Chapter 13
Hormones and Hierarchies

Erik L. Knight and Pranjal H. Mehta

Status hierarchies are a pervasive feature of human and animal societies, organizing individuals along a shared understanding of respect and influence. Group members at the top of a hierarchy are afforded several benefits, including greater access to valued resources (e.g., food, money, mating opportunities) and greater power over lower-ranking members. But hierarchies have broader benefits for the entire group as well, improving group efficiency, effectiveness, and productivity (Halevy et al. 2011; Ronay et al. 2012). These dynamics are evident in the status hierarchies of modern, democratic political systems. Hierarchical order within politics allows those with high status to determine the course of laws, presumably improving the effectiveness of the decision-making process. Meanwhile, earning the respect and votes of a majority of citizens brings with it specific benefits. High-status politicians surround themselves with a legion of staffers and interns who cater to their needs. Politicians also leverage their political sway to reap financial benefits for their constituents and for themselves. Compare this to a low-status position, like an intern who instead must do as he is told in order to gain access to his bosses' control of laws and finances, and is most likely underpaid, if paid at all.

But if you ask a politician why he wants an elected position, neither the perks of high status nor the hassles of low status will be mentioned. Instead, the politician will likely rely on an assertive, confident behavioral style to accentuate some verbal answer that emphasizes his many strengths and his opponent's weaknesses. In political settings, these dominant behaviors may be further evident in public forums like impassioned speeches or fiery head-to-head debates. These displays of social dominance and competitive behaviors represent one important route to attaining high status in both human and animal societies (Anderson and Kilduff 2009a; Cheng et al. 2013; Mazur and Booth 1998). Similar responses occur in many animal social groups where competitions for status often take the form of overtly aggressive behaviors when status is challenged (Mazur and Booth 1998). The nascent field of social endocrinology, which studies the intersection of behavioral endocrinology

E. L. Knight (✉) · P. H. Mehta
Department of Psychology, University of Oregon, Eugene, USA
e-mail: elk@uoregon.edu

and social-personality psychology, provides insights into the role of dynamic, hormonal systems in regulating these behaviors in pursuit of not just political status, but of status in any hierarchy.

For over 150 years, hormones—chemical messengers secreted from endocrine glands that communicate with systems throughout the body including the central nervous system—have been studied in animals as effectors of physiological and behavioral change. The earliest experimental work demonstrated that some unknown chemical from reimplanted testes could prevent the loss of typical male morphology (e.g., combs and waddles) and behavior (e.g., crowing) in a castrated rooster (Berthold 1849; Soma 2006). Later work identified this chemical as the hormone testosterone and showed that it is responsible for these and other forms of virility, like the development of specialized anatomy (e.g., deer's antlers; Lincoln et al. 1972) and the propensity to behave aggressively during the mating season (Wingfield et al. 1990). From these early roots, the field has expanded to explore other hormones, increasingly complex behaviors, and most recently has begun examining hormones and social processes in humans (Mehta and Josephs 2011).

In this chapter, we review the neuroendocrine systems that influence and respond to the behaviors that govern status. We focus on hormones and status in human hierarchies, but we rely on animal work to inform our discussion. We selectively review separate lines of research on testosterone and cortisol before turning to recent evidence on the interaction between these two hormones in status hierarchies. Sections on estradiol and oxytocin follow, and we conclude by examining future directions for research on the interplay between hormones and hierarchies.

Testosterone

Testosterone (T), a steroid hormone derived from cholesterol, is primarily produced in the testes in males, the ovaries in females, and in the adrenal glands in both sexes. T belongs to a class of hormones called androgens, which are those hormones that are responsible for the development and maintenance of masculine characteristics (Wu and Shah 2011). Secretion of T results from the coordinated action of the hypothalamic-pituitary-gonadal (HPG) axis: Gonadotropin-releasing hormone (GnRH) from the hypothalamus stimulates the release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) from the pituitary gland. LH and FSH then stimulate secretion of T from the gonads (Payne and Hales 2004). Higher levels of circulating testosterone are associated with increased dominant behaviors in several animal species. This relationship is strongest when social rank within the hierarchy is unstable. Sapolsky's (1991) research on wild baboons showed that during periods of social instability, due in part to an injured, alpha male within the baboon troop, T was positively related to aggressive and dominant behaviors. In times of stability, there was no relationship between T and these behaviors, a pattern seen in several other species as well (cichlid fish, Oliveira et al. 1996; lamb, Ruiz-de-la-Torre and Manteca 1999; birds, Wingfield et al. 1990). The animal literature thus suggests

that high-T levels motivate individuals to seek out higher status when social status is up for grabs.

The association between T and status pursuit also extends to humans. Social dominance tends to be associated with higher-basal T levels in both men and women (reviewed in Archer 2006; Mazur and Booth 1998; Carre et al. 2011; Eisenegger et al. 2011). T also influences affective and attentional processes related to status, increasing vigilance and emotional reactivity toward dominance cues, such as angry, threatening faces (Hermans et al. 2008; Terburg et al. 2012; van Honk et al. 1999; Wirth and Schultheiss 2007) and decreasing vigilance toward submissive cues such as fearful faces (van Honk et al. 2005). These effects of T on attention are thought to be unconscious, given that the effects of T are strongest when dominant and submissive faces are presented outside of conscious awareness (van Honk et al. 2005; Terburg et al. 2012; Wirth and Schultheiss 2007).

Basal Testosterone's Role in Status Seeking

The findings reviewed above are consistent with the hypothesis that T levels influence status-seeking motivation. To provide more direct tests of T's role in the desire for status, several studies were conducted in which social status was experimentally manipulated, primarily in dyadic competitive social interactions. In eight different studies, basal T was measured in saliva before a status manipulation, after which various affective, cognitive, physiological, and behavioral outcomes were measured (Jones and Josephs 2006; Josephs et al. 2003, 2006; Mehta et al. 2008; Newman et al. 2005). In all of these studies, the interaction between basal T and status predicted the outcomes under investigation. For example, high-T individuals who lost status performed poorly on complex cognitive tasks, paid more attention to status cues, and exhibited increases in negative affect (Josephs et al. 2006). High-T individuals who gained status showed the opposite pattern of response, performing well on complex cognitive tasks, paying less attention to status cues, and showing no signs of increased negative mood. Mehta et al. (2008) further showed that high-T individuals who lost status rose in cortisol, a neuroendocrine marker of psychological stress. Meanwhile, high-T individuals who gained status dropped in cortisol. Taken together, these results suggest that high-basal T levels are linked to a drive for high status. High-T individuals who achieve high-status experience psychological comfort (e.g., low negative affect) and adaptive functioning (e.g., strong cognitive performance). High-T individuals who fail to achieve high-status experience psychological distress (e.g., cortisol increases, high negative affect) and maladaptive functioning (e.g., poor cognitive performance).

Low-T individuals in these same studies reacted differently to changes in status. In some studies low-T individuals reacted to low and high status similar to individuals in control conditions in which status level was not experimentally manipulated (Newman et al. 2005) or was not threatened (Josephs et al. 2003). These results suggest that low-T individuals lack the strong drive for high status found in high-T

individuals. In other studies, low-T individuals found high-status positions aversive. When placed in high-status positions, low-T people were hypervigilant to status cues, showed elevated physiological arousal, and performed worse at cognitive tasks compared to low-T individuals placed in low-status positions (Josephs et al. 2006). These results suggest that when thrust into high-ranking positions, low-T individuals may experience arousal and maladaptive functioning out of a desire to return to a more comfortable low-status position. Thus, T may orient individuals toward or away from high status and influence psychological and behavioral responses within a hierarchy.

Women have approximately one-third the concentrations of T in saliva relative to men, but there were no sex differences in the predictive power of basal T on reactions to changes in status in these studies. Men and women high in T relative to other individuals of the same sex reacted negatively to a drop in status (Josephs et al. 2003, 2006; Mehta et al. 2008; Newman et al. 2005). Men and women low in T relative to other individuals of the same sex showed neutral (Josephs et al. 2003; Mehta et al. 2008; Newman et al. 2005) or negative reactions (Josephs et al. 2006) to a rise in status. These findings suggest that basal T is a biological marker of chronic status-seeking motivation in both men and women.

Basal T's role in behavior within status hierarchies is not limited to dyadic social interactions. Other work indicates that a group's specific composition of basal T and social rank may help (or undermine) group cohesion and effectiveness. In one study of 579 students enrolled in an introductory organizational behavior course, students provided saliva samples and were randomly assigned to small work groups. The groups worked on various projects over the course of a semester (Zyphur et al. 2009). Group members rated one another on status, and measures of overall group effectiveness were collected (group efficacy). There was no direct association between basal T and social status, but the (mis)match between T and status was associated with group functioning. Those groups in which high-T individuals had high status and low-T individuals had low status reported greater group efficacy than those groups in which low-T individuals had high status and high-T individuals had low status. These findings provide further evidence that high-T individuals are more comfortable in high-status positions than low-T individuals, which in turn impacts group functioning. When there is a match between basal T and status attainment (high-T individuals with high status, low-T individuals with low status), the group functions well. But when there is a mismatch between basal T and status attainment (low-T individuals with high status, high-T individuals with low status), the group functions poorly (c.f., Josephs et al. 2006) (see Ronay et al. 2012 for findings on prenatal testosterone exposure and group effectiveness).

Acute Changes in Testosterone and Status Seeking

The studies reported above examined basal T as a stable trait and demonstrated its association with status-seeking behaviors. However, not only does T influence

behavior, but behavior and the social environment influence T levels. Specifically, T concentrations fluctuate around basal levels in status-relevant social settings. According to the reciprocal model of T and status, a rise or drop in status should influence T levels, and these rapid T fluctuations should produce a reciprocal effect by influencing subsequent status-seeking behaviors (Mazur and Booth 1998). In particular, scholars have speculated that T increases may encourage further attempts at gaining status, while T decreases may lead individuals to flee the situation to avoid any further loss of status.

Empirical support for this reciprocal model comes from research in competitions, modeled in real-world sporting events and rigged laboratory settings. Several studies have shown that winners' T concentrations increase relative to losers for a few hours following a competition (reviewed in Archer 2006; Mazur and Booth 1998; Salvador and Costa 2009; van Anders and Watson 2006). Intriguingly, this win-lose effect can occur in vicarious experiences of winning or losing as well, such as in sports fans or in supporters of political candidates. In a study of soccer fans watching a match, fans of the winning team increased in T compared to fans of the losing team (Bernhardt et al. 1998). And in the 2008 Presidential Election, supporters of losing candidates (John McCain or Robert Barr) dropped in T relative to supporters of the winning candidate (Barack Obama) in the hours following the announcement of the election results (Stanton et al. 2009).

These effects of victory and defeat on T responses are not always found, suggesting a role for other psychosocial and physiological variables in modulating the post-competition T response. For example, implicit power motivation—an individual difference factor associated with an unconscious desire to obtain high-status positions—moderated the effects of victory and defeat on changes in T in one study (Schultheiss et al. 2005). High-power individuals rose in T after victory and dropped in T after defeat; low-power individuals showed the opposite pattern of T changes. Presumably, people high in power motive are chronically motivated to gain status. Thus, when their status drops, these individuals react strongly by dropping in T. When their status rises, they react strongly by rising in T. Other moderators of the win-lose effect on T changes include the cognitive and affective response to the competition (Salvador and Costa 2009), biological factors such as basal hormone profiles (Mehta and Josephs 2010), personality traits (trait anxiety, Maner et al. 2008), and environmental factors (home versus away game, Carré 2009).

Although this literature has uncovered several factors that predict T changes after changes in status, researchers had assumed that status-induced fluctuations in T influenced future status-seeking behaviors. Mehta and Josephs (2006) conducted the first empirical study in humans that examined the relationship between postcompetition T changes and subsequent social behavior. Status was experimentally manipulated with a rigged laboratory competition, and saliva samples were collected before and after the competition to measure changes in T (Mehta and Josephs 2006). After competing and after providing the second saliva sample, participants were given the option of competing against the same opponent in a second competition or completing an alternative, noncompetitive task (Mehta and Josephs 2006). Individuals who lost the competition and whose T concentration rose were

significantly more likely to choose to compete again than individuals who lost and whose T concentration dropped. Another study showed the same pattern of findings with measures of aggressive behavior (Carré et al. 2009; see also Carré et al. 2011). These findings are consistent with the reciprocal model and suggest that a rise in T after a drop in status motivates further attempts at gaining status, while a drop in T after a drop in status motivates individuals to avoid any further loss of status.

All together, the research on T in humans suggests that basal T taps into a person's chronic status-seeking motivation, analogous to a personality trait, whereas short-term changes in T tap into a person's state status-seeking motivation, analogous to mood (cf. Mehta et al. 2008).

