al., 2006), and all-cause cardiovascular mortality (Kumari, et al., 2010). Termination of cortisol release is effected by feedback inhibition of CRH following activation of glucocorticoid receptors located in the hippocampus and pituitary gland (Akana et al., 2001; de Kloet et al., 1998). Chronically elevated levels of circulating cortisol contribute to a decrease in glucocorticoid receptor sensitivity, and impaired sensitivity of glucocorticoid receptors has been shown to associate with a flatter diurnal slope (Bradbury et al., 1994; Miyanaga et al., 1990). Thus, heightened cortisol activity associated with subordination may reduce glucocorticoid receptor sensitivity and thereby contribute to the flatter diurnal slope we observed in individuals of lower dominance and lower SES defined by income level and occupational grade.

The diurnal cortisol slope is derived from a number of cortisol measurements across the day, and as such, variations in diurnal slope can be influenced by differences in levels of cortisol at any or all points of measurement from which the slope is derived. Thus, a flatter diurnal cortisol slope might be characterized in a number of ways; lower morning levels, higher evening levels, or both altered morning and evening levels. An examination of the relationship of dominance and SES based on income and occupation with cortisol levels at each time point from which slope is derived in our sample showed higher values of these predictors to associate with

higher cortisol levels at awakening (r's = .12, .20; *p*'s = .02, .001), but not at 4 or 9 hours after awakening or at bedtime. This suggests that in our sample, lower cortisol levels at awakening in lower status individuals accounted for a flatter diurnal slope.

5.4 CAR

The cortisol awakening response (CAR) is an acute rise in cortisol levels (~ 50% increase) in response to awakening that peaks at about 30 minutes after awakening. This acute rise in cortisol is distinct from the diurnal curve of declining cortisol levels over the day, on which it is superimposed, and variation in CAR is uncorrelated (r = -.37, $p>.05$) with cortisol levels across the day (Schmidt-Reinwald et al, 1999; Edwards et al., 2001). Compared to the diurnal cortisol slope, less is known about the mechanisms that alter the cortisol response to awakening, though recent studies have shown alterations in sleep and light exposure to influence magnitude of the CAR (see Figueriro & Rea, 2012). In addition, the interpretation of dysregulation in the awakening response is less clear than for the diurnal slope. For example, chronic stress has been associated with both reduced CAR (Buchanan, et al., 2004; De Vugt, 2005) and increased CAR (Schlotz, et al., 2004; Wüst, et al., 2000), and depression has been associated with a blunted CAR in some studies (Stetler, et al., 2005; Huber, et al., 2006), but an increased CAR in others (Bhagwager et al., 2005; Pruessner et al., 2003; see Fries, Dettenborn & Kirschbaum, 2009). Interestingly, a study examining the differences between workday and non-workday CAR found that a greater workday CAR was accompanied by lower ratings of control on workdays compared to non-workdays (Kunz-Ebrecht, et al., 2004). Thus, the greater CAR in lower status

individuals in our study might similarly associate with lower perceived control in these individuals.

5.5 WORKDAY/NON-WORKDAY DIFFERENCES

It is important to note that our findings with dominance and SES derived from income and occupation were exclusive to workday measurements of both CAR and diurnal slope, and did not extend to cortisol indices obtained on the single non-workday. Mixed-design analyses testing for interactions of status by day type (workday or non-workday) showed significant or marginally significant interactions of SES and SES derived from income and occupation by day type for both CAR and slope. Conversely, there were no significant interactions of dominance by day type for either CAR or slope. Participants woke up significantly later on the non-workday than on each of the three workdays, and because wake time was correlated with waking cortisol levels (a component of each of our HPA indices), we suspected that differences in findings between workdays and the single non-workday might reflect the fact that the sampling frame on the non-workday that was displaced later on the diurnal curve. Indeed, trait dominance and SES based on income and occupation were correlated with both wake time and waking cortisol levels. However, when wake time was entered as a covariate in analyses results were largely unaffected, suggesting that displacement on the diurnal curve did not account for workday/non-workday differences in results.

Another consideration in examining differences in workday and non-workday findings is the greater reliability of assessments on workdays, resulting from the availability of three days of workday measurement and only one non-workday assessment. While associations between status

and cortisol activity were not significant on all individual workdays, effect sizes for these associations were much higher for both CAR (β's= -.08; -.11) and slope (β's= -.04; -.18) on workdays than those obtained on the non-workday (β_{CAR} = .03, β_{Slope}= -.01, .01). Because workday/non-workday differences in results are not likely the result of a displaced sampling frame on the non-workday or increased reliability of workday assessments, it remains unclear why status would covary with CAR and diurnal slope on workdays but not the single non-workday. This distinction between workdays and non-workdays appears to be important, but few prior studies have examined workday/non-workday differences in cortisol activity. The two that have are limited to studies of the awakening response, both of which reported a larger CAR on workdays (Kunz-Ebrecht et al., 2004; Schlotz, et al., 2004). Interestingly, participants in these studies also reported experiencing greater stress and reduced control on workdays, suggesting that psychological states relevant to status might be more salient on workdays compared to non-workdays.

