Journal of Research in Personality 43 (2009) 942–949

Contents lists available at ScienceDirect

Journal of Research in Personality

journal homepage: www.elsevier.com/locate/jrp

Commentary

The hormonal correlates of implicit power motivation

Steven J. Stanton [a,*], Oliver C. Schultheiss [b]

[a] Duke University, Center for Cognitive Neuroscience, B203 LSRC Building Research Dr., Box 90999, Durham, NC 27708-0999, USA
[b] Friedrich-Alexander University, Department of Psychology and Sport Sciences, Kochstrasse 4, 91054 Erlangen, Germany

ARTICLE INFO

Article history:
Available online 7 April 2009

Keywords:
Implicit power motivation
Dominance
Aggression
Contest
Testosterone
Estradiol
Cortisol
Epinephrine
Norepinephrine
Competition

ABSTRACT

Attempts to link testosterone to dominance dispositions using self-report measures of dominance have yielded inconsistent findings. Similarly, attempts to link testosterone changes to a situational outcome like winning or losing a dominance contest have yielded inconsistent findings. However, research has consistently shown that an indirect measure of an individual's dominance disposition, implicit power motivation, is positively related to baseline testosterone levels and, in interaction with situational outcomes, predicts testosterone changes. We propose a hormonal model of implicit power motivation that describes how testosterone levels change as an interactive function of individuals' implicit power motivation *and* dominance situations. We also propose that estradiol, and not testosterone, plays a key role in dominance motivation in women.

1. Introduction

Over three decades ago, researchers began to investigate the biological underpinnings of implicit power motivation (n Power) (Steele, 1973). N Power is defined as a recurrent concern with and the ability to derive reward from having physical, mental, or emotional impact on other individuals or groups of individuals and to find the experience of others having impact on themselves to be aversive (Winter, 1973). Since then, there has been an increasing interest in and methodical study of the biological basis of n Power, including studies on the roles of the sympathetic catecholamines (McClelland, 1982; McClelland, Floor, Davidson, & Saron 1980; McClelland, Ross, & Patel, 1985; Steele, 1973), testosterone (Schultheiss, Campbell, & McClelland, 1999; Schultheiss & Rohde, 2002; Schultheiss et al., 2005; Stanton & Schultheiss, 2007), cortisol (Wirth, Welsh, & Schultheiss, 2006), estradiol (Stanton & Schultheiss, 2007), as well as the neural correlates of n Power (Schultheiss et al., 2008). Yet, a comprehensive integration of these independent findings is lacking in the literature. In this paper, we will review the literature on the biological basis of n Power and synthesize it with animal research on the physiology of dominance behavior in order to propose a comprehensive, biological model of n Power.

After introducing n Power and explaining how it is measured, we will discuss the biological underpinnings of n Power and dominance behavior with a focus on their relationships with the steroid hormone testosterone. Next, we will describe how hormone levels change as an interactive function of dominance situations and individuals' n Power. We will document a specific biological cascade that leads to changes in testosterone in men. This biological cascade is moderated at every step by n Power, suggesting that one's dominance disposition is intertwined with one's dominance physiology. In support of our model, we will document extensive animal literature that mapped the biological cascade that results from winning or losing dominance interactions and show that the human literature focusing on the role of n Power strongly converges with the animal literature. We will argue that the ability to document the overlap between the animal literature and the human literature is critical in understanding the underlying biological basis of n Power. We will also discuss the physiological, cognitive, and behavioral changes that result from the hormonal changes that occur after engaging in a dominance interaction. Lastly, we will propose possible directions for future research with a focus on studying the hormonal correlates of n Power in women.

2. Implicit power motivation

As the definition of n Power denotes, power-motivated individuals are concerned with having impact over others, and they derive reward and reinforcement from having this impact (Schultheiss, 2008; Winter, 1973). Power-motivated individuals are more likely to be successful in managerial positions (McClelland & Boyatzis, 1982; McClelland & Burnham, 1976) and to have productive, vi-

* Corresponding author. Fax: +1 919 681 0815.
E-mail address: steven.stanton@duke.edu (S.J. Stanton).

0092-6566/$ - see front matter © 2009 Elsevier Inc. All rights reserved.
doi:10.1016/j.jrp.2009.04.001

brant careers (McClelland & Franz, 1992). They tend to be perceived by others as competent and persuasive (Schultheiss & Brunstein, 2002). However, they also tend to make autocratic business decisions without utilizing the opinions of coworkers of lower status (Fodor & Smith, 1982). Power-motivated individuals take bigger risks in gambling situations to garner attention (McClelland & Teague, 1975; McClelland & Watson, 1973) and are more likely to own ostentatious products (Winter, 1973). Later in life, they often become more generative (McClelland, 1975). Power-motivated presidents are more likely to be considered "great" presidents and are also more likely to go to war with other nations (Winter, 1987; Winter, 1993; Winter, 1996). Power-motivated individuals are also more likely to be violent with their significant others, to abuse alcohol, to be politically radical, and to be sexually promiscuous (Lichter & Rothman, 1981; Mason & Blankenship, 1987; McClelland, Davis, Kalin, & Wanner 1972; Schultheiss, Dargel, & Rohde 2003a).

N Power is assessed by content-coding imaginative stories that research participants write in response to picture cues (typically 4–8); this procedure is called the Picture Story Exercise (PSE) (Smith, 1992; Winter, 1994). A trained coder codes the stories for power imagery, and these scores can be summed to yield an overall n Power score for the individual. The following types of thematic content are coded for n Power in participants' PSE stories: strong and forceful actions that have impact over others, controlling others, influencing or persuading others, offering unsolicited help or advice, impressing others, fame, prestige, reputation, and actions that elicit a strong emotional response in others. The majority of studies on implicit motives use two coders and require an inter-rater reliability correlation of $r > .85$ for the measurement to be considered valid and objective. The coding systems were empirically derived and refined over decades (McClelland, Atkinson, Clark, & Lowell, 1953; Winter, 1973; Winter, 1994). Schultheiss and Pang (2007) found robust retest measurements of stability for implicit motives at retest intervals ranging from 1 day to 1 year.