Cortisol

Cortisol, a steroid hormone of the glucocorticoid family, is produced in the adrenal glands and released as the end product of the hypothalamic-pituitary-adrenal axis (HPA-axis). Physical and psychological stress stimulates a hormonal cascade, wherein the hypothalamus secretes corticotropin-releasing hormone (CRH), which stimulates adrenocorticotropic hormone (ACTH) to be released from the anterior pituitary, which finally stimulates cortisol release (see Fig. 13.1). The primary function of cortisol is to mobilize glucose to fuel the "fight or flight" response to stress and enhance cardiovascular functioning (Sapolsky 1999). Cortisol also acts as a signal to inhibit further HPA-axis activity, forming a negative feedback loop that helps attenuate the stress response. Thus, cortisol release is a useful and adaptive response to stress in the short term that physiologically prepares the body for the rigors of reacting to a stressful experience.

But overexposure to cortisol due to chronic stress disrupts the negative feedback loop, leading to cyclically increasing cortisol concentrations and a generally dysregulated physiological response to stress. Continual cortisol secretion reduces the quantity and efficacy of glucocorticoid receptors in the brain and along the HPA-axis, resulting in an overall attenuation of glucocorticoid receptor activity in response to cortisol (Sapolsky et al. 1985; Cohen et al. 2012). This reduction in glucocorticoid receptor activity leads to an inability to effectively suppress the endocrine stress response, resulting in sustained activation of the HPA-axis. In fact, when impairment of the negative feedback loop results in excess secretion, cortisol itself becomes a liability, with its deleterious effects including neural atrophy (Sapolsky 1996), poor immune functioning (Cohen et al. 2012), and cardiovascular disease (Sapolsky 2004).

Cortisol typically increases sharply each morning shortly after awakening—termed the cortisol awakening response (CAR)—followed by a decline throughout the day—referred to as the diurnal cortisol slope (Fries et al. 2009). Chronic stress dysregulates this cycle, attenuating the awakening response and flattening the diurnal cortisol slope (Dowd et al. 2009; Miller et al. 2007). In summary, cortisol concentration rises in response to stressors and, if the stress is chronic, leads to
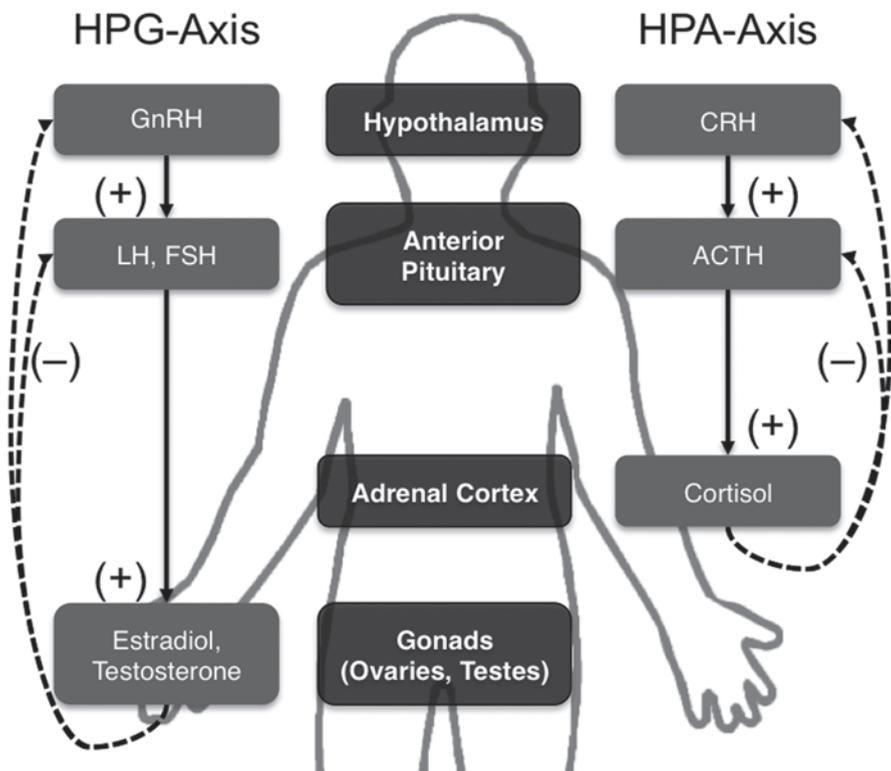
Fig. 13.1 Hypothalamic-pituitary-adrenal (*HPA*) and hypothalamic-pituitary-gonadal (*HPG*) axes and their cascading endocrine responses. *Solid lines* represent a stimulating effect; *dashed lines* represent inhibitory effects. Note that within the menstrual cycle, estradiol will stimulate production and release of LH once estradiol concentration reaches a certain threshold. Otherwise, estradiol will have a suppressive effect on the pituitary hormones. *GnRH* gonadotropin-releasing hormone, *LH* luteinizing hormone, *FSH* follicle-stimulating hormone, *CRH* corticotropin-releasing hormone, *ACTH* adrenocortiocotropic hormone

increasingly stronger cortisol responses to subsequent stressors as well as further exposure to cortisol through the dysregulation of the daily cycle of release. Cortisol is thus an integral part of the body's physiological reaction to stress exposure. Below we review the evidence linking cortisol to status processes in humans.

Correlational Evidence for a Status-Cortisol Link

Basal Cortisol and Status Socioeconomic status, a broad measure of individual or familial wealth and education within society or a community (Adler and Ostrove 1999), correlates negatively with basal cortisol concentrations in several human studies (Cohen et al. 2006a; Evans and English 2002; Evans and Kim 2007; Garcia

et al. 2008; Li et al. 2007; Lupien et al. 2000; Steptoe et al. 2003; Kapuku et al. 2002). This inverse relationship between status and basal cortisol has been found in other status-relevant domains as well. Leaders had lower-basal cortisol compared to nonleaders in a unique sample of military and business personnel (Sherman et al. 2012). The higher cortisol levels seen in lower-status individuals is typically attributed to cortisol dysregulation that results from exposure to increased quantity or severity of stressors inherent to low ranking within a status hierarchy (Sapolsky 2005). Alternatively, if low-status individuals are more frequently stressed, increased cortisol may simply represent more recent contact with an acute stressor (Gersten 2008).

While these results illustrate that lower status is linked to high-basal cortisol, other studies examining basal cortisol concentrations and status found no relationship (Goodman et al. 2005; Gersten 2008; Gadinger et al. 2011). But it may be that low-hierarchical rank is linked to the daily pattern of cortisol secretion, not just basal cortisol concentrations.

Diurnal Cortisol Slope and Status Status does indeed relate to differential patterns of daily cortisol secretion. As described above, healthy diurnal cortisol secretion consists of the sharp, CAR-related increase in cortisol concentration followed by the day-long decline measured by the diurnal cortisol slope (Fries et al. 2009). In several studies, the diurnal cortisol slope appears flattened in lower SES individuals compared to higher-SES individuals (Agbedia et al. 2011; Kumari et al. 2010; Li et al. 2007; Ranjit et al. 2005; Do et al. 2011; Hajat et al. 2010). Following the cortisol awakening response, low SES individuals' cortisol concentrations decline at a slower rate throughout the day, which may result in greater exposure to cortisol. Chronic stress is thought to lead to this abnormal diurnal cortisol pattern, but an exact psychosocial mechanism linking status, stress, and cortisol is still under investigation (discussed below).

Mere dysregulation of the HPA-axis is not the end of the potentially detrimental consequences of low status. Abnormal diurnal cortisol patterns have been studied as contributing to several pathologies that occur at higher rates among low-SES individuals, including high blood pressure (Phillips et al. 2000), strokes, cardiovascular disease, and Type 2 diabetes (Rosmond and Björntorp 2000a). Flatter diurnal cortisol slopes are also associated with increased all-cause mortality, especially due to cardiovascular disease (Kumari et al. 2011), and with earlier mortality due to breast cancer (Sephton et al. 2000).

Cortisol Reactivity and Status The previous work implicates low status in high-basal cortisol and blunted cortisol rhythms throughout the day. Some evidence also links low status to cortisol increases in response to acute physical and psychosocial stress. Lower-SES individuals show a greater rise in cortisol in response to laboratory psychosocial stressors than high-SES individuals (Fiocco et al. 2007; Kristenson et al. 1998; Adler et al. 2000). In another study low-SES individuals showed cortisol hyperactivity compared to high-SES individuals following a pharmacological challenge of HPA-axis functioning (Rosmond and Björntorp 2000b). In response to a dexamethasone suppression test—a test of the ability of the HPA-axis to suppress

cortisol secretion in response to dexamethasone, a cortisol agonist—low-SES individuals did not reduce cortisol concentrations as effectively as high-SES individuals (Rosmond and Björntorp 2000b). Taken together, these studies suggest that a given stressor will result in stronger activation of the HPA-axis and a slower return to baseline for a low-status individual. Repeated over the course of a day or life, this augmented reaction and prolonged recovery will expose the individual to more cortisol and to the concomitant negative effects of cortisol.

Mediators of the Status-Cortisol Link

The body of research reviewed above suggests that status is negatively related to cortisol concentrations in humans as measured in basal concentration, diurnal patterns, and acute fluctuations of cortisol concentration. Several variables have been proposed as putative mechanisms of this relationship, of which we will review four: Health behaviors, sense of control, hostility, and social support.

Health Behaviors Health behaviors have been investigated as an explanation for the relationship between status and cortisol, specifically within the context of SES. Several health behaviors were found to explain the relationship between measures of cortisol dysregulation and SES, including increased alcohol and tobacco use (Cohen et al. 2006a, b). Tobacco directly stimulates cortisol secretion in the short-term and increases basal cortisol concentrations in current smokers compared to ex- and non-smokers (Badrick et al. 2007). Alcohol use also raises basal cortisol concentrations (Thayer et al. 2006; Bernardy et al. 1996).

The psychological relationship between alcohol/tobacco use and status is less well understood. These behaviors exist at higher rates in low-SES environments, but it is unclear whether they are a response to or product of the environment, or some combination of both (Krueger and Chang 2008). Increased alcohol and tobacco use may reflect stress-induced derailment of impulse control or self-regulatory processes (Muraven and Baumeister 2000; Hull and Slone 2004). Or perhaps alcohol and tobacco consumption reflect a coping mechanism for dealing with low-SES stress, as both products are believed (by the users) to relieve tension and improve negative affect (Pampel et al. 2010; Hull and Slone 2004). Alternatively, the increased usage could result from environmental cultural norms, lower-education levels, or simply having less reason to invest in the future (Cutler and Lleras-Muney 2008; Pampel et al. 2010).

Sense of Control In stressful situations, lacking control over the stressor is known to produce substantial increases in cortisol compared to stressors that are directly controlled or predictable by an individual (Dickerson and Kemeny 2004). Being in a position at the top of a hierarchy lends itself to being or feeling in control, even over variables one could not possibly control such as the outcome of a roll of a die (Fast et al. 2009). So when a stressor is experienced by a high-status individual, she may be more likely to feel in control or actually be in control of that stressor, which

in turn attenuates the cortisol response. Consistent with this idea, self-reported sense of control accounts for the attenuating effect of status on cortisol when measured broadly as control over life circumstances and outcomes (Cohen et al. 2006b) and as interpersonal control over subordinates (Sherman et al. 2012). Indeed, instilling a sense of control has been suggested as an intervention for combating the negative effects of low SES. The "shift and persist" intervention proposes to improve stress coping of low-SES individuals by shifting attention toward what can be controlled within a stressful situation (Chen and Miller 2012). Redirecting attention to maximize one's sense of control may reduce the stress response, minimizing exposure to cortisol and the negative effects associated with increased cortisol concentration. The "persist" half of the proposed intervention relates to maintaining optimism and resiliency in the face of life's stressors. These traits have been found to predict lower-basal cortisol in individuals, even at lower SES (Lindfors and Lundberg 2002; Ryff et al. 2004).

Hostility Hostility is defined as an individual's proneness to anger and aggressive behavior based on a distrustful view of others (Kubzansky et al. 1999). Hostility is negatively related to SES, likely as a response to the difficult circumstances inherent to being at the bottom of a hierarchy (Elovainio et al. 2001). Hostile individuals tend to respond antisocially to stressors, which in turn begets further interpersonal hostility (Smith 1994; Gallo et al. 2006). Hostility may underlie the cyclic and detrimental nature of stress and low status: Continual stress may lead to the development of a hostile nature that in turn invites additional provocation from others, becoming an added stressor in itself. High-trait hostility also affects HPA-axis function, associating with increased daytime cortisol secretion and flattened cortisol slope (Pope and Smith 1991; Ranjit et al. 2009). Yet, while hostility has *not* been found to explain the relationship between SES and cortisol specifically, it does account for the relationship between SES and a summative measure of stress physiology, referred to as allostatic load, which incorporates cortisol as one of several physiological markers of stress (Hawkley et al. 2011; Kubzansky et al. 1999). Hostility explains the physiological repercussions of low-status stress, but more research is necessary to clarify if hostility relates directly to cortisol functioning or only to broader stress physiology.