Finally, it is important to mention that covariates accounted for a larger proportion of slope on workdays versus the single non-workday, though this may be a result of aggregating across the three individual workdays.

5.6 LIMITATIONS AND FUTURE DIRECTIONS

The results of this study should be interpreted in light of a few considerations. First, our status measures were obtained through self-report, which is subject to biases in self-presentation, although the fact that both status variables related to HPA activity suggests that findings were robust to any deficiency of self-report. In addition, while our study provided a more

comprehensive assessment of cortisol levels relative to many of the studies reviewed, we did not have an equal number of work and non-work days, and thus, any differences between work and non-work days should be examined further. Cross-sectional data collection in this study limits our ability to assess the direction of association between status and cortisol activity, leaving the possibility that CAR and diurnal cortisol activity might influence interpersonal dominance, income or occupational status. Finally, this sample is relatively homogeneous (i.e. middle-aged, predominantly Caucasian, well-educated), thus limiting the generalizability of our findings to a broader population. In spite of these limitations, our findings are the first to examine the association of two different status constructs (trait dominance and SES) to cortisol activity in the same sample. Our results confirmed a relationship between status and cortisol in humans, and found that both status variables associated consistently with our measures of cortisol activity, suggesting that these two constructs may possess a common attribute that associates with HPA output. Because cortisol is often considered a potential biological mediator of the relationship between psychosocial factors and risk for cardiovascular disease (CVD), future studies might examine the relationship between status and CVD, and the extent to which cortisol might account for these associations.

APPENDIX A

TABLES

Table 1. Demographic, status, and cortisol variable descriptive statistics

	Mean (SD) or %	Minimum	Maximum
Demographic			
Age, years	42.77 (7.35)	30.00	54.00
Sex (% Female)	52.9		
Race (% White)	82.5		
Current Smoker (%)	15.5		
Status			
SES Composite	0.01 (0.77)	-1.99	2.77
Income (adjusted for household)	49,892 (29,311)	2,866	212,750
Occupation	6.34 (1.72)	1.00	9.00
Education, years	16.93 (2.85)	9.00	24.00
Subjective SES	6.12 (1.50)	2.00	10.00
Dominance	1.82 (11.54)	-35.4 0	37.00
Average workday cortisol			
CAR	0.60 (0.59)	-0.34	2.91
Slope	-0.04 (0.01)	-0.07	0.00
AUC	7.44 (2.12)	2.70	15.12
Non-workday cortisol			
CAR	0.39 (0.75)	-0.95	3.70
Slope	-0.04 (0.02)	-0.11	0.02
AUC	7.01 (2.63)	1.26	18.88

Table 2. Correlations of cortisol levels on each sampling day

r		Workday 2	Workday 3	Non-workday
Awakening	Workday 1	.42	.46	.27
	Workday 2	--	.47	.30
	Workday 3		--	.30
+30 Minutes	Workday 1	.45	.41	.33
	Workday 2	--	.46	.31
	Workday 3		--	.24
+ 4 Hours	Workday 1	.32	.36	.22
	Workday 2	--	.33	.12
	Workday 3		--	.16
+ 9 Hours	Workday 1	.18	.16	.08
	Workday 2	--	.20	.19
	Workday 3		--	.09
Bedtime	Workday 1	.17	.18	.29
	Workday 2	--	.43	.19
	Workday 3		--	.24
Average r's	Workday : Workday		.33	
	Workday : Non-workday			.22

Note. All correlations are significant at $p<.001$

Table 3. Correlations of cortisol indices on each sampling day

		Workday 2	Workday 3	Non-workday
CAR	Workday 1	.27	.30	.20
	Workday 2	--	.28	.13
	Workday 3		--	.12
Diurnal Slope	Workday 1	33	.39	.29
	Workday 2	--	.34	.37
	Workday 3		--	.35
AUC	Workday 1	.45	.50	.42
	Workday 2	--	.54	.46
	Workday 3		--	.39
Average r's	Workday : Workday		.38	
	Workday : Non-workday			.29

