Stress Hormones, Physiology, and Behavior

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Abstract and Keywords

Stress, be it physical or psychological, can have a devastating long-term impact on an individual's development, health, and well-being, and yet can be adaptive in the short term (e.g., promoting immediate survival, triggering the desire to remedy social conflict). The stress response system involves physiological processes in reaction to a real or perceived threat, which serve a variety of purposes. This chapter reviews pertinent topics and research within the social neuroendocrine study of stress, including acute versus chronic stress, and how stress influences social behavior and status. Where appropriate, it offers critiques of current theoretical models and includes suggestions for future directions within this research area.

Keywords: stress, hormones, glucocorticoids, cortisol, health, behavior

Stress is any real or interpreted threat to the physiological or psychological integrity of an individual that results in physiological and/or behavioral responses (McEwen, 2000). It occurs in reaction to somatic challenges such as competition, environmental harshness, and disease, and is mediated by catecholamines (e.g., epinephrine and norepinephrine) and glucocorticoids (GCs; e.g., cortisol in humans and fish; corticosterone in amphibians, reptiles, and birds). GCs are a class of corticosteroids, which are steroid hormones that are released by the adrenal cortex as part of a physiological feedback system known as the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis regulates so-called stress hormones through positive and negative feedback systems initiated by neural input to the hypothalamus. Initial hypothalamic release of corticotropin-releasing hormone (CRH) to the anterior pituitary causes release of adrenocorticotropic hormone (ACTH) to the adrenal cortex. Next, GCs produced by the adrenal cortex enter the bloodstream and promote a number of physiological changes (e.g., increased respiration, blood oxygenation, glucose release), which divert energy from nonessential processes, such as

reproduction and food digestion, to those that promote immediate survival (e.g., muscle innervation, threat processing).

Although the stress response can be adaptive in the short term, it can have maladaptive consequences for long-term functioning and health. In this chapter, we review several of the most well-researched topics within the social neuroendocrine study of stress, provide an overview and critical analysis of current theoretical models (where appropriate), and provide a summary of potential future directions within the current literature.

Acute Stress Response

The stress response is vital to an organism's survival and has coevolved with a number of other physiological systems (e.g., dopaminergic reward systems, attachment and social buffering systems) to permit dynamic redistribution of somatic resources when the demands of internal or external events exceed immediately available resources (see, e.g., Lazarus & Folkman, 1984). Acute stress can trigger survival-related and other adaptive behaviors, such as increases in attention and memory. For example, amygdala arousal from watching emotional films enhances episodic memory for these films compared to neutral controls (Cahill et al., 1996). Acute stress also reduces attention toward irrelevant information during a Stroop task (Booth & Sharma, 2009), suggesting that acute stress redirects attention toward temporally imperative information, but reduces more flexible attentional processing (Gagnon & Wagner, 2016). Similarly, other work has shown that acute stress impairs recall of complex informational arrays and hinders performance during delayed recall and memory tasks (Olver, Pinney, Maruff, & Norman, 2015). Together, this evidence suggests that acute stress functionally redirects cognitive effort toward immediate threats to enhance immediate survival at the cost of dampening long-term neural plasticity (Farmer, Park, Bullard, & Diamond, (p. 352) 2014). However, acute stress can also act as a prompt to compel an organism to act in several ways, depending on various personal and contextual factors. Research has largely focused on two specific threat responses: the *fight-or-flight* and the *tend-and-befriend* threat responses.

Fight-or-Flight Versus Tend-and-Befriend

Throughout evolutionary history, humans and other organisms have recurrently encountered situations that required split-second decision making (e.g., an approaching predator, an aggressive competitor). Stressful situations present an energy allocation problem: one may either divert resources to (1) confronting the threat (if there is a high probability of overpowering the threat) or (2) fleeing from it (when the threat is likely to win). This process, known as the *fight-or-flight* response, innervates the body to respond to immediate stress and is thus adaptive in situations of acute stress (Cannon, 1929).

Upon recognition of a stressor, the sympathetic nervous system activates the fight-or-flight response (i.e., the sympathetic adrenal medullary [SAM] response) to begin redirecting energy. First, the dorsomedial amygdalar complex recognizes that a potential threat is near and sends neural impulses to the lateral and posterior hypothalamic

regions (Roldan, Alvarez-Pelaez, & de Molina, 1974). The hypothalamus signals to the adrenergic neurons of the thoracic spinal cord to secrete norepinephrine, and to the chromaffin cells of the adrenal medullae to produce catecholamines in a process known as catecholaminogenesis (Everly & Lating, 2013). The adrenal medullary catecholamines epinephrine and norepinephrine increase respiration, heart rate, and blood pressure, and improve blood flow to muscles (Everly & Lating, 2013). Likewise, they increase blood glucose levels and blood oxygenation and improve alertness, learning, and memory (Usdin, Kvetnansky, & Kopin, 1976).