N Power does not correlate with questionnaire measures of dominance or power, and n Power is more efficacious than self-reported dominance motivation in predicting dominance behavior (King, 1995; McClelland, 1987; McClelland, Koestner, & Weinberger, 1989; Schultheiss, 2001; Schultheiss, 2007; Schultheiss & Pang, 2007; Winter, 1973). Recently, other researchers have created alternative indirect measurements of power motivation and started to examine their convergence with n Power. Sheldon, King, Houser-Marko, Osbaldiston, and Gunz (2007) found that the Implicit Associations Test (IAT) developed for power motivation correlates with the PSE-based version presently discussed. Additionally, the Operant Motive Test (OMT) has been developed as an alternative measure of implicit motives (Kuhl, Scheffer, & Eichstaedt, 2003). These methods and results suggest that implicit power motivation can potentially be assessed in multiple ways, but these methods require further study to establish convergent measurement and criterion validity.

3. Relationships between implicit power motivation, baseline testosterone, and behavior

N Power is positively correlated with baseline testosterone, suggesting that high baseline levels of testosterone manifest themselves in aspects of an individual's personality (Schultheiss, 2007; Winter, 1973). Interestingly, n Power also positively predicts many of the same dominance behaviors that high levels of testosterone are associated with (e.g., entering influential occupations, spousal abuse, drug abuse, risk taking, and sexual promiscuity) (Schultheiss, 2007). Such findings suggest that there is a functional link between n Power and individual differences in testosterone levels. While n Power and testosterone are positively correlated, correlations are in the low positive range, which suggests that n Power and testosterone are not the exact psychological and biological equivalents of each other. Individuals' n Power is shaped by many factors including life experiences in asserting dominance, parenting styles, and heritability, in addition to biological factors like testosterone (McClelland, 1987).

When questionnaire measures of trait or state power motivation, dominance seeking, or aggressiveness are used, researchers rarely find any consistent relationship between individuals' questionnaire scores and their testosterone levels (Anderson, Bancroft, & Wu, 1992; Archer, Birring, & Wu, 1998; Bagatell, Heiman, Rivier, & Bremner, 1994; Dabbs, Jurkovic, & Frady, 1991; Doering et al., 1975; Huesmann, Eron, Lefkowitz, & Walder, 1984; Josephs, Sellers, Newman, & Mehta, 2006; Kreuz & Rose, 1972; Meyer-Bahlburg, Boon, Sharma, & Edwards, 1973; Monti, Brown, & Corriveau, 1977; Stanton & Schultheiss, 2007). Reviews of the testosterone literature have therefore concluded that self-report measures of power and dominance are of little value when studying the relationship between testosterone and dominance (cf. Archer, 2006; Archer, Graham-Kevan, & Davies 2005; Mazur & Booth, 1998; Schultheiss, 2007).

As with n Power and dominance behavior, it is notable that the positive association between testosterone and dominance emerges reliably only when behavioral measures of dominance are employed. Testosterone is positively associated with dominance behavior (Mazur & Booth, 1998). The most straightforward evidence for this link comes from studies in which testosterone was manipulated experimentally and its causal effects on behavior could be assessed. For example, in a randomized, placebo-controlled study, Pope and colleagues (2000) found that men treated with testosterone had both increased aggression and symptoms of mania when compared to controls. van Honk and colleagues (2001) showed that subjects who were administered testosterone had greater cardiac acceleration to dominance signals than those given placebo. Studies on the causal effects of testosterone on aggressive and dominance-related behavior are consistent with findings from correlational studies on testosterone and behavior. For instance, trial lawyers who argue in front of judge and jury are more likely to have high testosterone levels than lawyers not representing their clients in court (Dabbs, Alford, & Fielden, 1998). Prisoners with high testosterone are more likely to have a history of violent crime and to have other prisoners rate their behavior as more aggressive (Dabbs et al., 1991; Kreuz & Rose, 1972). When behavior ratings are derived from observers, positive relationships between testosterone and dominance or aggression are consistently observable (Jeffcoate, Lincoln, Selby, & Herbert, 1986; Lindman, Jarvinen, & Vidjeskog, 1987; Scaramella & Brown, 1978). These and many other findings document that, generally, high levels of testosterone promote the pursuit of dominance and status in socially acceptable ways, but that in some cases they can also lead to aggression, antisocial behavior, and sometimes violent crime (Mazur & Booth, 1998). Relationships between dominance and testosterone have been principally documented in men, and our understanding of the relationships between testosterone and dominance in women is less complete (Mazur & Booth, 1998). As we will later show, however, recent evidence suggests that estradiol is related to dominance in women similarly to the way that testosterone and dominance are related in men (Stanton & Schultheiss, 2007).

4. Dynamic biological model of implicit power motivation

Testosterone levels are not static; rather they are in constant flux and change in response to social interactions. In animals and

humans, testosterone levels change as a function of dominance contests and experiences, and these changes in testosterone feed forward to drive changes in behavior (e.g. willingness to compete in another contest) (Mazur, 1985; Mehta & Josephs, 2006; Sapolsky, 1987). However, attempts to predict similar testosterone changes in response to dominance contest outcomes in human subjects have yielded inconsistent results. While some studies have shown that testosterone rises in dominance contest winners and falls in losers (Booth, Shelley, Mazur, Tharp, & Kittok, 1989; Campbell, O'Rourke, & Rabow, 1988; Elias 1981; Mazur & Lamb, 1980), other studies have failed to report main effects of dominance contest outcomes on testosterone change (Edwards, Wetzel, & Wyner, 2006; Gonzales-Bono, Salvador, Serrano, & Ricarte, 1999; Kivlighan, Granger, & Booth, 2005; Maner, Miller, Schmidt, & Eckel, 2008; McCaul, Gladue, & Joppa, 1992; Mehta & Josephs, 2006; Salvador, Simon, Suay, & Llorens, 1987; Stanton & Schultheiss, 2007; also, for related reviews see Archer (2006), Archer et al. (2005)).

In response to the persistent problems associated with looking only at situational factors (winning or losing) in relation to the dynamic biology of human dominance, we will propose a comprehensive biological model of dominance interactions that includes individual differences in n Power, as well as situational factors. In addition to describing the specific biological changes that result after a dominance contest, we will also discuss how those biological changes promote changes in behavior.