Note. All correlations are significant at $p<.001$

Table 4. Analysis of Variance examining mean cortisol levels across days

		Mean	F (df)
Awakening	Workday 1	18.10[a]	4.25 (3, 1122)**
	Workday 2	18.05[a]	
	Workday 3	18.39[a]	
	Non-workday	16.58[b]	
+ 30 Minutes	Workday 1	25.60[a]	22.15 (2.92, 875)**
	Workday 2	25.17[a]	
	Workday 3	25.50[a]	
	Non-workday	21.16[b]	
+ 4 Hours	Workday 1	8.17	1.92 (2.85, 1067)
	Workday 2	8.43	
	Workday 3	8.61	
	Non-workday	7.91	
+ 9 Hours	Workday 1	5.79[a]	4.91 (2.88, 1072)**
	Workday 2	5.63[a]	
	Workday 3	6.12[a]	
	Non-workday	5.10[b]	
Bedtime	Workday 1	3.08	1.34 (2.66, 1109)
	Workday 2	3.05	
	Workday 3	3.25	
	Non-workday	2.78	

**p<.01

Table 5. Analysis of Variance examining mean collection time levels across days

		Mean	F (df)
Awakening	Workday 1	6.36[a]	277.69 (2.05, 765)**
	Workday 2	6.47[b]	
	Workday 3	6.46[b]	
	Non-workday	7.92[c]	
+ 30 Minutes	Workday 1	6.78[a]	218.84 (1.92, 232)**
	Workday 2	6.92[b]	
	Workday 3	6.86[a,b]	
	Non-workday	8.26[c]	
+ 4 Hours	Workday 1	10.62[a]	216.46 (2.18, 817)**
	Workday 2	10.77[b]	
	Workday 3	10.72[a, b]	
	Non-workday	12.14[c]	
+ 9 Hours	Workday 1	15.68[a]	167.59 (2.47, 917)**
	Workday 2	15.81[b]	
	Workday 3	15.73[a,b]	
	Non-workday	17.16[c]	
Bedtime	Workday 1	23.04[a]	10.61 (2.94, 1226)
	Workday 2	22.89[b]	
	Workday 3	22.82[b]	
	Non-workday	23.11[a]	

**p<.01

Table 6. Analysis of variance examining cortisol indices across days

		Mean	F (df)
CAR	Workday 1	.57[a]	4.90 (3, 783)**
	Workday 2	.64[a]	
	Workday 3	.61[a]	
	Non-workday	.41[b]	
Slope	Workday 1	-.042	1.00 (3, 1008)
	Workday 2	-.041	
	Workday 3	-.043	
	Non-workday	-.042	
AUC	Workday 1	7.16[a,]	6.48 (2.90, 807)**
	Workday 2	7.31[a, b]	
	Workday 3	7.59[b]	
	Non-workday	6.89[a, c]	

***p*<.01

Table 7. Correlations between age and status variables

r (*p*)	Composite SES	Income	Occupation	Education	Subjective SES	Dominance
Age	-.02 (.44)	.12** (.01)	-.08 (.07)	-.12* (.01)	.03 (.48)	.05 (.27)

Table 8. Independent-samples t-tests comparing mean SES and status by sex

		Mean	SD	*t*	*df*
Composite SES	Males	.12	.82	3.17**	488
	Females	-.10	.71		
Income	Males	52,995.26	32,212.34	2.20*	488
	Females	47,124.24	26,211.52		
Occupation	Males	6.50	1.90	1.82	472
	Females	6.21	1.54		
Education	Males	17.38	2.94	3.29**	488
	Females	16.53	2.72		
Subjective SES	Males	6.39	1.57	3.86**	488
	Females	5.88	1.39		
Dominance	Males	.72	12.23	-2.00	485
	Females	2.81	10.81		

*p<.05, **p<.01

Table 9. Independent-samples t-tests comparing mean SES and status by race

		Mean	(SD)	*t*	*df*
Composite SES	White	.12	.71	7.07**	488
	Non-White	-.52	.80		
Income	White	53,037.97	29,539.07	5.95**	488
	Non-White	35,717.49	23,656.86		
Occupation	White	6.62	1.51	7.76**	472
	Non-White	5.12	2.06		
Education	White	17.19	2.78	4.30**	488
	Non-White	15.74	2.91		
Subjective SES	White	6.28	1.44	4.84*	488
	Non-White	5.40	1.56		
Dominance	White	1.45	11.72	-1.62	485
	Non-White	3.49	10.58		

*p<.05, **p<.01

Table 10. Independent-samples t-tests comparing mean SES and status by smoking status

		Mean	(SD)	*t*	*df*
Composite SES	Non-Smoker	.09	.74	5.11**	487
	Current Smoker	-.42	.81		
Income	Non-Smoker	51,535.60	29,821.59	3.13**	487
	Current Smoker	41,571.47	24,824.16		
Occupation	Non-Smoker	6.53	1.61	5.56*	471
	Current Smoker	5.37	1.97		
Education	Non-Smoker	17.15	2.81	4.05**	487
	Current Smoker	15.74	2.81		
Subjective SES	Non-Smoker	6.20	1.47	2.42*	487
	Current Smoker	5.71	1.64		
Dominance	Non-Smoker	1.56	11.47	-1.13	484
	Current Smoker	3.24	11.97		