During this process, the adaptive fight-or-flight response halts biological functions that are unnecessary for immediate survival, including reproductive efforts, bladder muscle innervation, digestion, and blood flow to the skin and kidneys (Everly & Lating, 2013). Conversely, fight-or-flight increases heart rate to improve circulation, rate and depth of breathing to improve the exchange of gases, synthesis of glucose to provide energy, blood perfusion of muscles to increase strength and endurance, and blood clot reduction to minimize tissue damage (Nesse, Bhatnagar, & Ellis, 2016). Although it was originally believed that the fight-or-flight response decreased the function of the immune system to conserve energy (similar to its suppression of digestion and ovulation at times of stress), there is expanding evidence that immune responses actually increase in response to specific types of stressors (e.g., Dhabhar, 2002). Because some types of stress can lead to infection (e.g., an injury), immune system enhancement would be most beneficial for survival after specific types of acute stress such as an infection or injury (e.g., Dhabhar & McEwen, 2001). However, long-term exposure to this process may also intensify autoimmune and inflammatory diseases (Dhabhar, 2002), and thus, there likely exists an optimal balance that encourages the immune-enhancing or -suppressive responses depending on the situation. Indeed, during acute stressors like injury, glucocorticoids can enhance immune functioning, purportedly to prepare the immune system for potential infection (e.g., Dhabhar, Miller, Stein, McEwen, & Spencer, 1994). However, upon exposure to acute stressors such as a final examination (Glaser, Kiecolt-Glaser, Speicher, & Holliday, 1985) or life change (Cohen-Cole, Cogen, & Stevens, 1981), the immune system suppresses its response to conserve the energy needed to navigate the immediate stressor (McEwen et al., 1997). Overall, rather than acting specifically as an immune suppressant (e.g., Auphan, DiDonato, Rosette, Helmberg, & Karin, 1995), it appears glucocorticoids modulate their function to enhance or suppress the immune system response depending on the type of stress experienced (McEwen et al., 1997).

(p. 353) Similar to fight-or-flight, the *tend-and-befriend* threat response is a hormone-driven behavioral reaction to stress that typically occurs more often in females (Taylor et al., 2000). Via the release of oxytocin (OT), this response manifests as an increased motivation to protect offspring (tend) and seek out social group members for mutual defense and aid (befriend). Testosterone and arginine vasopressin (AVP) released during the fight-or-flight stress response have been found to exhibit the opposite effects of OT (see Taylor, 2006; Taylor et al., 2000, 2006). The original model developed by Taylor et al. (2000) describes tend-and-befriend as a female counterpart to the fight-or-flight response in males (Cannon, 1929). These authors highlight that, before 1995, research

investigating the fight-or-flight response had been conducted predominantly on male participants, with females constituting a mere 17 percent of the participants, which may have led researchers to overlook the more female-typical tend-and-befriend response.

In response to threat, both males and females show increased activation of the autonomic nervous system, which causes the release of OT, AVP, and CRH. However, OT is released in greater quantities in females (Taylor et al., 2000). OT is believed by some to encourage affiliative behavior, including maternal nurturance and seeking social contact from peers (e.g., Insel, 1997; Carter, 1998; see also Grebe & Gangestad, this volume), and may actively alleviate the biological stress responses by, for example, decreasing heart rate, blood pressure, HPA activity (Light et al., 2000), and cortisol level (Uvnas-Moberg, 1997, 1998). In addition, estrogen enhances the effects of OT, whereas androgens inhibit OT release (McCarthy, 1994), which may contribute to why women are more likely to respond to stress via tend-and-befriend than men. Certainly, women who report more deficiencies in their social relationships (i.e., reduced contact with various social support sources) and marital dissatisfaction have elevated OT levels (Taylor et al., 2006).

Overall, women consistently show a stronger affiliative response to stress than men do (Tamres, Janicki, & Helgeson, 2002; Taylor et al., 2000), and therefore a social-support-seeking response to stress may be particularly adaptive for females. Both men and women rely on group living for successful defense against predators and outgroup members, but human females also face greater threats from in-group human males (e.g., rape, assault, abuse of offspring). Furthermore, women often take on more parental responsibility for early offspring care, and pregnancy and nursing make women especially vulnerable to external threats (see Sear & Mace, 2008). Forming a network not only provides protection and help with raising offspring, but also serves to secure resources, such as housing and food. Given that a group is more likely than an individual to overcome or deter a threat, a social-support-seeking response is likely a protective mechanism for both the woman and her offspring. Correspondingly, evidence suggests that women, more than men, are geared for fostering and maintaining social relationships. For example, although the need for interpersonal connection seems to be a near-universal human trait, women tend to socialize more in new environments (Wheeler & Nezlek, 1977) and are more focused on maintaining belongingness than men (Baumeister & Leary, 1995). Even as children, girls have a stronger interest in maintaining meaningful and nurturing relationships, resulting in a higher number of relationships than male counterparts (Galambos, 2004; Nichols & Good, 1998). Also, women tend to score higher on measures of emotional intelligence and social skills than men (Bindu & Thomas, 2006; Petrides & Furnham, 2000), suggesting that women have specialized cognitive mechanisms for maintaining affiliation. Together, this research suggests that women compared to men are more likely to overtly prioritize social support responses to stress over fight-or-flight responses (e.g., Turton & Campbell, 2005) because evolutionary pressures favored an affiliative response.