Social Support The social networks of nonhuman primates provide several routes for high-status members of the hierarchy to cope with encountered stressors, for example, by being groomed by or aggressing toward subordinates (Sapolsky 2005). Likewise, humans generally benefit from social support, which has been shown to relate to lower-basal cortisol concentrations (Pinquart and Sörensen 2000; Uchino 2006; Taylor et al. 2000) and to reduce the cortisol response to acute laboratory stressors (Heinrichs et al. 2003). Unreliable social networks and perceptions of social isolation meanwhile are stressors in themselves (Uchino 2006; Hawkley et al. 2012).

Since one's status is dependent on the opinions of others, having high status may provide more social capital to buffer one's stress response, while low status may not provide as rich of social networks on which to rely. Accordingly, low SES

correlates with weaker social connections and less diverse social networks, which then accounts for increased cortisol concentration (Cohen et al. 2006a, b). The reasons for this negative relationship between SES and social support may stem from distrusting one's neighbors in low-income neighborhoods (Subramanian et al. 2003; Brehm and Rahn 1997) or from an inability to provide or seek support due to the chronic stressors inherent to low SES (Cattell 2001). Future work that elucidates the exact aspect of social support that mediates this relationship could provide targets to ameliorate some of the negative consequences of low SES.

Experimental Evidence for Status-Cortisol Link

The reviewed work indicates that status correlates negatively with cortisol in naturalistic studies, but the correlational nature of the evidence prohibits making causal statements of the direction of the effect for cortisol and status in human hierarchies. Although few experiments have focused on cortisol and status, recent work has begun to investigate causal explanations of the relationship between status and cortisol.

Status Causally Alters Cortisol Low status causally dysregulates HPA-axis activity in experimentally manipulated status hierarchies of nonhuman primates and other social animals, with results suggesting lower status leads to increased cortisol concentrations. In female rhesus macaques, subordination disrupts normal HPA-axis functioning, decreasing the cortisol awakening response and prolonging cortisol release following a stressor (Michopoulos et al. 2012). The relentless stress of low status causes diurnal and acute cortisol dysregulation in the lower-ranking macaques, an effect that has been found in experiments with several other species of social animals (cynomolgus monkeys: Jayo et al. 1993; mice: Avitsur et al. 2001; domestic pigs: Mendl et al. 1992; zebrafish: Filby et al. 2010).

While status appears to alter cortisol activity in the species of various social animals, this experimental hypothesis has not been extensively tested in humans. The few studies that have experimentally tested the effects of status in humans show that status inversely affects cortisol concentrations, similar to the results seen in longitudinal and cross-sectional studies. Cortisol increased following social stressors when low status was randomly assigned in an experimental hierarchy, while cortisol reactivity was attenuated when high status was assigned (Carney et al. under review). A separate study found that adopting postures typically associated with nonverbal displays of high and low rank in a hierarchy affected cortisol concentrations. The so-called "power positions"—which include an open, expansive posture for high rank and a diminutive posture for low rank—decreased cortisol for high-rank body postures and increased cortisol for low-rank postures (Carney et al. 2010). This evidence indicates that experimental manipulations of social rank in a hierarchy affect cortisol concentration and cortisol reactivity to stressors in humans. In addition to long-term effects on the HPA-axis, social status seems to have immediate consequences for cortisol functioning.

Cortisol Causally Alters Status While this experimental research indicates that status causally influences cortisol concentrations, some evidence suggests the reciprocal relationship may be true as well. Animal studies have shown that glucocorticoids affect the formation of hierarchies as part of dominance contests. Administering corticosterone (the rodent analog to cortisol) to subordinate rats following defeat in a social interaction improves the memory for and maintenance of the hierarchy (Timmer and Sandi 2010). In a study on rainbow trout, cortisol implanted under the animals' skin affected the results of competitive interactions with similar-sized fish and smaller conspecifics (DiBattista et al. 2005). Compared to a control condition, cortisol significantly decreased the likelihood of the treated trout to become the dominant fish in both the similar- and smaller-sized pairing. This limited evidence from animal research suggests that glucocorticoids may affect both the establishment of and memory for a status hierarchy through dominance contests. These results have not been tested in humans *per se* (who are unable to hold their breath long enough to replicate these results), but cortisol may affect psychological variables relevant to hierarchical behavior. For example, cortisol disrupts social approach and avoidance behaviors (Roelofs et al. 2005; van Peer et al. 2007), which are theoretically important to earn and maintain status (Anderson and Berdahl 2002). An exact role for cortisol in causally determining status within human hierarchies awaits future experimental research.

Cortisol Responses to Competition

Research on SES and status often studies status within static hierarchies, but the negative relationship between status and cortisol is found when the stepwise progress of obtaining high or low status in competitive settings is examined as well. As reviewed in the T section above, a dynamic model of status is found in naturalistic dominance contests like sporting events, laboratory competition, and political elections. Correlational studies of competitions show that gaining or maintaining status by winning a competition or supporting the winning side of a competition is associated with a drop in cortisol, while losing a competition or supporting the losing side is associated with a rise in cortisol (Stanton et al. 2010; Jimenez et al. 2012; Bateup et al. 2002).

Yet several studies have found a null relationship between competition outcome and cortisol (e.g., Oliveira et al. 2009), or even increased cortisol after winning (Suay et al. 1999). The inconsistent nature of this association suggests that factors other than competition outcome play a role in determining the relationship between dynamic changes in status in competition and cortisol. Similar to the research reviewed earlier on testosterone (Schultheiss et al. 2005), implicit power motive—a measure of a person's unconscious motivation to dominate others—is linked to cortisol responses to victory and defeat (Wirth et al. 2006). Individuals high in implicit power motive experienced rises in cortisol after losing a dominance contest and drops in cortisol after winning, whereas those low in implicit power

motive showed the opposite pattern of cortisol changes (Wirth et al. 2006). These findings suggest that being relegated to low-status positions (losing a competition) may cause cortisol to increase only for those individuals motivated to achieve high status (individuals high in power motive). For those individuals who do not desire high-status positions (individuals low in power motive), achieving high status may actually be stressful resulting in cortisol increases (Wirth et al. 2006). The role of individual differences and social factors in moderating the relationship between status and cortisol is an important topic for future research, a point we expand upon toward the end of the chapter.

The Dual-Hormone Hypothesis: Testosterone × Cortisol Interactions and Status

Social endocrinology research—and this chapter, so far—typically examines independent effects of hormones on behavior. While this approach has been fruitful for identifying some hormone-behavior relationships relevant to status, it may also contribute to inconsistent findings. For example, many studies find null associations between testosterone and status-related behaviors such as aggression (Archer 1998), and other studies find null associations between cortisol and status (Gadinger et al. 2011). These inconsistencies may arise because testosterone and cortisol may work interactively—not independently—to affect social behaviors linked to status. According to the *dual-hormone hypothesis* (Mehta and Josephs 2010), testosterone and cortisol should jointly regulate behavior such that testosterone should be positively related to status-seeking behaviors—and in turn, higher status—only when cortisol concentrations are low (Mehta and Josephs 2010). When cortisol concentrations are high, testosterone's effect on status-seeking behaviors should be blocked. A series of recent studies provide strong empirical support for the dual-hormone hypothesis (Edwards and Casto 2013; Mehta and Josephs 2010; Popma et al. 2007). For example, one study collected afternoon saliva samples to measure basal testosterone and cortisol and then videotaped participants in a position of leadership (Study 1, Mehta and Josephs 2010). Seven judges rated the leaders on nineteen social behaviors linked to dominance (e.g., engaged, gave clear instructions, directive, leader-like, confident, nervous, uncomfortable). All nineteen behaviors were aggregated to create an overall dominance factor (items such as nervous and uncomfortable were reverse scored prior to aggregation). As shown in Fig. 13.2a, dual-hormone profiles of T and C interactively predicted dominant leadership behaviors. Basal T was positively related to dominance only among leaders low in basal C (Fig. 13.2a, solid line), but basal T was unrelated to dominance among leaders high in basal C (Fig. 13.2a, striped line).

Social dominance is a key behavioral route to attaining high status across species (Anderson and Kilduff 2009b; Mazur and Booth 1998). Thus, the dual-hormone interaction may not only be related to dominant behaviors, but also to who attains high status. Two recent studies linked the testosterone-cortisol interaction

Fig. 13.2 Evidence for the
dual-hormone hypothesis in
three studies. **a** Adapted from
Study 1, Mehta and Josephs
(2010); **b** Adapted from
Edwards and Casto (2013).
c Adapted from Mehta et al.
(2013)
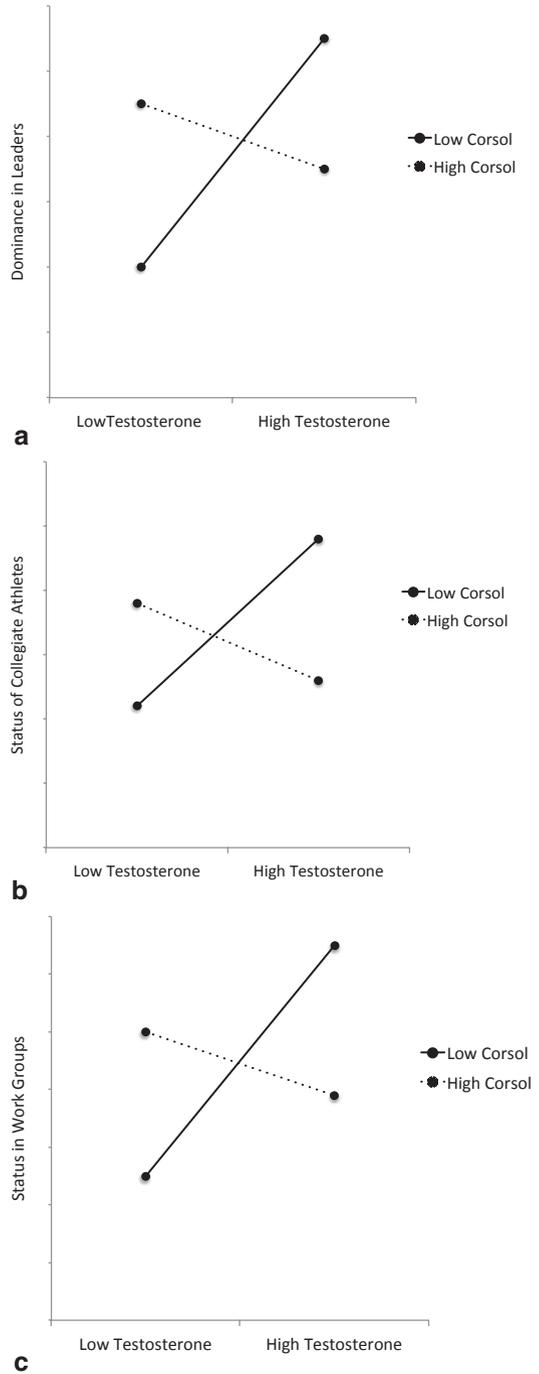
to social status. Edwards and Casto (2013) measured basal hormone levels in approximately 90 female collegiate female athletes (on soccer, softball, volleyball, and tennis teams) and collected measures of status within their teams. As shown in Fig. 13.2b, there was a dual-hormone interaction that closely matched Mehta and Josephs (2010). Higher testosterone was positively related to social status only among athletes low in basal cortisol (Fig. 13.2b, solid line), but basal testosterone and status were unrelated among athletes high in basal cortisol (Fig. 13.2b, striped line). A third study measured dual-hormone profiles, dominance, and status in MBA students at an elite business school. Students provided afternoon saliva samples and were randomly assigned to small work groups several weeks later (Mehta et al. 2013). The groups were videotaped performing a group decision-making task. At the end of the task, all participants ranked their fellow group members on leadership. These leadership rankings were aggregated to create an overall measure of status. Research assistants watched the videos and coded for dominant behaviors. Consistent with Mehta and Josephs (2010) and Edwards and Casto (2013), the testosterone-cortisol interaction predicted leadership rank (Fig. 13.2c) and dominant behaviors. Moreover, dominance partially mediated the association between the dual-hormone interaction and leadership rank. Overall, these studies demonstrate that the testosterone-cortisol interaction predicts the attainment of status through dominant behaviors. Higher testosterone is positively related to social dominance and in turn higher status only among individuals with low cortisol, but testosterone is unrelated to social dominance or status among individuals with high cortisol.