*p<.05, **p<.01

Table 11. Correlations between cortisol indices within averaged workdays and the single non-workday

r (*p*)		Workday					
		CAR		SLOPE		AUC	
Non-workday	CAR	--		.37	(.00)	-.34	(.00)
	SLOPE	.32	(.00)	--		-.09	(.12)
	AUC	-.22	(.00)	-.15	(.00)	--	

Table 12. Correlations between age and cortisol variables

r (*p*)		CAR	Slope	AUC
Age	Workday	.10 (.09)	.16** (.00)	.06 (.30)
	Non-workday	.12* (.02)	.06 (.20)	.08 (.12)

Table 13. Independent-samples t-tests comparing mean cortisol by sex

Workday		Mean	SD	*t*	*df*
CAR	Males	.53	.62	-1.91	301
	Females	.66	.56		
Slope	Males	-.046	.01	-4.50**	371
	Females	-.041	.01		
AUC	Males	7.59	2.02	1.11	317
	Females	7.33	2.23		
Non-workday					
CAR	Males	.35	.71	-1.12	378
	Females	.43	.79		
Slope	Males	-.043	.02	-.89	390
	Females	-.042	.02		
AUC	Males	7.04	2.67	.11	317
	Females	7.01	2.63		

*p<.05, **p<.01

Table 14. Independent-samples t-tests comparing mean cortisol by race

Workday		Mean	SD	*t*	*df*
CAR	White	.57	.54	-1.71	301
	Non-white	.73	.80		
Slope	White	-.04	.01	-7.33**	371
	Non-white	-.03	.01		
AUC	White	7.54	2.05	1.62	317
	Non-white	6.94	2.48		
Non-workday					
CAR	White	.36	.69	-2.34*	378
	Non-white	.61	1.01		
Slope	White	-.04	.02	-3.01**	390
	Non-white	-.03	.02		
AUC	White	7.08	2.69	.98	384
	Non-white	6.73	2.36		

*p<.05, **p<.01

Table 15. Independent-samples t-tests comparing mean cortisol by smoking status

Workday		Mean	SD	*t*	*df*
CAR	Non-smoker	.60	.59	.13	300
	Current Smoker	.59	.65		
Slope	Non-smoker	-.04	.01	-1.60	370
	Current Smoker	-.04	.01		
AUC	Non-smoker	7.38	2.03	-1.57	316
	Current Smoker	7.92	2.72		
Non-workday					
CAR	Non-smoker	.39	.73	-.41	377
	Current Smoker	.44	.88		
Slope	Non-smoker	-.042	.02	-2.63**	389
	Current Smoker	-.035	.02		
AUC	Non-smoker	7.00	2.67	-.56	383
	Current Smoker	7.20	2.51		

*p<.05, **p<.01

Table 16. Correlations between status variables

r (*p*)	Subjective SES	SES Composite	Education	Income	Occupation	Income + Occupation
Dominance	.14 (.00)	.04 (.38)	-.02 (.70)	.10 (.03)	.01 (.76)	.08 (.09)
Subjective SES	--	.55 (.00)	.42 (.00)	.46 (.00)	.39 (.00)	.49 (.00)
SES Composite		--	.76 (.00)	.74 (.00)	.82 (.00)	.90 (.00)
Education			--	.28 (.00)	.48 (.00)	.41 (.00)
Income				--	.43 (.00)	.85 (.00)
Occupation					--	.82 (.00)

Table 17. Correlations between status and cortisol variables

r (*p*)		CAR	Diurnal Slope	AUC
Composite SES	Average Workday	-.14** (.01)	-.21** (.00)	.02 (.70)
	Non-workday	-.05 (.33)	-.04 (.44)	.02 (.70)
Income	Average Workday	-.12* (.04)	-.15** (.00)	.03 (.62)
	Non-workday	-.01 (.93)	.03 (.54)	.01 (.89)
Occupation	Average Workday	-.16** (.01)	-.29** (.00)	.06 (.26)
	Non-workday	-.08 (.12)	-.12* (.02)	.02 (.68)
Education	Average Workday	-.06 (.29)	-.06 (.23)	-.02 (.67)
	Non-workday	-.03 (.53)	-.03 (.58)	.01 (.92)
Subjective SES	Average Workday	-.10 (.08)	-.13** (.01)	-.01 (.82)
	Non-workday	-.02 (.65)	.02 (.76)	-.07 (.19)
Dominance	Average Workday	-.13* (.02)	-.07 (.17)	.01 (.90)
	Non-workday	.05 (.34)	.02 (.66)	.05 (.32)