4.1. Implicit power motivation moderates testosterone responses to winning or losing a dominance contest

N Power consistently moderates the effect that dominance contest outcomes have on testosterone changes (Schultheiss, 2007). To go beyond correlational links between n Power and hormones, studies have used experimental variation of dominance contest outcomes to examine the effects of n Power on hormone changes (Schultheiss & Rohde, 2002; Schultheiss et al., 2005; Stanton & Schultheiss, 2007; Wirth et al., 2006). These studies placed two same-sex participants together to have them compete face-to-face on variations of implicit learning tasks. The fixed winner would win a majority of the rounds and the fixed loser would lose the same proportion of rounds. When individuals engage in these dominance competitions, the resulting changes in their testosterone levels depend not only on whether they win or lose, but also on their level of n Power (Schultheiss, 2007). Hormone changes in blood occur in a matter of seconds to minutes. Changes in salivary hormones manifest themselves roughly 15 min after the event that drives the release of the hormones into blood. In the contest studies, the researchers therefore collected pre-contest salivary samples, as well as several post-contest salivary samples to determine changes in hormone levels (Schultheiss & Stanton, 2009). Unlike studies that examined only the impact of situational factors (i.e., contest outcomes) on testosterone, the results of studies using n Power as a moderator have been consistently replicated (Schultheiss, 2007). When using dominance contest methods with experimentally-varied outcomes, studies with male German (Schultheiss & Rohde, 2002) and US students (Schultheiss et al., 1999; Schultheiss et al., 2005) found that n Power predicted testosterone increases after a contest victory and testosterone decreases after a defeat (see Fig. 1). Notably, in one study, the mere anticipation of a dominance victory was sufficient to make power-motivated men's testosterone levels rise (Schultheiss et al., 1999). Other methods of arousing n Power, like watching movies depicting dominance, also drive increases in testosterone (Schultheiss, Wirth, & Stanton, 2004). N Power is "aroused" when individuals are placed in a situation where they have the ability to fulfill the motive by being dominant, having the psychological experience of dominance, or through vicarious dominance. We use the term "arouse" as originally introduced by McClelland et al. (1953), that is, to denote the activation of a motivational need by the presence of suitable motivation incentives. Future work could also explore the effects of other power arousing stimuli such as role-playing, status-elevation, or remembering past powerful or powerless life events, or competitive negotiating on both n Power and testosterone to be able to generalize more broadly about the endocrine changes that result from power motivation arousal (Chen, Lee-Chai, & Bargh, 2001; Magee, Galinsky, & Gruenfeld, 2007).

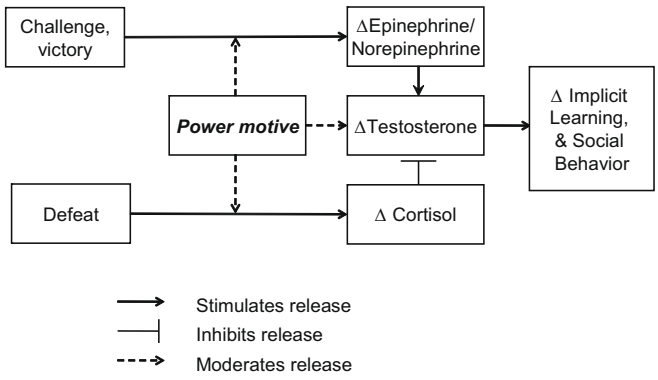

Fig. 1. Biological model of n Power for men.

4.2. Changes in behavior and physiology as a function of testosterone change

Contest-induced testosterone changes can have potentially adaptive effects. Testosterone increases promote the engagement in another dominance contest and lower one's threshold for aggressive engagement, a conclusion that is supported by both animal and human studies (Archer, 2006; Mazur, 1985). In mammals, engagement in another dominance contest may be to one's benefit after having won a contest, by allowing one to further ascend a dominance hierarchy through winning as facilitated by several physiological and learning enhancements (Albert, Jonik, & Walsh, 1992; Mazur, 1985; Wingfield, Hegner, Dufty, & Ball 1990). Testosterone increases could potentially lead to increased muscle anabolism, as shown through in vitro studies (Tsai & Sapolsky, 1996). In rats, testosterone increases have also been linked to reward and reinforcement (Alexander, Packard, & Hines, 1994), and in mice testosterone surges after winning contests can act as reinforcers for effective dominance behavior (Oyegbile & Marler, 2005). Research on human subjects also shows that victory-induced testosterone increases predict better implicit learning of behavior that was instrumental to winning the contest, whereas defeat-induced testosterone decreases predict impaired implicit learning of such behavior (Schultheiss & Rohde, 2002; Schultheiss et al., 2005) (see Fig. 1). These studies suggest that testosterone change is involved in learning the behaviors that lead to winning dominance contests. Conversely, decreases in testosterone as a function of losing make one less motivated to engage in another dominance contest and do not reinforce antecedent behaviors. Thus, after losing a dominance contest, decreases in testosterone make it less likely one will expend more energy on the costly pursuit of power.

The bulk of the evidence for effects of transient testosterone changes on learning and behavior comes from the animal literature. For example, Oyegbile and Marler (2005) showed in mice that testosterone increases as a function of winning dominance interactions, and the likelihood of future wins in dominance contests strongly increases after a series of prior testosterone-increasing wins, which suggests that testosterone increases also have a reinforcing effect on dominance pursuit. Research on this issue using

human participants is both difficult and rare, yet there are some studies that have attempted to test the changes in behavior and cognition following contest outcomes. In a study reporting on men's choices to compete again after a contest, Mehta and Josephs (2006) showed that contest-induced testosterone increases predicted men's inclination to engage in another contest, whereas testosterone decreases predicted men's behavioral withdrawal from dominance situations. Their finding affirms research in animals.

4.3. A primer on hormone axes and hormone release

Before outlining our biological model of n Power, we will present a primer on the hormone axes that are implicated in our model. In the following sections, we will argue that changes in testosterone release in the context of dominance contests result from changes in two classes of hormones and that n Power moderates the release of all hormones in the model. The first hormone is cortisol, which is produced by the cortex of the adrenal glands sitting on top of the kidneys. When animals experience stress, the hypothalamus, located at the base of the brain, receives signals from other brain areas (e.g., the amygdala). The hypothalamus releases corticotropin-releasing hormone to the pituitary gland, which in turn releases adrenocorticotropic hormone into the bloodstream. Adrenocorticotropic hormone then travels to the cortex of the adrenal gland, which releases the stress hormone cortisol into the bloodstream. The released cortisol travels to the periphery and also feeds back to the brain where it curtails the release of more cortisol from the adrenals. In its totality, this loop of hormonal communication comprises the hypothalamic–pituitary–adrenal (HPA) axis. With relevance to the current review, released cortisol travels to and acts on the testes, which are principally responsible for testosterone production and release in males (Sapolsky, 1991).