The underlying physiological mechanisms for dual-hormone effects on behavior remain unknown. Mehta and Josephs (2010) speculate that these dual-hormone interactions may occur through an inhibitory effect of cortisol on the pathway between testosterone and behavior. When cortisol is low, the pathway between testosterone and behavior functions efficiently, and higher testosterone should have a strong effect on behaviors such as dominance. When cortisol is high, the pathway between testosterone and behavior may be blocked (e.g., by down-regulating androgen receptors, cf. Mehta and Josephs 2010). Testosterone and cortisol may also interact on a psychological level given testosterone's association with status-seeking motivation and cortisol's association with social approach-inhibition. A combination of high status-seeking motivation (high testosterone) and social approach (low cortisol) may lead to social dominance and higher status, whereas a combination of high status-seeking motivation (high testosterone) and social inhibition (high cortisol) may cause submissive behaviors and lower status. These mechanisms fit within a broader evolutionary approach to understanding the roles of the stress (HPA) and reproductive (HPG) axes in modulating complex social behavior (Carré and Mehta 2011). High environmental stress (high cortisol) may inhibit the effect of testosterone on reproductively relevant behaviors such as competitive behavior and dominance, because such behaviors are metabolically costly and potentially dangerous. Only when environmental stress is low (low cortisol) might behaviors relevant to the pursuit of status be expressed. Nevertheless, these proposed mechanisms are highly speculative and lack direct empirical support. Delineating the precise pathways that give rise to testosterone-cortisol interactions awaits further research.

Estradiol

Estradiol is the most prevalent and potent molecule of the class of steroid hormones known as estrogens, which are typically thought of as the female sex hormones (McCarthy 2008; Stanton and Edelstein 2009). The set of biochemical reactions necessary to produce estradiol is complex: Estradiol starts as a molecule of cholesterol, the precursor to all steroid hormones, which is then converted to a glucocorticoid. An androgen molecule is produced next before it is finally converted to estradiol. This process occurs primarily in the ovarian granulosa cells of human and nonhuman females, as well as in the adrenal cortices in females and males (McCarthy 2008). Stimulation of estradiol secretion, like testosterone, is the end product of a cascade of hormones from the HPG-axis and mainly relates to female reproductive processes. As part of the menstrual cycle, the pulsatile release of gonadotropin-releasing hormone (GnRH) from the hypothalamus and follicle-stimulating hormone (FSH) from the pituitary stimulates a surge of estradiol. When this rapid increase on estradiol reaches a certain threshold, estradiol increases luteinizing hormone (LH) secretion from the pituitary, which in turn induces ovulation (Plant 2012). Sustained increases of estradiol during puberty are responsible for the development of female secondary sex characteristics and for skeletal growth in males and females (Rogol et al. 2002). Placental tissue also secretes estradiol, which contributes to maintaining pregnancies (Albrecht et al. 2000).

While testosterone relates to concern for status in men and women, the role of T in female status-relevant behavior is tenuous (Mazur and Booth 1998). But this may be due to the important role estradiol, and not testosterone, plays in fertility and reproductive behavior in women such that estradiol underlies female status-seeking and dominant behavior (Stanton and Schultheiss 2009; Schultheiss 2007). An evolutionary perspective suggests that during the fertile period of the menstrual cycle, gaining a high-status position might improve fitness since high status would garner access to benefits like the best mates, most food, and social support (c.f. Stanton and Edelstein 2009). Thus, because ovulation is induced by a surge of estradiol, this hormone may promote competitive or dominant behaviors in females in response to the advantages high status holds for fertile females.

In line with this theory, early research on female chimpanzees has shown that administering estradiol enhances status by increasing dominant behavior within a small, female hierarchy (Birch and Clark 1946). In humans, higher-basal estradiol concentrations relate to higher measures of implicit dominance motives, an indication of a preference for power and higher ranking in hierarchies (Stanton and Schultheiss 2007; Stanton and Edelstein 2009). In fact, as further evidence of the evolutionary link between estradiol and status, the relationship between estradiol and implicit dominance is stronger in single women than women in romantic relationships (Schultheiss et al. 2003; Stanton and Schultheiss 2007; Stanton and Edelstein 2009). Lacking a sexual partner, single women theoretically have a greater incentive to compete for status in response to the endocrine signal of ovulation and consequently show a stronger correlational relationship between estradiol and implicit dominance.

Changes in estradiol concentration are also associated with dominance and status in laboratory studies, much like men's testosterone fluctuations in competition. In women who scored high on implicit dominance, winning a competition was associated with an influx of estradiol while losing resulted in diminished estradiol concentration (Stanton and Schultheiss 2007; Stanton and Edelstein 2009). Women low in implicit dominance did not show this pattern of estradiol response. Estradiol thus relates to an implicit drive for dominance and status in females and responds to changes in status for individuals with high implicit dominance.

Some evidence suggests that the effects of estradiol on status are only found in interfemale competition. Theoretically, female primates in the fertile phase of their menstrual cycle are only in competition with other females for status and mates; males are competed over, not usually competitors in this regard. Estradiol then may only augment female dominance or status in same-sex hierarchies. The literature cited above all relied on female-female hierarchies and competitions but did not test for effects of competitor gender. One recent study supports the concept that estradiol specifically alters female-female competition by demonstrating that women with high-estradiol levels are more competitive in negotiations with other women, but not men (Severance 2011). Future work should continue to explore the role of estradiol in competition while examining social moderators like competitor gender.

Oxytocin

Oxytocin is a neuropeptide hormone produced in the hypothalamus and secreted from the posterior pituitary that has recently received significant attention as a neuroendocrine modulator of social behavior and social cognition (Bartz et al. 2011). Early work with the hormone showed its importance in peripheral physiology, especially in regards to maternal processes and parturition. For example, oxytocin produces uterine and cervical contractions, stimulates the milk let-down reflex in the mammary glands, and is used clinically to induce labor (Salonia et al. 2005; Bethlehem et al. 2013). In animal studies, oxytocin additionally induces maternal behaviors like maternal care in rodents and maternal bonding in sheep which, when coordinated with the oxytocin-induced maternal physiology, aid in the survival of offspring (Ross and Young 2009). Human oxytocin similarly relates to parental behaviors, with plasma and saliva concentrations predictive of postpartum, parent-child bonding, attachment, and infant monitoring (Feldman et al. 2007, 2010, 2011; Galbally et al. 2011). Oxytocin also positively relates to levels of bonding and interpersonal attraction in romantic relationships (Grewen et al. 2005; Tops et al. 2007), though some work indicates that oxytocin may actually signal relationship distress and a concomitant desire for more social contact (Turner et al. 1999; Taylor et al. 2006).

While oxytocin alters peripheral physiology and seems to relate to parental and interpersonal bonding in humans, associating peripheral oxytocin concentration to
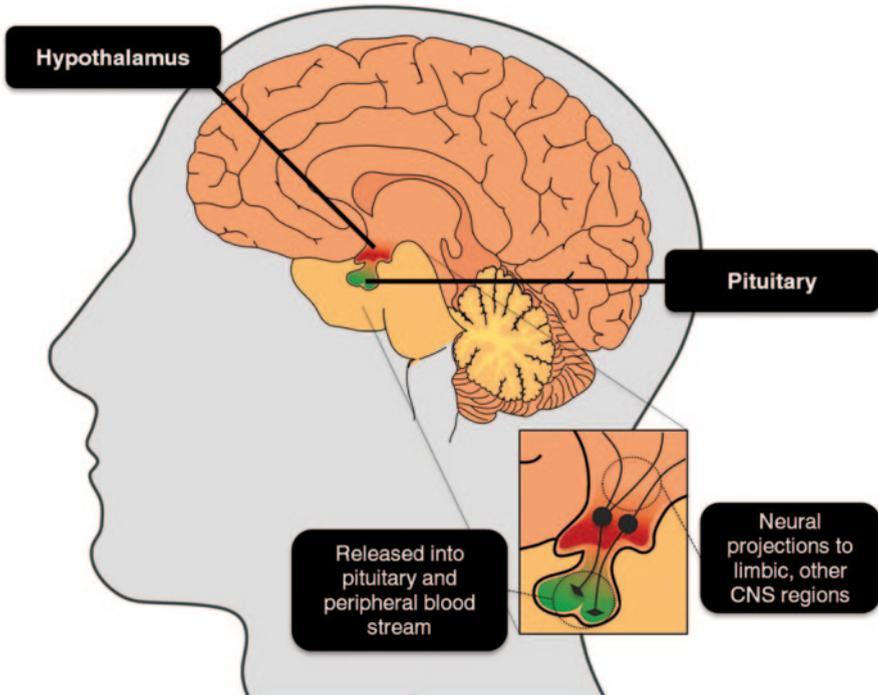
Fig. 13.3 Oxytocin is produced in the hypothalamus. Neural projections release oxytocin to limbic regions and other areas of the brain. Oxytocin is also released into the pituitary, where it is then secreted into the peripheral blood stream and carried to the rest of the body (Bethlehem et al. 2013)

social behavior remains controversial. The oxytocin molecule does not readily pass through the blood-brain barrier, the restrictive capillary anatomy that prevents certain molecules from entering the extracellular space of the central nervous system (Churchland and Winkeilman 2012). Unlike other hormones reviewed in this chapter, oxytocin secreted from the pituitary does not diffuse into the central nervous system, and thus peripheral basal concentration (i.e., measured in saliva or blood) does not correlate with cerebral spinal fluid concentrations (Kagerbauer et al. 2013). Instead, oxytocin is released into the central nervous system from neurons that project from the hypothalamus to limbic and other neural regions at concentrations that are distinct from peripheral levels (Bethlehem et al. 2013; Ross and Young 2009; Bartz et al. 2011; Fig. 13.3).

Oxytocin and Intergroup Status

Research on the prosocial effects of oxytocin in humans has accelerated in the past decade due to the development of an inhalable form of the hormone (Bartz et al. 2011). Intranasal administration of oxytocin deposits the hormone directly in the

cerebrospinal fluid where it can alter central neural physiology (Born et al. 2002). Initial work pointed to oxytocin directly increasing prosocial behaviors, earning oxytocin the monikers "love drug" and "cuddle chemical" (de Dreu 2012; Bartz et al. 2011). Broadly, oxytocin increases trust, generosity, and cooperation in economic decision-making games (Kosfeld et al. 2005; Zak et al. 2007) and increases interpersonal perceptions of trustworthiness and attractiveness (Zak et al. 2005; Theodoridou et al. 2009). Exogenous oxytocin also affects social cognition, improving memory for positively valenced faces (Guastella et al. 2008; Rimmele et al. 2009; Marsh et al. 2010) and improving empathic accuracy when the task is difficult (Bartz et al. 2011).

Yet, when examined in a social context, oxytocin appears to only promote prosocial behaviors directed toward in-group members (de Dreu 2012). So rather than affecting an individual's status like testosterone or estradiol, oxytocin affects behaviors related to improving the status of one's in-group. For example, oxytocin increased the likelihood for men to self-sacrifice economically for the benefit of an experimentally assigned in-group in a competition (de Dreu et al. 2010), especially when an in-group member is perceived to be threatened or vulnerable (de Dreu et al. 2012b). The men given oxytocin also reported a higher level of trust that their in-group members would reciprocate the self-sacrificial behavior, but did not exhibit an increase in distrust in or derogatory behavior toward the out-group.

The costly, prosocial behaviors found in these studies may signal an individual's unselfishness, generosity, and resource wealth to the other members of the group, who may then confer greater status to the altruistic individual (Hardy and Van Vugt 2006; Willer 2009). For example, donators to a public fund were rated as having higher status compared to nondonators and, in a second experiment, individuals who donated to in-groups but not competing out-groups were granted higher status than individuals who donated equally to in-groups and out-groups (Halevy et al. 2012). So while oxytocin-altered, altruistic behaviors directed toward in-group members (e.g., De Dreu et al. 2010) may result in individual status gains, the direct relationship between oxytocin and individual status has not yet been experimentally tested.