Table 18. Hierarchical linear regression analysis predicting CAR from dominance scores

	Average Workday CAR					Non-workday CAR			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2=.03*				
(Constant)	.11	.27		.49	(Constant)	-.35	.26		-1.36
Age	.004	.01	.05	.83	Age	.01	.01	.10	1.89
Sex	.11	.07	.09	1.60	Sex	.03	.08	.02	.34
Race	.13	.09	.08	1.42	Race	.23	.11	.11	2.21*
Step 2: R^2= .04*					*Step 2*: R^2= .03				
(Constant)	.04	.23		.21	(Constant)	-.33	.26		-1.30
Age	.01	.01	.06	.99	Age	.01	.01	.10	1.87
Sex	.13	.07	.11	1.89	Sex	-.02	.08	-.01	.27
Race	.14	.09	.09	1.49	Race	.23	.11	.11	2.08*
Dominance	-.08	.03	-.13	-2.30*	Dominance	.02	.04	.03	.57

*p<.05, **p<.01

Table 19. Hierarchical linear regression analysis predicting diurnal slope from dominance scores

	Average workday diurnal slope					Non-workday diurnal slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .16**					*Step 1*: R^2= .03**				
(Constant)	-.07	.003		-16.92**	(Constant)	-.06	.004		-9.23**
Age	.001	.001	.11	2.24*	Age	.001	.001	.04	.81
Sex	.01	.001	.18	3.65**	Sex	.001	.002	.02	.35
Race	.01	.002	.31	6.46**	Race	.01	.003	.17	3.31**
Step 2: R^2= .17*					*Step 2*: R^2= .03				
(Constant)	-.07	.003		-17.10**	(Constant)	-.06	.004		-9.15**
Age	.001	.001	.11	2.24*	Age	.001	.001	.04	.81
Sex	.01	.001	.19	3.89**	Sex	.001	.002	.02	.32
Race	.01	.002	.32	6.59**	Race	.01	.003	.17	3.29**
Dominance	-.001	.001	-.11	-2.20*	Dominance	.000	.001	.01	.19

*p<.05, **p<.01

Table 20. Hierarchical linear regression analysis predicting AUC from dominance scores

	Average workday AUC					Non-workday AUC			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2= .01				
(Constant)	8.52	.53		16.14**	(Constant)	7.59	.60		12.67**
Age	.09	.12	.04	.71	Age	.18	.14	.07	1.27
Sex	-.25	.24	-.06	-1.01	Sex	-.06	.28	-.01	-.22
Race	-.59	.33	-.10	-1.80	Race	-.41	.40	-.05	-1.03
Step 2: R^2= .02					*Step 2*: R^2= .01				
(Constant)	8.63	.53		16.19**	(Constant)	7.65	.60		12.66**
Age	.09	.12	.04	.75	Age	.17	.14	.06	1.23
Sex	-.28	.25	-.07	-1.15	Sex	-.08	.28	-.02	-.29
Race	-.63	.33	-.11	-1.91	Race	-.43	.40	-.06	-1.08
Dominance	-.16	.12	-.08	1.37	Dominance	.10	.14	.04	.75

*p<.05, **p<.01

Table 21. Hierarchical linear regression analysis predicting CAR from composite SES scores

	Average Workday CAR					Non-workday CAR			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2=.03				
(Constant)	.27	.23		1.83	(Constant)	.09	.17		.54
Age	.03	.01	.05	.76	Age	.08	.04	.10	1.94
Sex	.12	.07	.10	1.66	Sex	.04	.08	.03	.50
Race	.13	.09	.08	1.39	Race	.22	.11	.11	2.03
Smoking Status	-.002	.07	-.002	-.03	Smoking Status	-.04	.08	-.03	-.48
Step 2: R^2= .04*					*Step 2*: R^2= .03				
(Constant)	.38	.23		.2.40*	(Constant)	.12	.18		.67
Age	.03	.01	.05	.87	Age	.08	.04	.10	1.94
Sex	.11	.07	.09	1.55	Sex	.03	.08	.02	.43
Race	.07	.10	.04	.71	Race	.21	.11	.10	1.84
Smoking Status	-.04	.08	-.03	-.56	Smoking Status	-.05	.08	-.03	-.59
SES	-.08	.04	-.13	-2.09*	SES	-.02	.04	-.03	-.491

*p<.05, **p<.01

Table 22. Hierarchical linear regression analysis predicting diurnal slope from composite SES scores