Although, as noted, OT is associated with seeking social support in response to stress, which can in turn alleviate stress, it is important to emphasize that the social environment itself can also be the source of stress. For example, cortisol has been shown to increase after social rejection in both men and women (Blackhart, Eckel, & Tice, 2007), although women show elevated cortisol responses to social rejection versus achievement failure manipulations compared to men (Stroud, Salovey, & Epel, 2002), suggesting they may be more physiologically reactive to social rejection than men. The relationship between OT and cortisol led Taylor (2006) to propose a potential model for a stress affiliation, which is dependent on the success or failure of gaining social support via the tend-and-befriend stress response (see Figure 20.1 for a simplified iteration of Taylor's [2006] conceptual model). Although the buffering influence of OT on stress is discussed in more detail later (see Social Buffering), the model (p. 354) proposes that stress triggers increased affiliative efforts via increased OT in line with the tend-and-befriend stress response system. The subsequent effect on stress is then dependent on the outcome of this effort; negative outcomes (e.g., failure to gain social support) will increase stress, whereas positive outcomes (e.g., success at gaining social support) will decrease stress. Taylor provides a selection of available evidence for each component of the model in support, although much of this evidence is derived from animal studies. However, recently a body of evidence in humans has begun to emerge, further supporting the theory (e.g., Cardoso, Ellenbogen, Serravalle, & Linnen, 2013; von Dawans, Fischbacher, Kirschbaum, Fehr, & Heinrichs, 2012).

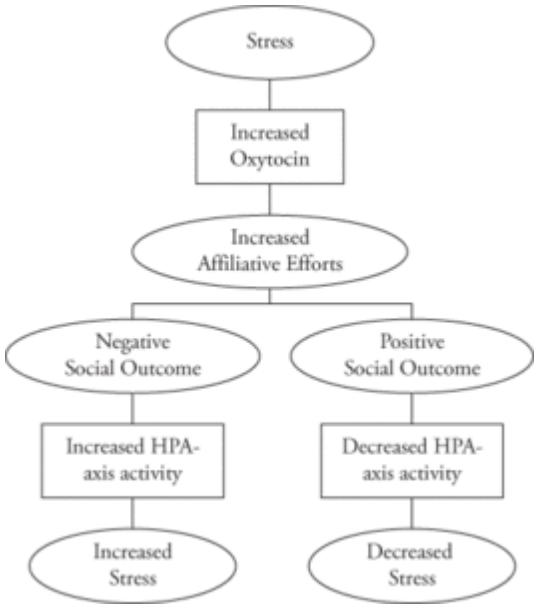

Figure 20.1 A theoretical cascade model of an affiliative responses to stress, extrapolated from Taylor (2006). In this model, stress increases oxytocin (OT), which leads to increased affiliative efforts. When these efforts yield negative social outcomes, increased hypothalamic-pituitary-adrenal (HPA) axis activity leads to increased stress. When affiliative efforts yield positive social outcomes, HPA activity and, by extension, stress decrease.

Hence, acute stress likely evolved to promote adaptive outcomes under difficult circumstances. Whereas the *fight-or-flight* response readies the body for action to escape or combat a threat, the *tend-and-befriend* response encourages the individual to seek social support to combat threat and provide additional attention to vulnerable dependents (e.g., children, kin), thus shielding their genetic (or social) assets from the threat. Either response highlights that, despite its unpleasant sensation, acute stress is typically an adaptive cognitive and

hormonal mechanism designed to promote surviving and thriving.

Chronic Stress

In contrast to acute stress, chronic stress is maladaptive. One of the first studies on chronic stress (Selye, 1956) found that repeated shocks produced stomach ulcers and a lowered immune system response in rats. Prolonged exposure to GCs lowers immune response by suppressing cytokines, blocking cytokine receptors, disrupting lymphocyte development, and destroying lymphocytes (for review, see Segerstrom & Miller, 2004). As such, individuals who experience chronic stress are more likely to get the common cold and have more frequent cold sore flare-ups (Cohen, Tyrrell, & Smith, 1991, 1993). Additionally, chronic stress has been associated with an increased likelihood of getting a respiratory infection, the acceleration of autoimmune disorders, and increased recurrence of chronic allergies (Boyce et al., 1977, 1993; Monroe & Hadjiyannakis, 2002; Pereira & Penedo, 2005).