The second class of hormones that are relevant to our model are the sympathetic catecholamines epinephrine and norepinephrine (also called adrenaline and noradrenaline, respectively), which are produced by the sympathetic–adrenal–medullary (SAM) axis. Unlike the long-loop nature of the HPA axis, which involves several "releasing" hormones that travel to targets to stimulate the release of yet more hormones, the medulla (core) of the adrenal gland is stimulated directly by the sympathetic nervous system through efferent nerves that emerge from the spinal cord. The sympathetic catecholamines are released from the adrenal medulla into the bloodstream. Like cortisol, the catecholamines travel to and have effects on the testes' release of testosterone in males (Sapolsky, 1991).

The catecholamines are released in response to arousal, broadly defined. SAM axis activation occurs even in the presence of weak stressors, resulting in the relatively frequent release of catecholamines (Sapolsky, 2002). In contrast, the HPA axis is less likely to become activated in the presence of weak stressors (Sapolsky, 2002). However, the HPA and SAM axes are both activated in response to strong stressors, which then results in the simultaneous release of cortisol and catecholamines (Goldstein & Kopin, 2008; Sapolsky, 2002).

The HPA and SAM axes also influence each other, but the exact nature of the interaction between the axes is a topic of continued debate and research. Rodent studies show that large increases in the sympathetic catecholamines have a stimulatory effect on the HPA axis (Axelrod & Reisine, 1984; Tsigos & Chrousos, 2002). However, the magnitude of the effect of the catecholamines on cortisol release in response to stress, independent of the cortisol release that would be produced by the HPA axis alone, is unclear. Conversely, rodent studies have shown that increases in cortisol also lead to modulation of epinephrine and norepinephrine release in response to stress (Kvetnansky et al, 1995). Yet, the direction of that modulation depends on the duration of the cortisol administration; one-shot administration of cortisol reduces the catecholamine response to stress (Kvetnansky et al., 1995; Tsigos & Chrousos, 2002), yet chronic cortisol administration increases the catecholamine response to stress (Kvetnansky et al., 1995). However, in humans, there is evidence that acute administration of cortisol fails to cause a reduction in the catecholamine response to stress, a finding that contradicts rodent studies (Malarkey, Lipkus, & Cacioppo, 1995). In studies looking at natural human stress responses, the SAM and HPA axes are activated at the same time. In contrast, the reported rodent studies use large hormone administrations *before* the stressor to examine the effect the hormone has on the reactivity of the opposing axis. Thus, the cross-talk of the hormone axes during their simultaneous response to a single stressor is not well-researched in humans. In addition to having regulatory effects on each other, we will subsequently present evidence that cortisol and the sympathetic catecholamines uniquely influence testosterone release and that n Power moderates the release of all these hormones in response to dominance interactions.

4.4. Rapid effects of sympathetic catecholamines and cortisol on testosterone release in males

Testosterone changes drive a host of physiological, psychological, and behavioral changes, but there is an integrated biological cascade that leads up to testosterone change. In animals, changes in cortisol, epinephrine, and norepinephrine have a causal effect on changes in testosterone. Sapolsky (1985, 1986, 1987) conducted several studies in baboons that pinpointed these biological precursors to changing levels of testosterone. To do so, he induced stress in wild male baboons darting them with an immobilizing drug – a stressful experience for the animals. The resulting biological response to stress depended on a baboon's rank. High-ranking baboons had a surge in testosterone and low-ranking baboons had a decrease in testosterone. In the high-ranking baboons, Sapolsky discovered that pharmacological blocking of catecholamine action also abolished increases in testosterone, which suggested that the catecholamine release in response to the darting had a stimulating effect on the testes, driving the rapid release of testosterone. The low-ranking baboons had cortisol surges after darting, and Sapolsky discovered that administering a cortisol-like substance also led to decreases in testosterone, which suggests that cortisol dampens the testes' sensitivity to endocrine signals (luteinizing hormone) that normally drive testosterone release. Thus, there are two mechanisms driving testosterone release in different directions.

Sapolsky (1987, 1991) concluded that changes in testosterone in response to stress reflected a balance between a catecholamine response to stressors that are perceived as manageable and a cortisol response to stressors that are perceived as uncontrollable. If individuals' catecholamine response is greater than their cortisol response, their testosterone levels will rise, which is more commonly observed in dominant individuals. In contrast, if individuals' cortisol response is greater than their catecholamine response, their testosterone levels will fall, which is more commonly observed in non-dominant individuals. Thus, Sapolsky's data suggest a balance model of testosterone release, in which the determining factor in predicting testosterone release is perception of a stressor and the accompanying ratio of released catecholamines and released cortisol in response to that stressor. Evidence from studies of n Power in humans studies parallel and corroborate Sapolsky's findings.

4.5. Implicit power motivation arousal leads to changes in catecholamine levels

Steele (1973) produced the first research on the biological components of n Power arousal by examining the activation of the

sympathetic nervous system as a function of n Power arousal (McClelland, 1987). Steele experimentally manipulated power motivation arousal by having participants listen to power-arousing, achievement-arousing or non-arousing audio tapes and then measured changes in metabolites of the sympathetic catecholamines, epinephrine and norepinephrine, as well as changes in implicit motives. Participants had significant increases in n Power and in epinephrine and norepinephrine only in the power-arousal condition. Moreover increases in epinephrine and norepinephrine were positively correlated with increases in n Power. Steele concluded that n Power arousal is uniquely tied to activation of the sympathetic nervous system as demonstrated by increases in the catecholamines. Building on Steele's (1973) research, McClelland and colleagues (1980) looked at the effect of power challenges on catecholamine levels of power-motivated college men. Power challenges varied from being physically threatened to having to deal with administration at the college. Men high in n Power and high in activity inhibition had elevated levels of epinephrine in response to the power challenges. (Activity inhibition is a measure of self-control that is associated with right-hemispheric brain functions including HPA and SAM axis regulation [Schultheiss, Riebel, & Jones, in press].) McClelland and colleagues (1985) measured changes in norepinephrine as a function of taking a stressful exam and n Power. They argued that the exam was a power challenge, because students' social status was principally determined by their academic performance. McClelland and colleagues found that students with a strong power motive (relative to their affiliation motive) had significant increases in norepinephrine, both immediately after the exam and 105 min later. In conjunction, these studies show that various types of n Power arousal drive increases in the catecholamines in power-motivated individuals (see Fig. 1).