	Average workday slope					Non-workday slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .17**					*Step 1*: R^2=.04**				
(Constant)	-.06	.003		-23.11**	(Constant)	-.06	.04		-12.81**
Age	.001	.001	.11	2.24*	Age	.001	.001	.04	.77
Sex	.004	.001	.17	3.57**	Sex	.001	.002	.02	.35
Race	.01	.002	.31	6.36**	Race	.01	.003	.16	3.15**
Smoking Status	.001	.001	.05	1.07	Smoking Status	.002	.002	.06	1.18
Step 2: R^2= .17					*Step 2*: R^2= .04				
(Constant)	-.06	.003		-21.37**	(Constant)	-.06	.004		-12.24**
Age	.001	.001	.11	2.35*	Age	.001	.001	.04	.71
Sex	.004	.001	.17	3.42**	Sex	.001	.002	.02	.46
Race	.01	.002	.28	5.58**	Race	.01	.003	.18	3.26**
Smoking Status	.001	.001	.03	.59	Smoking Status	.003	.002	.07	1.35
SES	-.001	.001	-.09	-1.73	SES	.001	.001	.05	.87

*p<.05, **p<.01

Table 23. Hierarchical linear regression analysis predicting AUC from composite SES scores

	Average workday AUC					Non-workday AUC			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2= .01				
(Constant)	8.48	.53		16.11**	(Constant)	7.54	.60		12.51**
Age	.07	.12	.03	.60	Age	.17	.14	.06	1.19
Sex	-.26	.24	-.06	-1.07	Sex	-.05	.28	-.01	-.19
Race	-.63	.33	-.11	-1.92	Race	-.41	.40	-.05	-1.03
Smoking Status	.32	.25	.07	1.25	Smoking Status	.07	.28	.01	.24
Step 2: R^2= .02					*Step 2*: R^2= .01				
(Constant)	8.52	.55		15.42**	(Constant)	7.40	.65		11.40**
Age	.07	.12	.04	.61	Age	.16	.14	.06	1.16
Sex	-.26	.25	-.06	-1.08	Sex	-.03	.28	-.005	-.09
Race	-.66	.34	-.11	-1.90	Race	-.33	.41	-.04	-.81
Smoking Status	.30	.26	.07	1.16	Smoking Status	.11	.29	.02	.38
SES	-.03	.14	-.02	-.24	SES	.09	.15	.03	.60

*p<.05, **p<.01

Table 24. Hierarchical linear regression analysis predicting CAR from SES I/O

	Average Workday CAR					Non-workday CAR			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2=.03*				
(Constant)	.27	.15		1.83	(Constant)	.09	.17		.54
Age	.03	.04	.05	.76	Age	.08	.04	.10	1.94
Sex	.12	.07	.10	1.66	Sex	.04	.08	.03	.50
Race	.13	.09	.08	1.39	Race	.22	.11	.11	2.03*
Smoking Status	-.002	.07	-.00	-.03	Smoking Status	-.04	.08	-.03	-.48
Step 2: R^2= .05**					*Step 2*: R^2= .03				
(Constant)	.39	.15		2.53*	(Constant)	-.13	.18		.71
Age	.04	.04	.07	1.12	Age	.08	.04	.11	1.99*
Sex	.11	.07	.09	1.63	Sex	.03	.08	.02	.43
Race	.05	.10	.03	.54	Race	.20	.11	.10	1.80
Smoking Status	-.05	.08	-.04	-.65	Smoking Status	-.05	.08	-.03	-.61
SES I/O	-.10	.04	-.17	-2.73**	SES I/O	-.03	.04	-.04	-.66

*p<.05, **p<.01

Table 25. Hierarchical linear regression analysis predicting diurnal slope from SES I/O

	Average workday slope					Non-workday slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .17**					*Step 1*: R^2=.04**				
(Constant)	-.06	.003		-23.11**	(Constant)	-.05	.014		-12.81**
Age	.001	.001	.11	2.24*	Age	.001	.001	.04	.77
Sex	.004	.001	.17	3.57**	Sex	.001	.002	.02	.35
Race	.01	.002	.31	6.36**	Race	.01	.003	.16	3.15**
Smoking Status	.001	.001	.05	1.07	Smoking Status	.002	.002	.06	1.18
Step 2: R^2= .19**					*Step 2*: R^2= .04				
(Constant)	-.06	.003		-21.40**	(Constant)	-.05	.004		-12.34**
Age	.002	.001	.13	2.64**	Age	.002	.001	.04	.69
Sex	.004	.001	.17	3.47**	Sex	.002	.002	.02	.40
Race	.01	.002	.26	5.20**	Race	.01	.003	.17	3.15**
Smoking Status	.000	.001	.01	.29	Smoking Status	.003	.002	.07	1.26
SES I/O	-.002	.001	-.16	-3.18**	SES I/O	.001	.001	.03	.49