Exogenous oxytocin also affects unconscious cognitive processes that alter moral decisions and promote positive associations with the in-group. Oxytocin decreased the likelihood to sacrifice a member of a cultural in-group to save an unnamed group of individuals from impending doom (e.g., a runaway trolley in the moral choice dilemma problems; de Dreu et al. 2011). After administering oxytocin, men also responded more quickly to stimuli that paired in-group members' names with positively valenced words on the Implicit Association Test (IAT; Greenwald et al. 2009) and more slowly to out-group members' names as an implicit sign of preference for in-group members and disdain for out-group members, respectively (de Dreu et al. 2011). All together, these exogenous administration studies demonstrate oxytocin's influence on behaviors and psychology that benefit the in-group in competitive and noncompetitive settings, which result in promoting an in-group's status over an out-group.

Oxytocin also improves group dynamics, specifically altering behavior related to the process of forming a group and to cohesiveness within a group. When picking

teams, for example, dominant-looking individuals may prove more useful on one's own team rather than on an opposing team. Accordingly, oxytocin increases the preference for alliances with men with threatening, dominant facial features as opposed to less threatening, trustful features in an intergroup competition (De Dreu et al. 2012a). And once a team is formed, cohesion among a group's members is an important predictor of the performance of the group (Evans and Dion 1991). Oxytocin improves this aspect of in-group functioning as well, biasing subjective judgments towards agreement with the in-group and disagreement with the out-group (Stallen et al. 2012). When in-group and out-group members' ratings of the attractiveness of inanimate objects were at odds, males given oxytocin more often conformed to in-group ratings, an effect not seen in participants given placebo.

Interestingly, the effects of oxytocin on group-level status do not depend on the extent of the differences between in-group and out-group or on the inherent importance of the in-group to an individual. Instead, oxytocin stimulates these prosocial, in-group behaviors across a variety of in- and out-groups. De Dreu et al. (2011) showed that oxytocin produced prosocial effects directed towards the in-group when the out-group was defined in terms of two different cultural out-groups (i.e., names of German or undefined Arabic descent for Dutch participants). Oxytocin even bolstered in-group status in arbitrarily defined, experimental groups that do not contain implicit cultural importance (Stallen et al. 2012; de Dreu et al. 2010, 2012a, b). In light of these findings, oxytocin seems to amplify in-group bias in minimal groups (Tajfel 1982), meaning that any grouping—experimentally or culturally defined—is enough for oxytocin to affect the promotion of the in-group's status over the out-group.

In sum, elevated levels of oxytocin seem to promote group-level status by increasing altruistic, protective behaviors toward an in-group and positive cognitive associations and decisions with in-group members. One caveat from this literature is the dependence on male participants in these intranasal oxytocin administration experiments. Safety concerns for women (e.g., the potential to induce labor) and the degree to which oxytocin concentration fluctuates during the menstrual cycle (Salonia et al. 2005) make males a convenient sample to study. Yet a recent study suggests exogenous oxytocin differentially affects males and females. Intranasal oxytocin improves the ability to label ambiguous social interactions but this effect was only true for women viewing kinship interactions and for men viewing competitive interactions (Fischer-Softy et al. 2013). Future research must determine if similar effects on group-level status emerge in women given oxytocin, or if these effects are only found in males.

Effects of Status on Oxytocin

Little work has focused on the effects of status on endogenous oxytocin concentrations due to the problems of inferring central effects from peripheral oxytocin and due to the safety concerns of measuring oxytocin in cerebrospinal fluid. But it is

possible that oxytocin is generally increased in well-regarded, high-status individuals as they may experience more prosocial interactions and positive attention from lower-ranking members due to the esteem associated with high-status positions. Consistent with this idea, high status in experimentally determined, female rhesus macaque hierarchies boosts oxytocin concentration compared to low-status positions (Michopoulos et al. 2011). In this case, grooming and submissive behaviors directed toward the high-status members increased as a result of their position in the hierarchy, which in turn augmented oxytocin concentration for the high but not low-status members. Although work in humans has so far focused on the effects of oxytocin on group status, one's individual status may affect oxytocin concentrations, especially within stable, decorous hierarchies.

Future Directions

Moving forward, research on the social endocrinology of status will benefit from considering the effects of social and biological moderators, neural mechanisms, health implications, other hormones, and greater attention to the diverse ways in which humans attain status. Considering these factors will produce a more comprehensive model of the influence of hormones within hierarchies.

Social and Biological Moderators

More work on social moderators within hierarchies will expand our understanding of the biosocial mechanisms of status. For example, most work on humans has focused on the negative association between status and cortisol within stable status hierarchies where there is no potential to gain or lose status. But in unstable hierarchies, high-status individuals may fear losing their status (Jordan et al. 2011), undermining their sense of control and increasing their psychological stress. Low-status individuals may hope for a better position in the hierarchy, and a "nothing-to-lose" perspective may result in lower stress. Hence, cortisol and stress may be higher in high status compared to low-status individuals in unstable hierarchies. Evidence supports this possibility in animals (Sapolsky 2005), but the effects of hierarchical instability on endocrine function and status in humans remain unclear.

Genes that affect hormone receptor and neurotransmitter function also present interesting avenues for future research on hormones and hierarchies. Testosterone influences social behaviors by binding to androgen receptors, implying that testosterone should have a stronger effect on status-relevant behaviors in individuals with heightened androgen receptor function. One study linked variability in the androgen receptor gene to status-seeking behaviors in men (i.e., self-reported dominance and prestige; Simmons and Roney 2011). This study failed to find evidence for an interaction between the androgen receptor gene and basal testosterone levels,

but the possibility remains that fluctuating levels of testosterone may interact with androgen receptor genes to influence social status. Other research shows that basal testosterone interacts with the serotonin transporter gene (5-HTTLPR) to regulate status-related processes. Individuals with high testosterone showed a stronger cortisol response to status-related stressors when these individuals were also carriers of the short allele of 5-HTTLPR (Josephs et al. 2012). Although untested, it is possible that this serotonergic gene by testosterone interaction may underlie behavioral responses to status threats as well.

Neural Mechanisms

Another aspect of the social endocrinology of status that needs further attention is the neural correlates of hormones and hierarchies. Studies utilizing hormones and functional magnetic resonance imaging (fMRI) have documented neural networks associated with testosterone (Höfer et al. 2013), oxytocin (Bethlehem et al. 2013), estradiol (Maki and Resnick 2001; Craig et al. 2008), and cortisol (Dedovic et al. 2009; Lovallo et al. 2010) that may underlie the effects these hormones have on status-relevant behavior. While some studies have begun to elucidate neural networks important for the perception of status (Chiao et al. 2009; Zink et al. 2008), the next leap forward for social endocrinology is to isolate neural networks that explain the relationship between hormones and behaviors related to dominance and status-seeking (e.g., reduced orbitofrontal cortex activity as a mediator of the association between testosterone and status-seeking behavior, Mehta and Beer 2010).

Health

Several decades of research have demonstrated that low status is associated with increased morbidity and mortality from cardiovascular disease, Type-2 diabetes, and obesity (Li et al. 2007; Rosmond and Björntorp 2000a), even in a continuously employed sample with universal access to healthcare (Chandola and Marmot 2010). Cortisol dysregulation has been implicated in leading to these poor health outcomes (Adler et al. 1994; Li et al. 2007), but future work should examine the causal relationships between acute stress, cortisol, and poor health outcomes inherent to low status. Other hormones should be investigated as well. For example, oxytocin and polymorphisms in its receptor gene reduce the cortisol (Chen et al. 2011) and cardiovascular (Norman et al. 2012) responses to stress. Thus, oxytocin could represent a pathway by which social connections alter health within a status hierarchy. Estradiol and testosterone are also thought to relate to cardiovascular health through subtle alterations of cardiovascular functioning (Pérez-López et al. 2010), though more research on these hormones is needed in the context of status hierarchies and health.

Vasopressin and other Hormones

Arginine vasopressin, or just vasopressin, is a neuropeptide hormone that has attracted attention as a potential modulator of status and dominant behavior in animal studies, but little is known about vasopressin's role in human social status. Like the molecularly similar hormone oxytocin, vasopressin improves social memory and cooperation (Benarroch 2013) but, unlike oxytocin, vasopressin *augments* the cortisol response to social stressors (Shalev et al. 2011). Additionally, the effects of testosterone on certain aspects of status may depend on an interaction with vasopressin. Flank marking and territorial aggression in Syrian hamsters, both indicators of status, depend on interactions between testosterone and vasopressin concentrations (Koolhaas et al. 1990; Albers and Cooper 1995). In humans, testosterone's role in coordinating a response to status threat likely works through vasopressin-mediated neural pathways (Bos et al. 2012), though few human studies have examined vasopressin in this context. Future work should focus on the effects of vasopressin in human hierarchies, particularly in relation to its interaction with testosterone. Other steroid hormones such as dehydrepiandrosterone (DHEA, and its sulfate form DHEA-S) may also be involved in behaviors implicated in status, possibly by buffering emotional responses to social stressors (Akinola and Mendes 2008).

Multiple Routes to Status

Social endocrinology research has focused almost exclusively on dominance as a behavioral route to individual status attainment, but humans rise in social hierarchies not only through dominance but also through prosocial behaviors such as building social connections and sharing expertise (Anderson and Kilduff 2009b; Cheng et al. 2013). More research is needed on the social and neuroendocrine mechanisms for these other routes to status. Although some research suggests that testosterone suppresses cooperative behaviors (Mehta et al. 2009), a recent study showed that testosterone enhances prosociality in contexts in which such behaviors may be beneficial for status. Testosterone administration decreased prosocial behavior when there was a threat in the social environment (the threat of betrayal), but testosterone boosted prosocial behavior when there was no threat (a benevolent social interaction) (Boksem et al. 2013). The authors speculate that testosterone may have increased prosociality in the nonthreatening situation because building social connections may be more important than dominance for achieving status in this context. Oxytocin and progesterone are other candidate hormones that may help an individual gain status through affiliative behaviors (Bartz et al. 2011; Wirth and Schultheiss 2006), but no research to date has studied these hormones in the context of individual-based (as opposed to group-based) status hierarchies. Future research should continue to examine the biosocial mechanisms that regulate prosocial routes to gaining and maintaining status in human hierarchies.

References

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: What we know and what we don't. *Annals of the New York Academy of Sciences, 896,* 3–15.

Adler, N. E., Boyce, T., Chesney, M. A, Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: The challenge of the gradient. *The American Psychologist, 49,* 15–24.

Adler, N. E., Epel, E. S., Castellazzo, G., & Ickovics, J. R. (2000). Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in healthy white women. *Health Psychology, 19,* 586–592.

Agbedia, O. O., Varma, V. R., Seplaki, C. L., Seeman, T. E., Fried, L. P., Li, L., et al. (2011). Blunted diurnal decline of cortisol among older adults with low socioeconomic status. *Annals of the New York Academy of Sciences, 1231,* 56–64.

Akinola, M., & Mendes, W. B. (2008). The dark side of creativity: Biological vulnerability and negative emotions lead to greater artistic creativity. *Personality and Social Psychology Bulletin, 34,* 1677–1686.

Albers, H. E., & Cooper, T. T. (1995). Effects of testosterone on the behavioral response to arginine vasopressin microinjected into the central gray and septum. *Peptides, 16,* 269–273.

Albrecht, E. D., Aberdeen, G. W., & Pepe, G. J. (2000). The role of estrogen in the maintenance of primate pregnancy. *American Journal of Obstetrics and Gynecology, 182,* 432–438.

Anderson, C., & Berdahl, J. L. (2002). The experience of power: Examining the effects of power on approach and inhibition tendencies. *Journal of Personality and Social Psychology, 83,* 1362–1377.

Anderson, C., & Kilduff, G. J. (2009a). Why do dominant personalities attain influence in face-to-face groups? The competence-signaling effects of trait dominance. *Journal of Personality and Social Psychology, 96,* 491–503.

Anderson, C., & Kilduff, G. J. (2009b). The pursuit of status in social groups. *Current Directions in Psychological Science, 18*, 295–298.

Archer, J. (1998). Problems with the concept of dominance and lack of empirical support for a testosterone-dominance link. *Behavioral and Brain Sciences, 21,* 363–363.

Archer, J. (2006). Testosterone and human aggression: An evaluation of the challenge hypothesis. *Neuroscience and Biobehavioral Reviews, 30,* 319–345.

Avitsur, R., Stark, J. L., & Sheridan, J. F. (2001). Social stress induces glucocorticoid resistance in subordinate animals. *Hormones and Behavior, 39,* 247–257.

Badrick, E., Kirschbaum, C., & Kumari, M. (2007). The relationship between smoking status and cortisol secretion. *Journal of Clinical Endocrinology & Metabolism, 92,* 819–824.

Bartz, J. A., Zaki, J., Bolger, N., & Ochsner, K. N. (2011). Social effects of oxytocin in humans: Context and person matter. *Trends in Cognitive Sciences, 15,* 301–309.