4.6. Implicit power motivation frustration leads to changes in cortisol levels

Wirth and colleagues (2006) explored the effects of n Power frustration on the stress hormone cortisol in both sexes. In humans and other mammalian species, cortisol acutely rises in response to situations that are perceived to be uncontrollable and stressful (Kirschbaum & Hellhammer, 2000; Sapolsky, 2002). Wirth and colleagues (2006) hypothesized that losing a dominance competition would be stressful and frustrating to a power-motivated individual. In two studies, Wirth and colleagues (2006) measured participants' baseline levels of n Power and cortisol, and had participants compete in one-on-one contests based on a cognitive task. The goal was to frustrate or satisfy power-motivated individuals by experimentally varying the contest outcome. Wirth and colleagues (2006) found that participants' cortisol levels changed as an interactive function of both the contest outcome and their n Power. Across both studies, in both sexes, higher levels of n Power were associated with a greater cortisol increase in losers and a greater cortisol decrease in winners. Similarly, Mehta and colleagues (2008) found that baseline levels of testosterone predicted cortisol changes after a dominance contest, in which high-testosterone men who lost had cortisol increases and those who won had cortisol decreases. They argued that baseline testosterone is a marker of dispositional power, which is a conceptually similar to our view of n Power. Wirth and colleagues (2006) argued that losing a dominance contest is particularly stressful for individuals who like to be dominant, that is, power-motivated individuals. Moreover, the stress of power-motive frustration via losing drives cortisol increases selectively in power-motivated individuals.

There are important parallels between Sapolsky's (1987) research and the research on n Power. It has been suggested that individual differences in n Power are similar to dominance ranks in animals, because power-motivated humans often behave in dominant ways similar to high-dominance members of other species as reflected in higher aggression, coercion, risk-taking, and sexual activity (cf. McClelland, 1975; Schultheiss, 2007; Schultheiss et al., 2003a). Moreover, as was described, humans' patterns of catecholamine and cortisol responses to power arousal and frustration, which vary as a function of individuals' n Power, mirror baboons' patterns of catecholamine and cortisol responses, which vary as a function of dominance rank. Sapolsky's research provides a link to n Power research by suggesting that the documented increases in catecholamines via power motivation arousal reported by McClelland and colleagues (1980, 1985) can also lead to increases in testosterone in power-motivated individuals. Sapolsky's research also suggests that the documented increases in cortisol as a function of losing a dominance contest can lead to decreases in testosterone in power-motivated individuals (see Fig. 1).

Our biological model of n Power in men predicts that changes in cortisol, epinephrine, and norepinephrine, as well as subsequent testosterone changes, as an interactive function of n Power and situations, should fall into a specific pattern (see Fig. 1). We have now highlighted evidence showing that n Power moderates changes in cortisol, epinephrine, norepinephrine, and testosterone in men. To review, in power-motivated individuals, n Power arousal corresponds not only to rising levels of catecholamines but also to rising levels of testosterone, while n Power frustration leads to increases in cortisol and decreases in testosterone. These results fit nicely with the theoretical benefits of testosterone changes. Power-motivated individuals enjoy dominance and find dominance experiences rewarding (Winter, 1973). For power-motivated individuals, rising levels of testosterone after winning are likely to drive psychological and physiological preparedness to pursue dominance again (cf. Mazur, 1985). Testosterone increases facilitate power-motivated individuals' pursuit of dominance. Falling levels of testosterone after losing do not drive psychological and physiological preparedness to pursue dominance again, which may be unwise given an antecedent loss (Mazur, 1985).

5. Future directions

Research on the biological basis of implicit motives has a number of unanswered questions that remain open for exploration, some of which we would like to highlight. While animal research has demonstrated direct effects of the catecholamines and cortisol on changes in testosterone, this has not been demonstrated directly in humans. Rather, the reviewed studies separately examined situation $\times$ n Power effects on each hormonal component of our model. It has yet to be shown within a single study that all components of this model fall into place, which is an important direction for future research. Moreover, such a study would also allow us to examine the possible cross-talk between the HPA and SAM axes in humans.

Despite a clear set of relationships between n Power and testosterone in men, studies have not consistently linked testosterone to n Power in women (Schultheiss, 2007). Broadly speaking, the majority of behavioral endocrinology research on dominance in humans has focused on testosterone and principally used male subjects (Mazur & Booth, 1998). However, animal studies have demonstrated that estradiol can positively influence dominance behavior or the motivation to attain dominance in females of several mammalian species (Bouissou, 1990; Faruzzi, Solomon, Demas, & Huhman, 2005; Michael & Zumpe, 1993; Zehr, Maestripieri, & Wallen, 1998; Zumpe & Michael, 1989). Some researchers have proposed that estradiol might have a more direct connection to dominance in women (Cashdan, 1995; Cashdan, 2003; Schultheiss, 2007). In response to these speculations, Stan-

ton and Schultheiss (2007) examined the relationship between n Power and estradiol in women. Replicating an earlier observation by Schultheiss, Dargel, and Rohde (2003b), they found that baseline estradiol levels and n Power were positively related. Further exploring the n Power-estradiol relationship, Stanton and Schultheiss (2007) employed a dominance contest method similar to the one previously used with men (Schultheiss et al., 2005) to examine estradiol changes after a dominance contest. They found that higher levels of n Power were associated with greater estradiol increases after winning. Conversely, after losing a dominance contest, higher levels of n Power were associated with greater estradiol decreases. While our model of catecholamine and cortisol changes driving testosterone changes works well in explaining the effects of n Power on men's testosterone responses to winning and losing a contest (Schultheiss, 2007), the extent to which catecholamine and cortisol responses to dominance challenges can have similar effects on estradiol release in women is unknown. Whereas research by Sapolsky (1985, 1986, 1987) explained the biological precursors to testosterone change in males, research has yet to document biological precursors to rapid estradiol changes in females. A greater focus on female dominance and its biological correlates is needed in both human and animal research.