*p<.05, **p<.01

Table 26. Hierarchical linear regression analysis predicting AUC from SES I/O

	Average workday AUC					Non-workday AUC			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2= .01				
(Constant)	8.48	.53		16.11**	(Constant)	7.54	.60		12.51**
Age	.07	.12	.03	.60	Age	.17	.14	.06	1.19
Sex	-.26	.24	-.06	-1.07	Sex	-.05	.28	-.01	-.19
Race	-.63	.33	-.11	-1.92	Race	-.41	.40	-.05	-1.03
Smoking Status	.32	.25	.07	1.26	Smoking Status	.07	.28	.01	.24
Step 2: R^2= .02					*Step 2*: R^2= .01				
(Constant)	8.39	.55		15.32**	(Constant)	7.50	.64		11.72**
Age	.06	.12	.03	.49	Age	.16	.14	.06	1.16
Sex	-.26	.24	-.06	-1.04	Sex	-.05	.28	-.01	-.16
Race	-.57	.34	-.10	-1.67	Race	-.38	.41	-.05	-.93
Smoking Status	.35	.26	.08	1.34	Smoking Status	.08	.29	.01	.28
SES I/O	.08	.14	.04	.59	SES I/O	.03	.15	.01	.21

*p<.05, **p<.01

Table 27. Hierarchical linear regression analysis predicting CAR from subjective SES scores

	Average Workday CAR					Non-workday CAR			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2=.03*				
(Constant)	.27	.15		1.83	(Constant)	.09	.17		.54
Age	.03	.04	.05	.76	Age	.08	.04	.10	1.94
Sex	.12	.07	.10	1.66	Sex	.04	.08	.03	.50
Race	.13	.09	.08	1.39	Race	.22	.11	.11	2.03*
Smoking Status	-.002	.07	-.002	-.03	Smoking Status	-.04	.08	-.03	-.48
Step 2: R^2= .03					*Step 2*: R^2= .03				
(Constant)	.36	.16		2.31	(Constant)	.06	.18		.35
Age	.04	.04	.062	1.04	Age	.08	.04	.10	1.88
Sex	.09	.07	.08	1.33	Sex	.05	.08	.03	.60
Race	.09	.10	.06	.95	Race	.23	.11	.11	2.09*
Smoking Status	-.02	.07	-.02	-.25	Smoking Status	-.03	.08	-.02	-.37
Subjective SES	-.07	.04	-.11	-1.82	Subjective SES	.03	.04	-.03	.64

*p<.05, **p<.01

Table 28. Hierarchical linear regression analysis predicting diurnal slope from subjective SES scores

	Average workday slope					Non-workday slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .17**					*Step 1*: R^2=.04**				
(Constant)	-.06	.003		-23.11**	(Constant)	-.05	.004		-12.81**
Age	.001	.001	.11	2.24*	Age	.001	.001	.04	.77
Sex	.004	.001	.17	3.57**	Sex	.001	.002	.02	.35
Race	.01	.002	.31	6.36**	Race	.01	.003	.16	3.15**
Smoking Status	.001	.001	.05	1.07	Smoking Status	.002	.002	.06	1.18
Step 2: R^2= .17					*Step 2*: R^2= .04				
(Constant)	-.07	.03		-21.81**	(Constant)	-.06	.004		-12.59**
Age	.001	.001	.11	2.30*	Age	.001	.001	.03	.67
Sex	.004	.001	.17	3.42**	Sex	.001	.002	.03	.50
Race	.01	.002	.30	6.02**	Race	.01	.003	.17	3.28**
Smoking Status	.001	.001	.05	.97	Smoking Status	.03	.002	.07	1.31
Subjective SES	.000	.001	-.03	-.61	Subjective SES	.001	.001	.05	1.00

*p<.05, **p<.01

Table 29. Hierarchical linear regression analysis predicting AUC from subjective SES scores

	Average workday AUC					Non-workday AUC			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2= .01				
(Constant)	8.48	.53		16.11**	(Constant)	7.54	.60		12.51**
Age	.07	.12	.03	.60	Age	.17	.14	.06	1.19
Sex	-.26	.24	-.06	-1.07	Sex	-.05	.28	-.01	-.19
Race	-.63	.33	-.11	-1.92	Race	-.41	.40	-.05	-1.03
Smoking Status	.32	.25	.07	1.25	Smoking Status	.07	.28	.01	.24
Step 2: R^2= .02					*Step 2*: R^2= .02				
(Constant)	8.54	.56		15.39**	(Constant)	7.74	.62		12.44**
Age	.08	.12	.04	.65	Age	.18	.14	.07	1.29
Sex	-.28	.25	-.06	-1.11	Sex	-.11	.28	-.02	-.40
Race	-.66	.34	-.11	-1.95	Race	-.47	.40	-.06	-1.19
Smoking Status	.31	.26	.07	1.21	Smoking Status	.02	.29	.00	.06
Subjective SES	-.05	.13	-.02	-.37	Subjective SES	-.18	.14	-.07	-1.29