Since Selye's (1956) study, considerable research has investigated and confirmed the maladaptive effects of chronic stress (e.g., Coe & Lubach, 2003; Repetti, Taylor, & Seeman, 2002; Stowell, Kiecolt-Glaser, & Glaser, 2001; Taylor, Repetti, & Seeman, 1997). The stress response is a trade-off between long-term and short-term functioning. Processes essential for long-term survival (e.g., the immune system) are suspended to increase the likelihood of immediate survival. Because the stress response suppresses functions that are not vital for immediate survival, chronic stress can result in an increased susceptibility to infectious disease, psychological deficits, growth reduction, and reproductive issues (Ader, Felton, & Cohen, 1991; Glaser & Kiecolt-Glaser, 2014). Chronic stress continually mobilizes energy at the cost of energy storage, and the result is fatigue, muscle loss, and weakness (Bower et al., 2005, 2007). Additionally, the prolonged increase in heart rate weakens the heart muscles over time and increases plaque buildup (Booth-Kewley & Friedman, 1987; Rozanski, Blumenthal, & Kaplan, 1999). An increase in GCs is also associated with greater appetite and blockage of glucose reuptake, which can result in increased fat depositions, particularly in the midsection (Brindley & Rolland, 1989). As such, chronic stress is associated with an increased risk for cardiovascular disease, diabetes, and obesity (Booth-Kewley & Friedman, 1987). Growth functions are also suspended during the stress response by inhibiting the release of growth hormone (GH; Kosten, Jacobs, Mason, Wahby, & Atkins, 1984). (p. 355) When this occurs over a prolonged period in children, the result is psychosocial dwarfism, where a child typically grows to only one-half of the expected height for his or her age group (Green, Campbell, & David, 1984). These children have low endogenous GH and, depending on both their age and the duration of the chronic stress, can be unresponsive to exogenous GH supplementation (Albanese et al., 1994; Sapolsky, 1998).

Chronic stress can also lead to a variety of reproductive issues (Rabin, Gold, Margioris, & Chrousos, 1988; Whirledge & Cidlowski, 2010). An increase in stress-related hormones (e.g., β-endorphins and CRH) inhibits the release of gonadotropin-releasing hormone

(GnRH), resulting in reduced gonadotropins such as luteinizing hormone (LH) and follicle-stimulating hormone (FSH; Briski & Sylvester, 1991; Dubey & Plant, 1985). Both LH and FSH are involved in reproductive processes in males and females. In females, GCs inhibit the release of, and reduce sensitivity to, gonadotropins, which increases the likelihood of anovulatory cycles (i.e., menstrual cycles where an ovum is not released; Whirledge & Cidlowski, 2010). GCs also inhibit the secretion of progesterone, which is involved in the maturation of the uterine wall in preparation for egg implantation, decreasing the likelihood of successful implantation if ovulation occurs and in extreme cases halting menstruation completely. In males, GCs decrease testicular sensitivity to LH and lower testosterone, which can decrease sperm count and quality (Bambino & Hsueh, 1981; Saez, Morera, Haour, & Evain, 1977). Moreover, chronic stress activates the sympathetic nervous system and deactivates the parasympathetic nervous system, which increases the likelihood of erectile dysfunction and premature ejaculation (Agarwal, Nandipati, Sharma, Zippe, & Raina, 2006; Bancroft & Janssen, 2000).

Chronic stress likewise has negative consequences on the brain and peripheral nervous system due to high levels of GCs causing cellular atrophy and impaired neurogenesis. Specifically, chronic stress has been linked to neural degeneration in areas of the brain such as the hippocampus, which is involved in learning and memory functions (McEwen & Sapolsky, 1995; Sapolsky, Krey, & McEwen, 1985). Several studies have supported the link between decreased hippocampal volume and decreased performance on spatial tasks in rats (Luine, 2002; Luine, Villegas, Martinez, & McEwen, 1994). In humans, this has best been studied in individuals with Cushing’s syndrome, a disorder characterized by the overproduction of GCs. Individuals with Cushing’s syndrome have lower hippocampal volume than average and perform worse on verbal recall tasks (Bourdeau et al., 2002; Starkman, Gebarski, Berent, & Schteingart, 1992). Furthermore, the HPA axis can be damaged by chronic stress, resulting in a stunted cortisol response to stressors (Glaser & Kiecolt-Glaser, 2014). This is particularly true when the stress occurs early in development; pre- and perinatal chronic stress has been linked to HPA axis malfunction in both rats and rhesus macaques (Clarke, 1993). Similarly, individuals who were sexually abused as children have been found to have damage to their HPA axis, which is thought to be linked to the chronic stress the child experienced as a result of the abuse (Heim, Newport, et al., 2000). In sum, there is a plethora of evidence supporting the link between consistent stressors in one’s environment and its negative impact on health.

Although chronic stress has negative effects on health and bodily functions, the body has protective methods of preventing these effects (Kang, Coe, & McCarthy, 1996; Liu et al., 2002). There are a number of characteristics that modulate the body’s resilience to the side effects of chronic stress (Coe & Lubach, 2003). One such characteristic is age, whereby children and the elderly are most susceptible to negative health outcomes from stress. An increased risk of disease when faced with a stressful environment was found in both elderly humans and monkeys (Bailey & Coe, 2002; Coe & Ershler, 2001; Kiecolt-Glaser, Marucha, Mercado, Malarkey, & Glaser, 1995; Uno, 1997). Infants also show a marked drop in immune function when removed from their mother in humans, rats, and squirrel monkeys (Ader & Friedman, 1965; Coe & Lubach, 2003). Another factor is the

duration of the stressor; if a stressor is present for less than one month, there is no significant increase in the risk of illness. However, if a stressor is present for one month or more, then the risk of the individual developing an illness increases significantly (Cohen et al., 1998; Lepore, Miles, & Levy, 1997). This suggests that the body may be effective at combating the negative effects of chronic stress for a certain period of time only.