Replication of the estradiol effects in women as a response to dominance contests is imperative. Further, if estradiol changes also mediate behaviors that are instrumental to the outcome of a dominance contest, that would suggest that estradiol change is not only a response to the situation, but is also critically linked to the shaping of the behaviors are instrumental to the contest outcome. Documenting such behavioral mediation by estradiol in women would make estradiol a more complete parallel to testosterone in men, since testosterone changes mediate such behaviors in men (Schultheiss & Rohde, 2002; Schultheiss et al., 2005). Moreover, the subsequent changes in behavior and social cognition as an effect of estradiol change in women are also unknown and would be a potential area for future exploratory research. By placing such experiments in a broader context, exploration of changes in real-life outcome behaviors as a function of testosterone or estradiol change in response to dominance contests would bolster this line of research with greater ecological validity.

Exploration of a potential relationship between frustrated n Power, cortisol, and depression is also a worthy path for research. Frustrated n Power has been linked to immune system impairment, heart disease, and excessive consumption of alcohol (see McClelland, 1987 for a review), and the positive link between n Power frustration and cortisol release could compellingly extend this line of research into explorations of psychopathology including depression.

From a neuropsychological perspective, fMRI holds considerable promise for the examination of the neurological basis of individual differences, and researchers in personality neuroscience are beginning to exploit this tool. Schultheiss and colleagues (2008) recently published the first study to examine the moderating role of n Power on patterns of brain activation. With relevance to the biological model of n Power, the hypothalamus is largely in control of hormone axes (hypothalamic–pituitary–gonadal and hypothalamic–pituitary–adrenal), as well as aspects of dominance behavior. Studies of dominance could use neuroimaging to measure the relationship between brain activation of the hypothalamus and its connection with other parts of the emotional brain and subsequent hormone release as a function of n Power. Such work could help further uncover how the brain orchestrates the complex hormonal responses to dominance challenges and stressors in the context of implicit power motivation.

Acknowledgments

Parts of the research reported in this article were supported by a National Institute of Mental Health Grant (R03 MH63069-01/02), NSF Grant BCS 0444301, Deutsche Forschunsgemeinschaft Grants SCHU 1210/1-2, 1-3, and 2-2, and a Rackham Predoctoral Fellowship from the University of Michigan. We would like to thank Jacinta C. Beehner for helpful comments on an earlier version of this manuscript.

References

Albert, D. J., Jonik, R. H., & Walsh, M. L. (1992). Hormone-dependent aggression in male and female rats: experiential, hormonal, and neural foundations. *Neuroscience and Biobehavioral Reviews, 16*(2), 177–192.

Alexander, G. M., Packard, M. G., & Hines, M. (1994). Testosterone has rewarding affective properties in male rats: implications for the biological basis of sexual motivation. *Behavioral Neuroscience, 108*, 424–428.

Anderson, R., Bancroft, J., & Wu, F. (1992). The effects of exogenous testosterone on sexuality and mood of normal men. *Journal of Clinical Endocrinology and Metabolism, 75*, 1503–1507.

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

Archer, J., Birring, S. S., & Wu, F. C. W. (1998). The association between testosterone and aggression among young men: empirical findings and a meta-analysis. *Aggressive Behavior, 24*, 411–420.

Archer, J., Graham-Kevan, N., & Davies, M. (2005). Testosterone and aggression: a reanalysis of book, Starzyk and Quinsey's (2001) study. *Aggression and Violent Behavior, 10*, 241–261.

Axelrod, J., & Reisine, T. D. (1984). Stress hormones: their interaction and regulation. *Science, 224*, 452–459.

Bagatell, C., Heiman, J., Rivier, J., & Bremner, W. (1994). Effects of endogenous testosterone and estradiol on sexual behavior in normal young men. *Journal of Clinical Endocrinology and Metabolism, 78*, 711–716.

Booth, A., Shelley, G., Mazur, A., Tharp, G., & Kittok, R. (1989). Testosterone, and winning and losing in human competition. *Hormones and Behavior, 23*, 556–571.

Bouissou, M. F. (1990). Effects of estrogen treatment on dominance relationships in cows. *Hormones and Behavior, 24*, 376–387.

Campbell, B., O'Rourke, M., & Rabow, M. (1988). *Pulsatile response of salivary testosterone and cortisol to aggressive competition in young males*. Paper presented at the annual meeting of the American Association of Physical Anthropologists, Kansas City, Missouri.

Cashdan, E. (1995). Hormones, sex, and status in women. *Hormones and Behavior, 29*, 354–366.

Cashdan, E. (2003). Hormones and competitive aggression in women. *Aggressive Behavior, 29*, 107–115.

Chen, S., Lee-Chai, A. Y., & Bargh, J. A. (2001). Relationship orientation as a moderator of the effects of social power. *Journal of Personality and Social Psychology, 80*, 173–187.

Dabbs, J. R., Alford, E. C., & Fielden, J. A. (1998). Trial lawyers and testosterone: blue-collar talent in a white-collar world. *Journal of Applied Social Psychology, 28*, 84–94.

Dabbs, J. M., Jr., Jurkovic, G., & Frady, R. (1991). Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology, 19*, 469–478.

Doering, C., Brodie, K., Kraemer, H., Moos, R., Becker, H., & Hamburg, D. (1975). Negative affect and plasma testosterone. *Psychosomatic Medicine, 37*, 484–491.

Edwards, D. A., Wetzel, K., & Wyner, D. R. (2006). Intercollegiate soccer: saliva cortisol and testosterone are elevated during competition, and testosterone is related to status and social connectedness with teammates. *Physiology and Behavior, 87*, 135–143.

Elias, M. (1981). Serum cortisol, testosterone, and testosterone-binding globulin responses to competitive fighting in human males. *Aggressive Behavior, 7*, 215–224.

Faruzzi, A. N., Solomon, M. B., Demas, G. E., & Huhman, K. L. (2005). Gonadal hormones modulate the display of submissive behavior in socially defeated female Syrian hamsters. *Hormones and Behavior, 4*, 569–575.

Fodor, E. M., & Smith, T. (1982). The power motive as an influence on group decision making. *Journal of Personality and Social Psychology, 42*, 178–185.

Goldstein, D. S., & Kopin, I. J. (2008). Adrenomedullary, adrenocortical, and sympathoneural response to stressors: a meta-analysis. *Endocrine Regulations, 42*, 111–119.