Bateup, H. S., Booth, A., Shirtcliff, E. A., & Granger, D. A. (2002). Testosterone, cortisol, and women's competition. *Evolution and Human Behavior, 23,* 181–192.

Benarroch, E. E. (2013). Oxytocin and vasopressin: Social neuropeptides with complex neuromodulatory functions. *Neurology, 80,* 1521–1528.

Bernhardt, P. C., Dabbs, J. M., Fielden, J. A., & Lutter, C. D. (1998). Testosterone changes during vicarious experiences of winning and losing among fans at sporting events. *Physiology & Behavior, 65,* 59–62.

Bernardy, N. C., King, A. C., Parsons, O. A., & Lovallo, W. R. (1996). Altered cortisol response in sober alcoholics: An examination of contributing factors. *Alcohol (Fayetteville, N. Y.), 13,* 493–498.

Bethlehem, R. A., van Honk, J., Auyeung, B., & Baron-Cohen, S. (2013). Oxytocin, brain physiology, and functional connectivity: A review of intranasal oxytocin fMRI studies. *Psychoneuroendocrinology, 38,* 962–974.

Berthold, A. A. (1849). Transplantation of testes (trans: D.P. Quiring). *Bulletin of the History of Medicine, 1944,* 399–401.

Birch, H. G., & Clark, G. (1946). Hormonal modification of social behavior II. The effects of sex-hormone administration on the social dominance status of the female-castrate chimpanzee. *Psychosomatic Medicine, 8,* 320–331.

Boksem, M. A. S., Mehta, P. H., Van den Bergh, B., van Son, V., Trautmann, S. T., Roelofs, K, Smidts, A., & Sanfey, A. G. (2013). Testosterone inhibits trust, but promotes reciprocity. *Psychological Science, 24,* 2306–2314.

Born, J., Lange, T., Kern, W., McGregor, G. P., Bickel, U., & Fehm, H. L. (2002). Sniffing neuropeptides: A transnasal approach to the human brain. *Nature Neuroscience, 5,* 514–516.

Bos, P. A., Hermans, E. J., Ramsey, N. F., & van Honk, J. (2012). The neural mechanisms by which testosterone acts on interpersonal trust. *NeuroImage, 61,* 730–737.

Brehm, J., & Rahn, W. (1997). Individual-level evidence for the causes and consequences of social capital. *American Journal of Political Science, 41,* 999–1023.

Carney, D. R., Yap, A. J., Lucas, B. J., Mehta, P. H., McGee, J. A., & Wilmuth, C. (under review). Power buffers stress—for better and for worse. *Journal of Personality and Social Psychology*.

Carney, D. R., Cuddy, A. J., & Yap, A. J. (2010). Power posing: Brief nonverbal displays affect neuroendocrine levels and risk tolerance. *Psychological Science, 21,* 1363–1368.

Carré, J. M. (2009). No place like home: Testosterone responses to victory depend on game location. *American Journal of Human Biology, 21,* 392–394.

Carré, J. M., & Mehta, P. H. (2011). Importance of considering testosterone-cortisol interactions in predicting human aggression and dominance. *Aggressive Behavior, 37,* 1–3.

Carré, J. M., Putnam, S. K., & McCormick, C. M. (2009). Testosterone responses to competition predict future aggressive behaviour at a cost to reward in men. *Psychoneuroendocrinology, 34,* 561–570.

Carré, J. M., McCormick, C. M., & Hariri, A. R. (2011). The social neuroendocrinology of human aggression. *Psychoneuroendocrinology, 36,* 935–944.

Cattell, V. (2001). Poor people, poor places, and poor health: The mediating role of social networks and social capital. *Social Science & Medicine, 52,* 1501–1516.

Chandola, T., & Marmot, M. G. (2010). Socioeconomic status and stress. In R. Contrada & A. Baum (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 185–193). New York: Springer.

Chen, E., & Miller, G. E. (2012). "Shift-and-Persist" strategies: Why being low in socioeconomic status isn't always bad for health. *Perspectives on Psychological Science, 7,* 135–158.

Chen, F. S., Kumsta, R., von Dawans, B., Monakhov, M., Ebstein, R. P., & Heinrichs, M. (2011). Common oxytocin receptor gene (OXTR) polymorphism and social support interact to reduce stress in humans. *Proceedings of the National Academy of Sciences, 108,* 19937–19942.

Cheng, J. T., Tracy, J. L., Foulsham, T., Kingstone, A., & Henrich, J. (2013). Two ways to the top: Evidence that dominance and prestige are distinct yet viable avenues to social rank and influence. *Journal of Personality and Social Psychology, 104,* 103–125.

Chiao, J. Y., Harada, T., Oby, E. R., Li, Z., Parrish, T., & Bridge, D. J. (2009). Neural representations of social status hierarchy in human inferior parietal cortex. *Neuropsychologia, 47,* 354–363.

Churchland, P. S., & Winkielman, P. (2012). Modulating social behavior with oxytocin: How does it work? What does it mean? *Hormones and Behavior, 61,* 392–399.

Cohen, S., Doyle, W. J., & Baum, A. (2006a). Socioeconomic status is associated with stress hormones. *Psychosomatic Medicine, 68,* 414–420.

Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., & Seeman, T. (2006b). Socioeconomic status, race, and diurnal cortisol decline in the coronary artery risk development in young adults (CARDIA) study. *Psychosomatic Medicine, 68,* 41–50.

Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., & Turner, R. B. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences, 109,* 5995–5999.

Craig, M. C., Fletcher, P. C., Daly, E. M., Rymer, J., Brammer, M., Giampietro, V., & Murphy, D. G. (2008). Physiological variation in estradiol and brain function: A functional magnetic

resonance imaging study of verbal memory across the follicular phase of the menstrual cycle. *Hormones and Behavior, 53,* 503–508.

Cutler, D. M., & Lleras-Muney, A. (2008). Education and health: Evaluating theories and evidence. In R. F. Schoeni, J. S. House, G. A. Kaplan, & H. Pollack (Eds.), *Making Americans healthier: Social and economic policy as health policy* (pp. 29–60). New York: Russell Sage.

de Dreu, C. K. (2012). Oxytocin modulates cooperation within and competition between groups: An integrative review and research agenda. *Hormones and Behavior, 61,* 419–428.

de Dreu, C. K., Greer, L. L., Handgraaf, M. J., Shalvi, S., Van Kleef, G. A., Baas, M., et al. (2010). The neuropeptide oxytocin regulates parochial altruism in intergroup conflict among humans. *Science, 328,* 1408–1411.

de Dreu, C. K., Greer, L. L., Van Kleef, G. A., Shalvi, S., & Handgraaf, M. J. (2011). Oxytocin promotes human ethnocentrism. *Proceedings of the National Academy of Sciences, 108,* 1262–1266.

de Dreu, C. K., Greer, L. L., Handgraaf, M. J., Shalvi, S., & Van Kleef, G. A. (2012a). Oxytocin modulates selection of allies in intergroup conflict. *Proceedings of the Royal Society B: Biological Sciences, 279,* 1150–1154.

de Dreu, C. K., Shalvi, S., Greer, L. L., Van Kleef, G. A., & Handgraaf, M. J. (2012b). Oxytocin motivates non-cooperation in intergroup conflict to protect vulnerable in-group members. *PloS ONE, 7,* e46751.

Dedovic, K., D'Aguiar, C., & Pruessner, J. C. (2009). What stress does to your brain: A review of neuroimaging studies. *Canadian Journal of Psychiatry, 54,* 6–15.

DiBattista, J. D., Anisman, H., Whitehead, M., & Gilmour, K. M. (2005). The effects of cortisol administration on social status and brain monoaminergic activity in rainbow trout (*Oncorhynchus mykiss*). *The Journal of Experimental Biology, 208,* 2707–2718.

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355–391.

Do, D. P., Diez Roux, A. V., Hajat, A., Auchincloss, A. H., Merkin, S. S., Ranjit, N., et al. (2011). Circadian rhythm of cortisol and neighborhood characteristics in a population-based sample: The multi-ethnic study of atherosclerosis. *Health & Place, 17,* 625–632.

Dowd, J. B., Simanek, A. M., & Aiello, A. E. (2009). Socio-economic status, cortisol and allostatic load: A review of the literature. *International Journal of Epidemiology, 38,* 1297–1309.

Edwards, D. A., & Casto, K. V. (2013). Women's intercollegiate athletic competition: Cortisol, testosterone, and the dual-hormone hypothesis as it relates to status among teammates. *Hormones and Behavior, 64,* 153–160.

Eisenegger, C., et al. (2011). The role of testosterone in social interaction. *Trends in Cognitive Sciences, 15,* 263–271.

Elovainio, M., Kivimäki, M., Kortteinen, M., & Tuomikoski, H. (2001). Socioeconomic status, hostility and health. *Personality and Individual Differences, 31,* 303–315.

Evans, C. R., & Dion, K. L. (1991). Group cohesion and performance a meta-analysis. *Small Group Research, 22,* 175–186.

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development, 73,* 1238–1248.

Evans, G. W., & Kim, P. (2007). Childhood poverty and health: Cumulative risk exposure and stress dysregulation. *Psychological Science, 18,* 953–957.

Fast, N. J., Gruenfeld, D. H., Sivanathan, N., & Galinsky, A. D. (2009). Illusory control: A generative force behind power's far-reaching effects. *Psychological Science, 20,* 502–508.

Feldman, R., Weller, A., Zagoory-Sharon, O., & Levine, A. (2007). Evidence for a neuroendocrinological foundation of human affiliation: Plasma oxytocin levels across pregnancy and the postpartum period predict mother-infant bonding. *Psychological Science, 18,* 965–970.

Feldman, R., Gordon, I., Schneiderman, I., Weisman, O., & Zagoory-Sharon, O. (2010). Natural variations in maternal and paternal care are associated with systematic changes in oxytocin following parent-infant contact. *Psychoneuroendocrinology, 35,* 1133–1141.

Feldman, R., Gordon, I., & Zagoory-Sharon, O. (2011). Maternal and paternal plasma, salivary, and urinary oxytocin and parent-infant synchrony: Considering stress and affiliation co components of human bonding. *Developmental Science, 14,* 752–761.

Filby, A. L., Paull, G. C., Bartlett, E. J., Van Look, K. J., & Tyler, C. R. (2010). Physiological and health consequences of social status in zebrafish (*Danio rerio*). *Physiology & Behavior, 101,* 576–587.

Fiocco, A. J., Joober, R., & Lupien, S. J. (2007). Education modulates cortisol reactivity to the Trier Social Stress Test in middle-aged adults. *Psychoneuroendocrinology, 32,* 1158–1163.

Fischer-Shofty, M., Levkovitz, Y., & Shamay-Tsoory, S. G. (2013). Oxytocin facilitates accurate perception of competition in men and kinship in women. *Social Cognitive and Affective Neuroscience, 8,* 313–317.

Fries, E., Dettenborn, L., & Kirschbaum, C. (2009). The cortisol awakening response (CAR): Facts and future directions. *International Journal of Psychophysiology, 72,* 67–73.

Gadinger, M. C., Loerbroks, A., Schneider, S., Thayer, J. F., & Fischer, J. E. (2011). Associations between job strain and the cortisol/DHEA-S ratio among management and nonmanagement personnel. *Psychosomatic Medicine, 73,* 44–52.

Galbally, M., Lewis, A. J., IJzendoorn, M. V., & Permezel, M. (2011). The role of oxytocin in mother-infant relations: A systematic review of human studies. *Harvard Review of Psychiatry, 19,* 1–14.

Gallo, L. C., Smith, T. W., & Cox, C. M. (2006). Socioeconomic status, psychosocial processes, and perceived health: An interpersonal perspective. *Annals of Behavioral Medicine, 31,* 109–119.

Garcia, M. C., de Souza, A., Bella, G. P., Grassi-Kassisse, D. M., Tacla, A. P., & Spadari-Bratfisch, R. C. (2008). Salivary cortisol levels in Brazilian citizens of distinct socioeconomic and cultural levels. *Annals of the New York Academy of Sciences, 1148,* 504–508.

Gersten, O. (2008). Neuroendocrine biomarkers, social relations, and the cumulative costs of stress in Taiwan. *Social Science & Medicine, 66,* 507–519. (1982).

Goodman, E., McEwen, B. S., Huang, B., Dolan, L. M., & Adler, N. E. (2005). Social inequalities in biomarkers of cardiovascular risk in adolescence. *Psychosomatic Medicine, 67,* 9–15.

Greenwald, A. G., Poehlman, T. A., Uhlmann, E. L., & Banaji, M. R. (2009). Understanding and using the implicit association test: III. Meta-analysis of predictive validity. *Journal of Personality and Social Psychology, 97,* 17–41.