Although there is compelling evidence that chronic stress is maladaptive, other specifics regarding how chronic stress impacts health are less clear. Some research suggests that cortisol is the main catalyst for the negative health outcomes associated with stress (Cohen, Kessler, & Gordon, 1995). There are two major theoretical models for the relationship between stress, cortisol, and health. The first (p. 356) model posits that chronic stress results in elevated cortisol (i.e., hypercortisolism) due to HPA axis hyperactivity. The increase in cortisol results in tissue damage and dysregulation of biological systems (Cohen et al., 1995; Schaeffer & Baum, 1984). The second model postulates that chronic stress results in decreased levels of cortisol (i.e., hypocortisolism) due to HPA axis habituation. Decreased cortisol results in fatigue and increased pain sensitivity and leaves the body more vulnerable to disease (Heim, Ehlert, & Hellhammer, 2000; Raison & Miller, 2003; Sternberg, Chrousos, Wilder, & Gold, 1992; Yehuda, 2000). For decades, it was accepted that chronic stress resulted in hypercortisolism, and a considerable number of studies have provided evidence to support this link (e.g., Arnetz et al., 1987; Baum, Gatchel, & Schaeffer, 1983; Schaeffer & Baum, 1984). For example, stressful tasks such as public speaking or mental arithmetic can increase cortisol levels (Kirschbaum, Pirke, & Hellhammer, 1993). However, recent studies by Yehuda and colleagues (Yehuda, 2000; Yehuda, Resnick, Schmeidler, Yang, & Pitman, 1998; Yehuda, Golier & Kaufman, 2005) have called into question whether there are exceptions to this relationship. Hypocortisolism has been most consistently found in individuals who have experienced a traumatic event and, subsequently, developed posttraumatic stress disorder (PTSD; see Yehuda, 1997). This relationship was first observed in Vietnam veterans and then replicated in Holocaust survivors, sexually abused women, and Bosnian prisoners of war (Bourne, Rose, & Mason, 1967, 1968; Dekaris et al., 1993; Yehuda, 2000; Yehuda et al., 1998, 2005). Hypocortisolism has also been found in individuals who have experienced chronic medical disorders, such as chronic pain, fibromyalgia, and asthma (Catley, Kaell, Kirschbaum, & Stone, 2000; Crofford et al., 1995). Furthermore, hypocortisolism has been found in people without medical conditions who have other forms of chronic stress in their lives, for example, parents who had a child with a fatal illness (Friedman, Chodoff, Mason, & Hamburg, 1963) or individuals with a high amount of work stress (Caplan, Cobb, & French, 1979).

What these two major findings suggest is that chronic stress has the ability to both increase and decrease cortisol levels in the body. Considering the strong evidence backing each of these models, the best explanation is an integration of the two (Gunnar & Vazquez, 2001; Heim, Ehlert, & Hellhammer, 2000; Miller, Chen, & Zhou, 2007; Raison & Miller, 2003). One factor that appears to have the greatest impact on cortisol levels is time since the onset of the stressor, whereby time since onset is negatively related to

cortisol level (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Hellhammer & Wade, 1993 Miller, Cohen, & Ritchey, 2002). If the chronic stressor is no longer a part of the environment (e.g., a soldier coming home from a war zone), there is a greater likelihood of hypocortisolism. By comparison, someone who continues to experience the stressor (e.g., unemployment) is more likely to have hypercortisolism (Fries et al., 2005; Hellhammer & Wade, 1993; Miller et al., 2007). This may explain why Yehuda and colleagues (1996, 1998, 2005) found hypocortisolism in those who were suffering from PTSD after experiencing a trauma. Thus, hypocortisolism seems to develop after a period of hyperactivity of the HPA axis, which would explain the perceived contradiction in previous research.

Status

Social Dominance and Dominance Hierarchies

Not only are stress hormones implicated in physiological reactions to stressful environmental cues, but also they appear to play an important role, along with the androgen testosterone, in the maintenance of social dominance and dominance hierarchies (Eisenegger, Haushofer, & Fehr, 2011; Mehta & Josephs, 2010). Dominance hierarchies form in social groups where individuals must compete to obtain resources (e.g., food, mating opportunities) and each individual's ability to compete is different (Sapolsky, 2005). Dominance hierarchies can be described in terms of both their linearity (i.e., the number of binary dominance relationships established within a group and how permanent those relationships are) and their steepness (i.e., how successful an individual of one rank will be against another in a competitive encounter; DeVries, Stevens, & Vervaecke, 2006). In these scenarios, dominant individuals (i.e., those who are best able to acquire and monopolize resources) and subordinate animals (i.e., those who are not as capable) have distinctive physiological and psychological states. This, in part, is because one's rank determines how much physical and psychological stress an individual typically encounters. How different these states are, however, depends on the type of hierarchy that is formed in each species and within each sex.