*p<.05, **p<.01

Table 30. Hierarchical linear regression analysis predicting CAR and slope from dominance adjusting for time of awakening

	Average workday CAR					Average workday Slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .05**					*Step 1*: R^2= .16**				
(Constant)	.80	.31		2.59**	(Constant)	-.07	.006		-12.69**
Age	.002	.01	.02	.33	Age	.001	.001	.12	2.33*
Sex	.10	.07	.08	1.44	Sex	.005	.001	.18	3.66**
Race	.17	.09	.10	1.81	Race	.01	.002	.31	6.32**
Wake Time	-.10	.03	-.18	-3.20**	Wake Time	.001	.001	.03	.68
Step 2: R^2= .07*					*Step 2*: R^2= .17*				
(Constant)	.74	.31		2.34*	(Constant)	-.07	.006		-12.91**
Age	.002	.01	.03	.49	Age	.001	.001	.12	2.36**
Sex	.12	.07	.10	1.74	Sex	.01	.001	.19	3.92**
Race	.17	.09	.11	1.88	Race	.01	.002	.31	6.44**
Wake Time	-.10	.03	-.18	-3.21**	Wake Time	.001	.001	.04	.85
Dominance	-.01	.003	-.13	-2.32*	Dominance	-.001	.001	-.12	-2.26*

*p<.05, **p<.01

Table 31. Hierarchical linear regression analysis predicting CAR and slope from dominance adjusting for time of awakening

	Average workday CAR					Average workday Slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .06**					*Step 1*: R^2= .17**				
(Constant)	.83	.31		2.67**	(Constant)	-.07	.006		-12.70**
Age	.001	.01	.02	.26	Age	.001	.001	.12	2.32*
Sex	.10	.07	.09	1.51	Sex	.004	.001	.17	3.58**
Race	.17	.09	.10	1.79	Race	.01	.002	.30	6.23**
Smoking Status	-.01	.07	-.01	-.18	Smoking Status	.001	.001	.05	1.03
Wake Time	-.10	.03	-.19	-3.36**	Wake Time	.001	.001	.03	.64
Step 2: R^2= .09**					*Step 2*: R^2= .19**				
(Constant)	.95	.31		3.06**	(Constant)	-.07	.006		-12.31**
Age	.003	.01	.04	.62	Age	.001	.001	.13	2.66**
Sex	.10	.07	.08	1.46	Sex	.004	.001	.17	3.47**
Race	.08	.10	.05	.86	Race	.01	.002	.26	5.15**
Smoking Status	-.07	.07	-.05	-.90	Smoking Status	.001	.001	.01	.28
Wake Time	-.11	.03	-.21	-3.61**	Wake Time	.001	.001	.02	.33
SES I/O	-.13	.04	-.19	-3.13**	SES I/O	-.003	.001	-.16	-3.13**

*p<.05, **p<.01

Table 32. Hierarchical linear regression analysis predicting CAR and Slope from dominance and SES I/O entered simultaneously

	Average workday CAR					Average workday Slope			
	B	SE	β	*t*		B	SE	β	*t*
Step 1: R^2= .02					*Step 1*: R^2= .01				
(Constant)	.12	.07		.54	(Constant)	-.07	.004		-16.92**
Age	.004	.09	.04	.75	Age	.001	.001	.11	2.21*
Sex	.12	.07	.10	1.65	Sex	.004	.001	.17	3.56**
Race	-.63	.09	.08	1.39	Race	.01	.002	.31	6.35**
Smoking Status	-.002	.07	-.001	-.02	Smoking Status	.001	.001	.05	1.09
Step 2: R^2= .06					*Step 2*: R^2= .02				
(Constant)	.10	.23		.43	(Constant)	-.07	.004		-16.91**
Age	.01	.01	.07	1.19	Age	.001	.001	.12	2.57**
Sex	.13	.07	.11	1.87	Sex	.01	.001	.18	3.65**
Race	.07	.10	.04	.68	Race	.01	.002	.27	5.34**
Smoking Status	-.04	.08	-.03	-.46	Smoking Status	.001	.001	.02	.43
SES I/O	-.09	.04	-.15	-2.41*	SES I/O	-.002	.001	-.15	-2.86**
Dominance	-.07	.03	-.11	-1.92	Dominance	-.001	.001	-.09	-1.81

*p<.05, **p<.01

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