Grewen, K. M., Girdler, S. S., Amico, J., & Light, K. C. (2005). Effects of partner support on resting oxytocin, cortisol, norepinephrine, and blood pressure before and after warm partner contact. *Psychosomatic Medicine, 67,* 531–538.

Guastella, A. J., Mitchell, P. B., & Mathews, F. (2008). Oxytocin enhances the encoding of positive social memories in humans. *Biological Psychiatry, 64,* 256–258.

Hajat, A., Diez-Roux, A., Franklin, T. G., Seeman, T., Shrager, S., Ranjit, N., et al. (2010). Socioeconomic and race/ethnic differences in daily salivary cortisol profiles: The multi-ethnic study of atherosclerosis. *Psychoneuroendocrinology, 35,* 932–943.

Halevy, N., Chou, E. Y., & Galinsky, A. D. (2011). A functional model of hierarchy why, how, and when vertical differentiation enhances group performance. *Organizational Psychology Review, 1,* 32–52.

Halevy, N., Chou, E. Y., Cohen, T. R., & Livingston, R. W. (2012). Status conferral in intergroup social dilemmas: Behavioral antecedents and consequences of prestige and dominance. *Journal of Personality and Social Psychology, 102,* 351–366.

Hardy, C. L., & Van Vugt, M. (2006). Nice guys finish first: The competitive altruism hypothesis. *Personality and Social Psychology Bulletin, 32,* 1402–1413.

Hawkley, L. C., Lavelle, L. A., Berntson, G. G., & Cacioppo, J. T. (2011). Mediators of the relationship between socioeconomic status and allostatic load in the Chicago health, aging, and social relations study (CHASRS). *Psychophysiology, 48,* 1134–1145.

Hawkley, L. C., Cole, S. W., Capitanio, J. P., Norman, G. J., & Cacioppo, J. T. (2012). Effects of social isolation on glucocorticoid regulation in social mammals. *Hormones and Behavior, 62,* 314–323.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54,* 1389–1398.

Hermans, E. J., Ramsey, N. F., & van Honk, J. (2008). Exogenous testosterone enhances responsiveness to social threat in the neural circuitry of social aggression in humans. *Biological Psychiatry, 63,* 263–270.

Höfer, P., Lanzenberger, R., & Kasper, S. (2013). Testosterone in the brain: Neuroimaging findings and the potential role for neuropsychopharmacology. *European Neuropsychopharmacology, 23,* 79–88.

Hull, J. G., & Slone, L. B. (2004). Alcohol and self-regulation. In K. D. Vohs & R. F. Baumeister (Eds.), *Handbook of self-regulation: Research, theory, and applications* (pp. 466–491). New York: Guilford.

Jayo, J. M., Shively, C. A., Kaplan, J. R., & Manuck, S. B. (1993). Effects of exercise and stress on body fat distribution in male *cynomolgus* monkeys. *International Journal of Obesity and Related Metabolic Disorders, 17,* 597–604.

Jiménez, M., Aguilar, R., & Alvero-Cruz, J. R. (2012). Effects of victory and defeat on testosterone and cortisol response to competition: Evidence for same response patterns in men and women. *Psychoneuroendocrinology, 37,* 1577–1581.

Jones, A. C., & Josephs, R. A. (2006). Interspecies hormonal interactions between man and the domestic dog (*Canis familiaris*). *Hormones and Behavior, 50,* 393–400.

Jordan, J., Sivanathan, N., & Galinsky, A. D. (2011). Something to lose and nothing to gain: The role of stress in the interactive effect of power and stability on risk taking. *Administrative Science Quarterly, 56,* 530–558.

Josephs, R. A., Newman, M. L., Brown, R. P., & Beer, J. M. (2003). Status, testosterone, and human intellectual performance: Stereotype threat as status concern. *Psychological Science, 14,* 158–163.

Josephs, R. A., Sellers, J. G., Newman, M. L., & Mehta, P. H. (2006). The mismatch effect: When testosterone and status are at odds. *Journal of Personality and Social Psychology, 90,* 999–1013.

Josephs, R. A., Telch, M. J., Hixon, J. G., Evans, J. J., Lee, H., Knopik, V. S., McGeary, J. E., et al. (2012). Genetic and hormonal sensitivity to threat: Testing a serotonin transporter genotype x testosterone interaction. *Psychoneuroendocrinology, 37,* 752–761.

Kagerbauer, S. M., Martin, J., Schuster, T., Blobner, M., Kochs, E. F., & Landgraf, R. (2013). Plasma oxytocin and vasopressin do not predict neuropeptide concentrations in the human cerebrospinal fluid. *Journal of Neuroendocrinology*, *25,* 668–673.

Kapuku, G. K., Treiber, F. A., & Davis, H. C. (2002). Relationships among socioeconomic status, stress induced changes in cortisol, and blood pressure in African American males. *Annals of Behavioral Medicine, 24,* 320–325.

Koolhaas, J. M., Van den Brink, T. H. C., Roozendaal, B., & Boorsma, F. (1990). Medial amygdala and aggressive behavior: Interaction between testosterone and vasopressin. *Aggressive Behavior, 16,* 223–229.

Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. (2005). Oxytocin increases trust in humans. *Nature, 435,* 673–676.

Kristenson, M., Kucinskienë, Z., Bergdahl, B., Calkauskas, H., Urmonas, V., & Orth-Gomér, K. (1998). Increased psychosocial strain in Lithuanian versus Swedish men: The LiVicordia study. *Psychosomatic Medicine, 60,* 277–282.

Krueger, P. M., & Chang, V. W. (2008). Being poor and coping with stress: Health behaviors and the risk of death. *American Journal of Public Health, 98,* 889–896.

Kubzansky, L. D., Kawachi, I., & Sparrow, D. (1999). Socioeconomic status, hostility, and risk factor clustering in the normative aging study: Any help from the concept of allostatic load? *Annals of Behavioral Medicine, 21,* 330–338.

Kumari, M., Badrick, E., Chandola, T., Adler, N. E., Epel, E., Seeman, T., et al. (2010). Measures of social position and cortisol secretion in an aging population: Findings from the Whitehall II study. *Psychosomatic Medicine, 72,* 27–34.

Kumari, M., Shipley, M., Stafford, M., & Kivimaki, M. (2011). Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: Findings from the Whitehall II study. *Journal of Clinical Endocrinology & Metabolism, 96,* 1478–1485.

Li, L., Power, C., Kelly, S., Kirschbaum, C., & Hertzman, C. (2007). Life-time socio-economic position and cortisol patterns in mid-life. *Psychoneuroendocrinology, 32,* 824–833.

Lincoln, G. A., Guinness, F., & Short, R. V. (1972). The way in which testosterone controls the social and sexual behavior of the red deer stag (*Cervus elaphus*). *Hormones and Behavior, 3,* 375–396.

Lindfors, P., & Lundberg, U. (2002). Is low cortisol release an indicator of positive health? *Stress and Health, 18,* 153–160.

Lovallo, W. R., Robinson, J. L., Glahn, D. C., & Fox, P. T. (2010). Acute effects of hydrocortisone on the human brain: An fMRI study. *Psychoneuroendocrinology, 35,* 15–20.

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. *Biological Psychiatry, 48,* 976–980.

Maki, P. M., & Resnick, S. M. (2001). Effects of estrogen on patterns of brain activity at rest and during cognitive activity: A review of neuroimaging studies. *NeuroImage, 14,* 789–801.

Maner, J. K., Miller, S. L., Schmidt, N. B., & Eckel, L. A. (2008). Submitting to defeat: Social anxiety, dominance threat, and decrements in testosterone. *Psychological Science, 19,* 764–768.

Marsh, A. A., Henry, H. Y., Pine, D. S., & Blair, R. J. R. (2010). Oxytocin improves specific recognition of positive facial expressions. *Psychopharmacology, 209,* 225–232.

Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences, 21,* 353–397.

McCarthy, M. M. (2008). Estradiol and the developing brain. *Physiological Reviews, 88,* 91–134.

Mehta, P. H., & Beer, J. S. (2010). Neural mechanisms of the testosterone-aggression relation: The role of orbitofrontal cortex. *Journal of Cognitive Neuroscience, 22,* 2357–2368.

Mehta, P. H., & Josephs, R. A. (2006). Testosterone change after losing predicts the decision to compete again. *Hormones and Behavior, 50,* 684–692.

Mehta, P. H., & Josephs, R. A. (2010). Testosterone and cortisol jointly regulate dominance: Evidence for a dual-hormone hypothesis. *Hormones and Behavior, 58,* 898–906.

Mehta, P. H., & Josephs, R. A. (2011). Social endocrinology: Hormones and social motivation. In D. Dunning (Ed.), *Frontiers of social psychology: Social motivation* (pp. 171–190). New York: Psychology Press.

Mehta, P. H., Jones, A. C., & Josephs, R. A. (2008). The social endocrinology of dominance: Basal testosterone predicts cortisol changes and behavior following victory and defeat. *Journal of Personality and Social Psychology, 94,* 1078–1093.

Mehta, P. H., Wuehrmann, E. V., & Josephs, R. A. (2009). When are low testosterone levels advantageous? The moderating role of individual versus intergroup competition. *Hormones and Behavior, 56,* 158–162.

Mehta, P. H., Lawless, N., & Carney, D. (2013). *Dual-hormone profiles of leadership*. Manuscript in preparation, University of Oregon.

Mendl, M., Zanella, A. J., & Broom, D. M. (1992). Physiological and reproductive correlates of behavioural strategies in female domestic pigs. *Animal Behaviour, 44,* 1107–1121.

Michopoulos, V., Checchi, M., Sharpe, D., & Wilson, M. E. (2011). Estradiol effects on behavior and serum oxytocin are modified by social status and polymorphisms in the serotonin transporter gene in female rhesus monkeys. *Hormones and Behavior, 59,* 528–535.

Michopoulos, V., Reding, K. M., Wilson, M. E., & Toufexis, D. (2012). Social subordination impairs hypothalamic-pituitary-adrenal function in female rhesus monkeys. *Hormones and Behavior, 62,* 389–399.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133,* 25–45.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin, 126,* 247–259.

Newman, M. L., Sellers, J. G., & Josephs, R. A. (2005). Testosterone, cognition, and social status. *Hormones and Behavior, 47,* 205–211.

Norman, G. J., Hawkley, L., Luhmann, M., Ball, A. B., Cole, S. W., Berntson, G. G., & Cacioppo, J. T. (2012). Variation in the oxytocin receptor gene influences neurocardiac reactivity to social stress and HPA function: A population based study. *Hormones and Behavior, 61,* 134–139.

Oliveira, R. F., Almada, V. C., & Canario, A. V. (1996). Social modulation of sex steroid concentrations in the urine of male cichlid fish *Oreochromis mossambicus*. *Hormones and Behavior, 30,* 2–12.

Oliveira, T., Gouveia, M. J., & Oliveira, R. F. (2009). Testosterone responsiveness to winning and losing experiences in female soccer players. *Psychoneuroendocrinology, 34,* 1056–1064.

Pampel, F. C., Krueger, P. M., & Denney, J. T. (2010). Socioeconomic disparities in health behaviors. *Annual Review of Sociology, 36,* 349–370.

Payne, A. H., & Hales, D. B. (2004). Overview of steroidogenic enzymes in the pathway from cholesterol to active steroid hormones. *Endocrine Reviews, 25,* 947–970.

Pérez-López, F. R., Larrad-Mur, L., Kallen, A., Chedraui, P., & Taylor, H. S. (2010). Review: Gender differences in cardiovascular disease: Hormonal and biochemical influences. *Reproductive Sciences, 17,* 511–531.

Phillips, D. I., Walker, B. R., Reynolds, R. M., Flanagan, D. E., Wood, P. J., Osmond, C., et al. (2000). Low birth weight predicts elevated plasma cortisol concentrations in adults from 3 populations. *Hypertension, 35,* 1301–1306.

Pinquart, M., & Sörensen, S. (2000). Influences of socioeconomic status, social network, and competence on subjective well-being in later life: A meta-analysis. *Psychology and Aging, 15,* 187–224.

Plant, T. M. (2012). A comparison of the neuroendocrine mechanisms underlying the initiation of the preovulatory LH surge in the human, old world monkey and rodent. *Frontiers in Neuroendocrinology, 33,* 160–168.

Pope, M. K., & Smith, T. W. (1991). Cortisol excretion in high and low cynically hostile men. *Psychosomatic Medicine, 53,* 386–392.