Dominance hierarchies exist on a spectrum ranging from egalitarian, where no individual is assigned a rank and dominance is achieved with the support of subordinate animals, to despotic, where one individual is dominant and suppresses subordinate (p. 357) animals (van Schaik, 1989). In egalitarian hierarchies, subordination is not associated with increased levels of physiological stress, whereas rank can influence how much stress an individual is exposed to in despotic hierarchies (although this relationship depends on the stability of the hierarchy; Sapolsky, 2005). Despotic hierarchies can also be stable or unstable, depending on whether or not rank is inherited in that species (reviewed in Sapolsky, 2005). When rank is inherited, subordinate animals consistently experience the highest levels of stress. In groups where rank is fluid, whether or not dominants or subordinates experience the most stress depends on the stability of the hierarchy. In these types of hierarchies, high rank can be maintained through either physical

aggression or psychological intimidation. In species where aggression is used to maintain rank, dominant animals at the top of the hierarchy bear a higher burden (e.g., increased parasite load) due to continually elevated levels of cortisol (Muehlenbein & Watts, 2010), putatively as a response to the demands of maintaining that dominance (e.g., via fighting, protecting resources, having to remain on high alert; Cavigelli & Caruso, 2015). Conversely, in groups where psychological intimidation is used by dominants to suppress subordinates, subordinates experience more stress, presumably because of their decreased access to necessary physical resources (Sapolsky, 2005).

Males and females also tend to have different types of intrasexual dominance hierarchies. For males, maintaining social rank is associated with more frequent aggressive behaviors that may result in injury due to enhanced male weaponry (e.g., increased body size, specialized teeth or claws; Cavigelli & Caruso, 2015). In female hierarchies, on the other hand, rank is maintained by more complex, subtle aggression and affiliative behaviors (Sapolsky, 2005). Additionally, whereas males are more driven by access to mates and reproductive opportunities (Ellis, 1995), female dominance hierarchies determine access to quality food resources and protection (Sterck, Watts, & van Schaik, 1997).

Acts of dominance and rankings in dominance hierarchies appear to be associated with the androgen testosterone, and testosterone may motivate individuals to perform behaviors that help them attain and maintain status within social groups (Eisenegger et al., 2011). Testosterone is produced by both sexes in primate species, and is positively correlated with aggression and rank in dominance hierarchies in male (Muller & Wrangham, 2004) and female (Beehner, Phillips-Conroy, & Whitten, 2005) primates. For example, aggressive behavior in male rhesus monkeys is positively associated with elevated levels of plasma testosterone (Rose, Holaday, & Bernstein, 1971), and higher ranking chimpanzees exhibit more aggressive behaviors and have higher levels of testosterone compared to their lower ranking counterparts (Muller & Wrangham, 2004; Muehlenbein, Watts, & Whitten, 2004). Testosterone level is also positively associated with aggressive behaviors (Archer, 2006) and self-perceived dominance (Welling, Moreau, Bird, Hansen, & Carré, 2016) in human males, although the relationship between dominant behavior and testosterone in humans is more mixed (e.g., Johnson, Burk, & Kirk, 2007; Mazur & Booth, 1998).

It has recently been proposed that these discrepancies in findings between testosterone and dominant behaviors and rank in dominance hierarchies can be reconciled when also considering the role of cortisol in dominance interactions and cortisol’s relationship with testosterone (Mehta & Josephs, 2010). Cortisol is known to interact with testosterone in multiple physiological ways (reviewed in Viau, 2002). For example, cortisol is known to disrupt the hypothalamic-pituitary-gonadal (HPG) axis and reproductive function in both males and females (Handa, Burgess, Kerr, & O’Keefe, 1994). The HPG axis controls testosterone production, and increased levels of cortisol suppress the production of endogenous testosterone (Cumming, Quigley, & Yen, 1983). Accounting for these interactions between cortisol and testosterone, Mehta and Josephs (2010) proposed a dual-hormone hypothesis, wherein cortisol modulates the effect that testosterone has on

behavior, such that higher levels of testosterone are associated with more dominant behaviors, but only in individuals who also have low levels of cortisol. They found that cortisol and testosterone coregulate leadership (i.e., dominance) behavior in men and women, and that testosterone and cortisol levels influenced men's competitive behaviors. Importantly, the ratio of cortisol to testosterone was not a significant predictor of an individual's dominant behavior: Testosterone was only associated with increased dominant behaviors in individuals low in cortisol. Indeed, either there was no relationship between testosterone and dominance behaviors in individuals with both high testosterone and cortisol or the relationship was actually reversed, such that participants with high testosterone *and* cortisol displayed less dominant behaviors. Overall, cortisol appears to strongly influence dominance and status in primates, including humans, but these (p. 358) effects may depend on interactions with other hormones, such as testosterone. Similarly, several hormones appear to provide a buffering effect that alleviates some of the harm of stress hormones.