Gonzales-Bono, E., Salvador, A., Serrano, M. A., & Ricarte, J. (1999). Testosterone, cortisol, and mood in a sports team competition. *Hormones and Behavior, 35*, 55–62.

Huesmann, L., Eron, L., Lefkowitz, M., & Walder, L. (1984). Stability of aggression over time and generations. *Developmental Psychology, 20*, 1120–1134.

Jeffcoate, W., Lincoln, N., Selby, C., & Herbert, M. (1986). Correlation between anxiety and serum prolactin in humans. *Journal of Psychosomatic Research, 29*, 217–222.

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*(6), 999–1013.

King, L. (1995). Wishes, motives, goals, and personal memories: relations of measures of human motivation. *Journal of Personality, 63*, 985–1007.

Kirschbaum, C., & Hellhammer, D. H. (2000). Salivary cortisol. In G. Fink (Ed.), *Encyclopedia of stress*. San Diego: Academic Press.

Kivlighan, K. T., Granger, D. A., & Booth, A. (2005). Gender differences in testosterone and cortisol response to competition. *Psychoneuorendocrinology, 30*, 58–71.

Kreuz, L., & Rose, R. (1972). Assessment of aggressive behavior and plasma testosterone in a young criminal population. *Psychosomatic Medicine, 34*, 321–332.

Kuhl, J., Scheffer, D., & Eichstaedt, J. (2003). Der operante motiv-test (OMT): Ein neuer ansatz zur messung impliziter motive [The operant motive test (OMT): A new approach to implicit motive assessment]. In F. Rheinberg & J. Steinmeier-Pelster (Eds.), *Diagnostik von motivation und selbstkonzept* (pp. 129–150). Goettingen, Germany: Hogrefe.

Kvetnansky, R., Pacak, K., Fukuhara, K., Viskupic, E., Hiremagalur, B., Nankova, B., et al. (1995). Sympathoadrenal system in stress: interaction with the hypothalamic–pituitary–adrenocortical system. *Annals of the New York Academy of Sciences, 771*, 131–158.

Lichter, S. R., & Rothman, S. (1981). Jewish ethnicity and radical culture: a social psychological study of political activists. *Political Psychology, 3*, 116–157.

Lindman, R., Jarvinen, P., & Vidjeskog, J. (1987). Verbal interactions of aggressively and nonaggressively predisposed males in a drinking situation. *Aggressive Behavior, 13*, 187–196.

Magee, J. C., Galinsky, A. D., & Gruenfeld, D. H. (2007). Power, propensity to negotiate, and moving first in competitive interactions. *Personality and Social Psychology Bulletin, 33*, 200–212.

Malarkey, W. B., Lipkus, I. M., & Cacioppo, J. T. (1995). The dissociation of catecholamine and hypothalamic–pituitary–adrenal responses to daily stressors using dexamethasone. *Journal of Clinical Endocrinology, 80*, 2458–2463.

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.

Mason, A., & Blankenship, V. (1987). Power and affiliation motivation, stress, and abuse in intimate relationships. *Journal of Personality and Social Psychology, 52*, 203–210.

Mazur, A. (1985). A biosocial model of status in face-to-face primate groups. *Social Forces, 64*, 377–402.

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

Mazur, A., & Lamb, T. (1980). Testosterone, status, and mood in human males. *Hormones and Behavior, 14*, 236–246.

McCaul, K., Gladue, B., & Joppa, M. (1992). Winning, losing, mood, and testosterone. *Hormones and Behavior, 26*, 486–506.

McClelland, D. C. (1975). *Power: The inner experience*. New York: Irvington.

McClelland, D. C. (1982). The need for power, sympathetic activation, and illness. *Motivation and Emotion, 6*, 31–41.

McClelland, D. C. (1987). *Human motivation*. New York: Cambridge University Press.

McClelland, D. C., Atkinson, J. W., Clark, R. A., & Lowell, E. L. (1953). *The achievement motive*. New York: Appleton-Century-Crofts.

McClelland, D. C., & Boyatzis, R. E. (1982). Leadership motive pattern and long-term success in management. *Journal of Applied Psychology, 67*, 737–743.

McClelland, D. C., & Burnham, D. H. (1976). Power is the great motivator. *Harvard Business Review, 54*, 100–110.

McClelland, D. C., Davis, W. N., Kalin, R., & Wanner, E. (1972). *The drinking man*. New York: Free Press.

McClelland, D. C., Floor, E., Davidson, R. J., & Saron, C. (1980). Stressed power motivation, sympathetic activation, immune function, and illness. *Journal of Human Stress, 6*, 11–19.

McClelland, D. C., & Franz, C. E. (1992). Motivational and other sources of work accomplishments in mid-life: a longitudinal study. *Journal of Personality, 60*, 679–707.

McClelland, D. C., Koestner, R., & Weinberger, J. (1989). How do self-attributed and implicit motives differ? *Psychological Review, 96*, 690–702.

McClelland, D. C., Ross, G., & Patel, V. (1985). The effect of an academic examination on salivary norepinephrine and immunoglobulin levels. *Journal of Human Stress, 11*, 52–59.

McClelland, D. C., & Teague, G. (1975). Predicting risk preferences among power related tasks. *Journal of Personality, 43*, 266–285.

McClelland, D. C., & Watson, R. I. (1973). Power motivation and risk-taking behavior. *Journal of Personality, 41*, 121–139.

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

Meyer-Bahlburg, H., Boon, D., Sharma, M., & Edwards, J. (1973). Aggressiveness and testosterone measures in man. *Psychosomatic Medicine, 36*, 269–274.

Michael, R. P., & Zumpe, D. (1993). A review of hormonal factors influencing the sexual and aggressive behavior of macaques. *American Journal of Primatology, 30*, 213–241.

Monti, P., Brown, W., & Corriveau, D. (1977). Testosterone and components of aggressive and sexual behavior in man. *American Journal of Psychiatry, 134*, 692–694.

Oyegbile, T. O., & Marler, C. A. (2005). Winning fights elevates testosterone levels in California mice and enhances future ability to win fights. *Hormones and Behavior, 48*(3), 259–267.