Popma, A., Vermeiren, R., Geluk, C. A., et al. (2007). Cortisol moderates the relationship between testosterone and aggression in delinquent male adolescents. *Biological Psychiatry, 61,* 405–411.

Ranjit, N., Young, E. A., & Kaplan, G. A. (2005). Material hardship alters the diurnal rhythm of salivary cortisol. *International Journal of Epidemiology, 34,* 1138–1143.

Ranjit, N., Diez-Roux, A. V., Sanchez, B., Seeman, T., Shea, S., Shrager, S., & Watson, K. (2009). Association of salivary cortisol circadian pattern with cynical hostility: Multi-ethnic study of atherosclerosis. *Psychosomatic Medicine, 71,* 748–755.

Rimmele, U., Hediger, K., Heinrichs, M., & Klaver, P. (2009). Oxytocin makes a face in memory familiar. *The Journal of Neuroscience, 29,* 38–42.

Roelofs, K., Elzinga, B. M., & Rotteveel, M. (2005). The effects of stress-induced cortisol responses on approach-avoidance behavior. *Psychoneuroendocrinology, 30,* 665–677.

Rogol, A. D., Roemmich, J. N., & Clark, P. A. (2002). Growth at puberty. *Journal of Adolescent Health, 31,* 192–200.

Ronay, R., Greenaway, K., Anicich, E. M., & Galinsky, A. D. (2012). The path to glory is paved with hierarchy: When hierarchical differentiation increases group effectiveness. *Psychological Science, 23,* 669–677.

Rosmond, R., & Björntorp, P. (2000a). The hypothalamic-pituitary-adrenal axis activity as a predictor of cardiovascular disease, type 2 diabetes and stroke. *Journal of Internal Medicine, 247,* 188–197.

Rosmond, R., & Björntorp, P. (2000b). Occupational status, cortisol secretory pattern, and visceral obesity in middle-aged men. *Obesity Research, 8,* 445–450.

Ross, H. E., & Young, L. J. (2009). Oxytocin and the neural mechanisms regulating social cognition and affiliative behavior. *Frontiers in Neuroendocrinology, 30,* 534–547.

Ruiz-de-la-Torre, J. L., & Manteca, X. (1999). Effects of testosterone on aggressive behaviour after social mixing in male lambs. *Physiology and Behavior, 68,* 109–113.

Ryff, C. D., Singer, B. H., & Love, G. D. (2004). Positive health: Connecting well-being with biology. *Philosophical Transactions-Royal Society of London Series B Biological Sciences, 359,* 1383–1394.

Salonia, A., Nappi, R. E., Pontillo, M., Daverio, R., Smeraldi, A., Briganti, A., et al. (2005). Menstrual cycle-related changes in plasma oxytocin are relevant to normal sexual function in healthy women. *Hormones and behavior, 47,* 164–169.

Salvador, A., & Costa, R. (2009). Coping with competition: Neuroendocrine responses and cognitive variables. *Neuroscience and Biobehavioral Reviews, 33,* 160–170.

Sapolsky, R. M. (1991). Testicular function, social rank and personality among wild baboons. *Psychoneuroendocrinology, 16,* 281–293.

Sapolsky, R. M. (1996). Why stress is bad for your brain. *Science, 273,* 749–750.

Sapolsky, R. M. (1999). Glucocorticoids, stress, and their adverse neurological effects: Relevance to aging. *Experimental Gerontology, 34*, 721–732.

Sapolsky, R. M. (2004). Social status and health in humans and other animals. *Annual Review of Anthropology, 33,* 393–418.

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science, 308,* 648–652.

Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1985). Prolonged glucocorticoid exposure reduces hippocampal neuron number: Implications for aging. *The Journal of Neuroscience, 5,* 1222–1227.

Schultheiss, O. C. (2007). A biobehavioral model of implicit power motivation: Arousal, reward, and frustration. In E. Harmon-Jones & P. Winkielman (Eds.), *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 176–196). New York: Guilford.

Schultheiss, O. C., Dargel, A., & Rohde, W. (2003). Implicit motives and gonadal steroid hormones: Effects of menstrual cycle phase, oral contraceptive use, and relationship status. *Hormones and Behavior, 43,* 293–301.

Schultheiss, O. C., Wirth, M. M., Torges, C. M., Pang, J. S., Villacorta, M. A., & Welsh, K. M. (2005). Effects of implicit power motivation on men's and women's implicit learning and testosterone changes after social victory or defeat. *Journal of Personality and Social Psychology, 88,* 174–188.

Sephton, S. E., Sapolsky, R. M., Kraemer, H. C., & Spiegel, D. (2000). Diurnal cortisol rhythm as a predictor of breast cancer survival. *Journal of the National Cancer Institute, 92,* 994–1000.

Severance, L. (2011). A biosocial approach to negotiation (Doctoral dissertation).

Shalev, I., Israel, S., Uzefovsky, F., Gritsenko, I., Kaitz, M., & Ebstein, R. P. (2011). Vasopressin needs an audience: Neuropeptide elicited stress responses are contingent upon perceived social evaluative threats. *Hormones and Behavior, 60,* 121–127.

Sherman, G. D., Lee, J. J., Cuddy, A. J., Renshon, J., Oveis, C., Gross, J. J., & Lerner, J. S. (2012). Leadership is associated with lower levels of stress. *Proceedings of the National Academy of Sciences, 109,* 17903–17907.

Simmons, Z. L., & Roney, J. R. (2011). Variation in CAG repeat length of the androgen receptor gene predicts variables associated with intrasexual competitiveness in human males. *Hormones and Behavior, 60,* 306–312.

Soma, K. K. (2006). Testosterone and aggression: Berthold, birds and beyond. *Journal of Neuroendocrinology, 18,* 543–551.

Smith, T. W. (1994). Concepts and methods in the study of anger, hostility, and health. In W. Siegman & T. W. Smith (Eds.), *Anger, hostility, and the heart* (pp. 23–42). Hillsdale: Erlbaum.

Stallen, M., De Dreu, C. K., Shalvi, S., Smidts, A., & Sanfey, A. G. (2012). The herding hormone: Oxytocin stimulates in-group conformity. *Psychological Science, 23,* 1288–1292.

Stanton, S. J., & Edelstein, R. S. (2009). The physiology of women's power motive: Implicit power motivation is positively associated with estradiol levels in women. *Journal of Research in Personality, 43,* 1109–1113.

Stanton, S. J., & Schultheiss, O. C. (2007). Basal and dynamic relationships between implicit power motivation and estradiol in women. *Hormones and Behavior, 52,* 571–580.

Stanton, S. J., & Schultheiss, O. C. (2009). The hor.monal correlates of implicit power motivation. *Journal of Research in Personality, 43,* 942–949.

Suay, F., Salvador, A., González-Bono, E., Sanchís, C., Martínez, M., Martínez-Sanchis, S., et al. (1999). Effects of competition and its outcome on serum testosterone, cortisol and prolactin. *Psychoneuroendocrinology, 24,* 551–566.

Stanton, S. J., Beehner, J. C., Saini, E. K., Kuhn, C. M., & LaBar, K. S. (2009). Dominance, politics, and physiology: Voters' testosterone changes on the night of the 2008 United States Presidential Election. *Plos ONE, 4,* e7543.

Stanton, S. J., LaBar, K. S., Saini, E. K., Kuhn, C. M., & Beehner, J. C. (2010). Stressful politics: Voters' cortisol responses to the outcome of the 2008 United States Presidential election. *Psychoneuroendocrinology, 35,* 768–774.

Steptoe, A., Kunz-Ebrecht, S., Owen, N., Feldman, P. J., Willemsen, G., Kirschbaum, C., & Marmot, M. (2003). Socioeconomic status and stress-related biological responses over the working day. *Psychosomatic Medicine, 65,* 461–470.

Subramanian, S. V., Lochner, K. A., & Kawachi, I. (2003). Neighborhood differences in social capital: A compositional artifact or a contextual construct? *Health & Place, 9,* 33–44.

Tajfel, H. (1982). Social psychology of intergroup relations. *Annual Review of Psychology, 33,* 1–39.

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. A., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107,* 411–429.

Taylor, S. E., Gonzaga, G. C., Klein, L. C., Hu, P., Greendale, G. A., & Seeman, T. E. (2006). Relation of oxytocin to psychological stress responses and hypothalamic-pituitary-adrenocortical axis activity in older women. *Psychosomatic Medicine, 68,* 238–245.

Terburg, D., Aarts, H., & van Honk, J. (2012). Testosterone affects gaze aversion from angry faces outside of conscious awareness. *Psychological Science, 23,* 459–463.

Thayer, J. F., Hall, M., Sollers, J. J. III, & Fischer, J. E. (2006). Alcohol use, urinary cortisol, and heart rate variability in apparently healthy men: Evidence for impaired inhibitory control of the HPA axis in heavy drinkers. *International Journal of Psychophysiology, 59,* 244–250.

Theodoridou, A., Rowe, A. C., Penton-Voak, I. S., & Rogers, P. J. (2009). Oxytocin and social perception: Oxytocin increases perceived facial trustworthiness and attractiveness. *Hormones and Behavior, 56,* 128–132.

Timmer, M., & Sandi, C. (2010). A role for glucocorticoids in the long-term establishment of a social hierarchy. *Psychoneuroendocrinology, 35,* 1543–1552.

Tops, M., van Peer, J. M., Korf, J., Wijers, A. A., & Tucker, D. M. (2007). Anxiety, cortisol, and attachment predict plasma oxytocin. *Psychophysiology, 44,* 444–449.

Turner, R. A., Altemus, M., Enos, T., Cooper, B., & McGuinness, T. (1999). Preliminary research on plasma oxytocin in normal cycling women: Investigating emotion and interpersonal distress. *Psychiatry, 62,* 97–113.

Uchino, B. N. (2006). Social support and health: A review of physiological processes potentially underlying links to disease outcomes. *Journal of Behavioral Medicine, 29,* 377–387.

van Anders, S. M., & Watson, N. V. (2006). Social neuroendocrinology—effects of social contexts and behaviors on sex steroids in humans. *Human Nature—An Interdisciplinary Biosocial Perspective, 17,* 212–237.

van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., et al. (1999). Correlations among salivary testosterone, mood, and selective attention to threat in humans. *Hormones and Behavior, 36,* 17–24.

van Honk, J., Peper, J., & Schutter, D. (2005). Testosterone reduces unconscious fear but not consciously experienced anxiety: Implications for the disorders of fear and anxiety. *Biological Psychiatry, 58,* 218–225.

van Peer, J. M., Roelofs, K., Rotteveel, M., van Dijk, J. G., Spinhoven, P., & Ridderinkhof, K. R. (2007). The effects of cortisol administration on approach-avoidance behavior: An event-related potential study. *Biological Psychology, 76,* 135–146.

Willer, R. (2009). Groups reward individual sacrifice: The status solution to the collective action problem. *American Sociological Review, 74,* 23–43.

Wingfield, J. C., Hegner, R. E., Dufty, A. M. Jr., & Ball, G. F. (1990). The "challenge hypothesis": Theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. *American Naturalist, 136,* 829–846.

Wirth, M. M., & Schultheiss, O. C. (2006). Effects of affiliation arousal (hope of closeness) and affiliation stress (fear of rejection) on progesterone and cortisol. *Hormones and Behavior, 50,* 786–795.

Wirth, M. M., & Schultheiss, O. C. (2007). Basal testosterone moderates responses to anger faces in humans. *Physiology and Behavior, 90,* 496–505.

Wirth, M. M., Welsh, K. M., & Schultheiss, O. C. (2006). Salivary cortisol changes in humans after winning or losing a dominance contest depend on implicit power motivation. *Hormones and Behavior, 49,* 346–352.

Wu, M. V., & Shah, N. M. (2011). Control of masculinizationof the brain and behavior. *Current Opinion in Neurobiology, 21,* 116–123.

Zak, P. J., Kurzban, R., & Matzner, W. T. (2005). Oxytocin is associated with human trustworthiness. *Hormones and Behavior, 48,* 522–527.

Zak, P. J., Stanton, A. A., & Ahmadi, S. (2007). Oxytocin increases generosity in humans. *PLoS ONE, 2,* e1128.

Zink, C. F., Tong, Y., Chen, Q., Bassett, D. S., Stein, J. L., & Meyer-Lindenberg, A. (2008). Know your place: Neural processing of social hierarchy in humans. *Neuron, 58,* 273–283.

Zyphur, M. J., Narayanan, J., Koh, G., & Koh, D. (2009). Testosterone-status mismatch lowers collective efficacy in groups: Evidence from a slope-as-predictor multilevel structural equation model. *Organizational Behavior and Human Decision Processes, 110,* 70–79.