Social Buffering

Oxytocin and Arginine Vasopressin

OT and AVP share a long evolutionary history and mediate a number of socioemotional behaviors in both humans and nonhumans (Carter, Grippo, Pournajafi-Nazarloo, Ruscio, & Porges, 2008; Heinrichs & Domes, 2008; Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). OT neural systems are believed to have originally emerged in mammals to promote affiliative, prosocial, and nurturing behaviors between mothers and infants (Donaldson & Young, 2008) and have putatively been co-opted to regulate basic aspects of sexuality (i.e., sexual arousal, motivation, and orgasm; Borrow & Cameron, 2012; Garrison et al., 2012) and other social bonds (Feldman, 2012; Feldman, Monakhov, Pratt, & Ebstein, 2016). Indeed, receptor distribution for mesotocin (i.e., the functional analogue of OT in birds) is associated with flock size, and mesotocin administration increases flock formation, whereas mesotocin antagonists reduce this social behavior (Goodson, Schrock, Klatt, Kabelik, & Kingsbury, 2009). Likewise, AVP has been shown to mediate prosocial behaviors, aggression, and territoriality in several species, particularly in males (Caldwell, Lee, Macbeth, & Young, 2008; Donaldson & Young, 2008; Young & Wang, 2004). OT and AVP also appear to aid in social synchrony (Apter-Levi, Zagoory-Sharon, & Feldman, 2014). For instance, Bowen and McGregor (2014) found that rats treated with OT and AVP increase defensive aggregation when exposed to an environmental stressor (i.e., cat fur), suggesting that these molecules coordinate social behaviors that assist in responding to environmental threats.

OT and AVP receptors are distributed throughout various brain regions associated with stress and anxiety regulation (Landgraf & Neumann, 2004), and their activation has been shown to modulate experiences of stress in adaptive ways alongside the dopaminergic reward system (Ludwig & Leng, 2006). Some evidence suggests that OT attenuates HPA activity in rodents and nonhuman primates (Neumann & Landgraf, 2012; Parker,

Buckmaster, Schatzberg, & Lyons, 2005) and is associated with lower plasma and salivary cortisol in response to environmental stressors (Cardoso, Ellenbogen, Orlando, Bacon, & Joober, 2013; Ditzen et al., 2009; Legros, 2001). Other studies have shown that peripheral and intranasally administered OT levels are positively associated with circulating cortisol, particularly when subjects expect a stressful experimental manipulation (Brown, Cardoso, & Ellenbogen, 2016). By comparison, AVP has anxiogenic effects (Heinrichs, von Dawans, & Domes, 2009). Rats who naturally or transgenically fail to produce AVP demonstrate lower anxiety (Bielsky, Hu, Szegda, Westphal, & Young, 2004; Zelena et al., 2008). Moreover, elevated plasma AVP is present in several anxiety disorders (Surget & Belzung, 2008) and is associated with territoriality (Caldwell et al., 2008; Donaldson & Young, 2008; Young & Wang, 2004) and behavioral aggression in men after exposure to stress (Moons, Way, & Taylor, 2014). Together, this evidence suggests that OT and AVP play an important role in regulating physiological stress; however, the exact mechanism by which this function is accomplished has yet to be conclusively identified.

Recent efforts at consolidating the disparate effects of OT and AVP on HPA activity have focused on their role in attenuating psychosocial stress (i.e., social buffering; Hostinar, Sullivan, & Gunnar, 2014). Social support and the presence of conspecifics appear to dampen the HPA axis response in both humans (e.g., Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003; Rosal, King, Ma, & Reed, 2004; Taylor et al., 2008) and nonhuman animals (e.g., Hennessy, 1984; Vogt, Coe, & Levine, 1981). For example, maternal contact appears to be formative during HPA axis development in infancy (Gunnar, Hostinar, Sanchez, Tottenham, & Sullivan, 2015). Indeed, maternally deprived infant rats show chronic HPA hyperresponding compared to control rats (Suchecki, Nelson, Van Oers, & Levine, 1995). Likewise, peers also reduce psychosocial stress, though this effect is moderated by individual and interpersonal features (e.g., gender, familiarity, species-typical social organization; Hennessy, Kaiser, & Sachser, 2009). This social buffering appears to be mediated, in part, by circulating levels of OT and AVP (Hostinar et al., 2014). Social activities enhance OT release (Carter, 1998) and, in humans, social support paired with OT administration dampens HPA axis activity in males (Heinrichs et al., 2003). Likewise, Ditzen et al. (2007) found that support from a romantic partner lowers salivary cortisol, but only when paired with physical contact (i.e., a massage). In a sample of international migrants whose OT and AVP were measured shortly after arrival in their host country and reassessed two and five months later, greater baseline levels of OT predicted (p. 359) increases in social relationship satisfaction and social support and decreases in loneliness over time. By comparison, greater social integration was associated with higher plasma AVP over time in the same study (Gouin, Pournajafi-Nazarloo, & Carter, 2015). Collectively, this evidence suggests that OT and AVP are functionally similar but will attenuate or enhance stress in a manner that depends on characteristics of an organism’s social and physical environment.

The actions of OT and AVP in promoting and regulating social behavior and stress also appear to differ between sexes and depend on interactions with gonadal steroids. OT and its receptor are expressed in higher quantity in women (Carter, 2007), and OT gene promoters are stimulated by exposure to estrogen (Lee, Macbeth, Pagani, & Young, 2009;

Mohr & Schmitz, 1991; Richard & Zingg, 1990), whereas AVP synthesis is stimulated by androgens (DeVries & Villalba, 1997). Plasma OT is associated with relationship dissatisfaction in women but not men, whereas plasma AVP is associated with relational distress in men but not women (Taylor, Saphire-Bernstein, & Seeman, 2010). These sex differences may underlie the previously noted divergent responses in males and females to social stressors, whereby women exhibit more OT-based affiliative responses to threat (i.e., tend-and-befriend) than do men (Taylor et al., 2000).