Pope, H. G., Jr., Kouri, E. M., & Hudson, J. I. (2000). Effects of supraphysiologic doses of testosterone on mood and aggression in normal men: a randomized controlled trial. *Archives of General Psychiatry, 57*(2), 133–140.

Salvador, A., Simon, V., Suay, F., & Llorens, L. (1987). Testosterone and cortisol responses to competitive fighting in human males. *Aggressive Behavior, 13*, 9–13.

Sapolsky, R. M. (1985). Stress-induced suppression of testicular function in the wild baboon: role of glucocorticoids. *Endocrinology, 116*, 2273–2278.

Sapolsky, R. M. (1986). Stress-induced elevation of testosterone concentration in high ranking baboons: role of catecholamines. *Endocrinology, 118*, 1630–1635.

Sapolsky, R. M. (1987). Stress, social status, and reproductive physiology in free-living baboons. In D. Crews (Ed.), *Psychobiology and reproductive behavior: An evolutionary perspective* (pp. 291–322). Englewood Cliffs: Prentice Hall.

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

Sapolsky, R. M. (2002). Endocrinology of the stress-response. In J. B. Becker, S. M. Breedlove, D. Crews, & M. M. McCarthy (Eds.), *Behavioral endocrinology* (pp. 409–450). Cambridge: MIT Press.

Scaramella, T., & Brown, W. (1978). Serum testosterone and aggressiveness in hockey players. *Psychosomatic Medicine, 40*, 262–263.

Schultheiss, O. C. (2001). An information processing account of implicit motive arousal. In M. L. Maehr & P. Pintrich (Eds.). *Advances in motivation and achievement, new directions in measures and methods* (Vol. 12, pp. 1–41). Greenwich: JAI Press.

Schultheiss, O. C. (2007). A biobehavioral model of implicit power motivation: arousal, reward, and frustration. In *Social neuroscience: Integrating biological and psychological explanations of social behavior* (pp. 176–196). New York: Guilford Press.

Schultheiss, O. C. (2008). Implicit motives. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed., pp. 603–633). New York: Guilford Press.

Schultheiss, O. C., & Brunstein, J. C. (2002). Inhibited power motivation and persuasive communication: a lens model analysis. *Journal of Personality, 70*, 553–582.

Schultheiss, O. C., Campbell, K. C., & McClelland, D. C. (1999). Implicit power motivation moderates men's testosterone responses to imagined and real dominance success. *Hormones and Behavior, 36*, 234–241.

Schultheiss, O. C., Dargel, A., & Rohde, W. (2003a). Implicit motives and sexual motivation and behavior. *Journal of Research in Personality, 37*, 224–230.

Schultheiss, O. C., Dargel, A., & Rohde, W. (2003b). 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., Riebel, K., & Jones, N. M. (in press). Activity inhibition: A predictor of lateralized brain function during stress? *Neuropsychology*.

Schultheiss, O. C., & Pang, J. S. (2007). Measuring implicit motives. In R. W. Robins, R. C. Fraley, & R. Kruegger (Eds.), *Handbook or research methods in personality psychology* (pp. 322–344). New York: Guilford Press.

Schultheiss, O. C., & Rohde, W. (2002). Implicit power motivation predicts men's testosterone changes and implicit learning in a contest situation. *Hormones and Behavior, 41*, 195–202.

Schultheiss, O. C., & Stanton, S. J. (2009). Assessment of salivary hormones. In E. Harmon-Jones & J. Beer (Eds.), *Methods in the neurobiology of social and personality psychology* (pp. 17–44). New York, NY: Guilford.

Schultheiss, O. C., Wirth, M. M., & Stanton, S. J. (2004). Effects of affiliation and power motivation arousal on salivary progesterone and testosterone. *Hormones and Behavior, 46*(5), 592–599.

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.

Schultheiss, O. C., Wirth, M. M., Waugh, C. E., Stanton, S. J., Meier, E., & Reuter-Lorenz, P. (2008). Exploring the motivational brain: effects of implicit power motivation on brain activation in response to facial expressions of emotion. *Social, Cognitive, and Affective Neuroscience, 3*, 333–343.

Smith, C. P. (Ed.). (1992). *Motivation and personality: Handbook of thematic content analysis*. New York: Cambridge University Press.

Sheldon, K. M., King, L., Houser-Marko, L. A., Osbaldiston, R., & Gunz, A. (2007). Comparing IAT and TAT measures of power versus intimacy motivation. *European Journal of Personality, 21*, 263–280.

Stanton, S. J., & Schultheiss, O. C. (2007). Basal and dynamic associations between implicit power motivation and estradiol in women. *Hormones and Behavior, 52*(5), 571–580.

Steele, R. S. (1973). *The physiological concomitants of psychogenic motive arousal in college males*. Unpublished dissertation thesis, Harvard University, Boston, MA.

Tsai, L. W., & Sapolsky, R. M. (1996). Rapid stimulatory effects of testosterone upon myotubule metabolism and sugar transport, as assessed by silicon microphysiometry. *Aggressive Behavior, 22*, 357–364.

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research, 53*, 865–871.

van Honk, J., Tuiten, A., Hermans, E., Putman, P., Koppeschaar, H., Thijssen, J., et al. (2001). A single administration of testosterone induces cardiac accelerative responses to angry faces in healthy young women. *Behavioral Neuroscience, 115*, 238–242.

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.

Winter, D. G. (1973). *The power motive*. Free Press.

Winter, D. G. (1987). Enhancement of an enemy's power motivation as a dynamic of conflict escalation. *Journal of Personality and Social Psychology, 52*, 41–46.

Winter, D. G. (1993). Power, affiliation, and war: three tests of a motivational model. *Journal of Personality and Social Psychology, 65*, 532–545.

Winter, D. G. (1994). *Manual for scoring motive imagery in running text* (4th ed.). Unpublished manuscript, Department of Psychology, University of Michigan, Ann Arbor.

Winter, D. G. (1996). *Personality: Analysis and interpretation of lives*. New York: McGraw-Hill.

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.

Zehr, J. L., Maestripieri, D., & Wallen, K. (1998). Estradiol increases female sexual initiation independent of male responsiveness in rhesus monkeys. *Hormones and Behavior, 33*, 95–103.

Zumpe, D., & Michael, R. P. (1989). Female dominance rank and behavior during artificial menstrual cycles in social groups if rhesus monkeys (Macaca mulatta). *American Journal of Primatology, 17*, 287–304.