Opioids

Although commonly associated with analgesic effects (e.g., D'Amato, 1998; D'Amato & Castellano, 1989; Kieffer & Gavériaux-Ruff, 2002; Panksepp, 2004), opioids can also promote social attachments and buffer the experience of stress. The brain opioid systems are activated during play in rat pups (Panksepp & Bishop, 1981), social grooming in nonhuman primates (Panksepp, 2004), and positive social interactions in humans (Eisenberger, 2012; Hsu et al., 2013). Additionally, opioids are released in infants upon social (Blass & Fitzgerald, 1988; Panksepp & Bishop, 1981) and physical contact with mothers (Weller & Feldman, 2003) and are known to moderate infant distress vocalizations (DVs; e.g., Kalin, Shelton, & Barksdale, 1988). DVs are produced by infants separated from their normal social environment (e.g., their mother, littermates) and are seen in many vertebrate species, such as mice, rats, chickens, guinea pigs, kittens, puppies, monkeys, and humans (Panksepp, Herman, Conner, Bishop, & Scott, 1978). Furthermore, in rhesus macaques, the anterior cingulate cortex (which has a high concentration of opiate receptors; e.g., Wise & Herkenham, 1982) has been found to play a primary role in the induction of separation calls (Robinson, 1967).

In addition to endogenous opioids, exogenous opioids also alter DVs produced by infants. Low doses of morphine (an opioid receptor agonist; Eisenberger, 2012) reduce DVs from separated infant rats (Carden & Hofer, 1990b), guinea pigs (Herman & Panksepp, 1978), chicks (Panksepp, Vilberg, Bean, Coy, & Kastin, 1978), dogs (Panksepp et al., 1978), and primates (Kalin et al., 1988). By comparison, the administration of naloxone (an opioid antagonist; MacDonald & Leary, 2005) increases DVs in guinea pigs and chicks (Panksepp et al., 1978) and reverses the mitigating effects of littermate presence and morphine administration on reduced DVs in infant rats (e.g., Carden & Hofer, 1990a, 1990b). Exogenous opioids also alter social affiliation in primates, guinea pigs, and rats (MacDonald & Leary, 2005). For example, opioid receptor agonists reduce social interactions with conspecifics, likely by mimicking the rewards of social interactions and thus removing the motivation of pursuing social interactions (Eisenberger, 2012). Conversely, opioid receptor antagonists increase social interaction attempts, likely by blocking the rewards of social interaction and thus motivating its pursuit (Eisenberger, 2012). Furthermore, the effects of both endogenous and exogenous opioids are regulated by μ-opioid receptors. Infant mice lacking the μ-opioid receptor gene, for example, do not experience pain relief from morphine (Eisenberger, 2012), and infant rats lacking the same gene do not exhibit DVs when separated from their mother and littermates (Kehoe & Blass, 1986; Moles, Kieffer, & D'Amato, 2004). It is apparent that social isolation

reduces endogenous opioid levels, inducing social distress and DVs in many vertebrates. On the other hand, social interaction increases endogenous opioids, which reduces social distress and simultaneously reinforces later social interactions (e.g., Nelson & Panksepp, 1998). Ultimately, the opioid system is essential in social behavior, as it both mediates stress response and encourages further social affiliations by rewarding prior social interactions and attachments.

Future Directions

Evidently, chronic stress is maladaptive for a variety of physical and psychological processes. However, the negative effects of chronic stress are mediated by factors such as age and duration of chronic stress, (p. 360) as well as other hormones like OT. Furthermore, future research should work to further integrate the hypo- and hypercortisolism models. At present, endocrinologists have a basic theoretical framework (Miller et al., 2007), but it would be beneficial to understand the catalysts for how the HPA axis switches from hyper- to hypoactivity. There is also a need for a better understanding of the role that cortisol has in dysregulating bodily functions, breaking down tissue, and increasing susceptibility to disease.

Research should further investigate the relationship between stress hormones and dominance, particularly with respect to the interaction with testosterone (Mehta & Josephs, 2010), in both humans and nonhuman primates. Furthermore, possible interactions with other hormones, such as those involved in social buffering, could be explored. Similarly, the role of individual differences in mediating a person's response to both long-term and short-term stress could lead to important clinical applications for the treatment of PTSD and other anxiety-related disorders. One such individual difference factor is sex, whereby women are more likely to suffer from an anxiety disorder during their lifetime compared to men (e.g., McLean, Asnaani, Litz, & Hofmann, 2011; see also Pigott et al., this volume). It is possible that such a sex difference is partially explained by differences in stress-related coping strategies, such as the increased tend-and-befriend response among women compared to men (Tamres et al., 2002; Taylor et al., 2000) or, perhaps relatedly, women's higher OT response to stress (e.g., Taylor et al., 2000). Indeed, recent research suggests that OT may serve to enhance the social salience of environmental cues (Shamay-Tsoory & Abu-Akel, 2016), which could explain women's heightened response to social rejection (Stroud et al., 2002) and, by extension, increased social anxiety (Kessler et al., 2012). In general, more research is needed to further parse apart the various adaptive and maladaptive workings of the human stress response system.

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