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Testosterone and Human Aggression

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Hormones modulate various physiological, morphological, and behavioral processes critical to survival and reproduction (Ketterson & Nolan, 1992). Importantly, hormone–behavior associations are bidirectional, whereby hormones may modulate social behavior and social behavior may feed back to influence hormone concentrations. In this chapter, we provide a basic overview of research examining associations between testosterone and human aggression. We begin with a brief review of the literature on the relationship between baseline testosterone concentrations and human aggression. Next, we review evidence that competitive interactions rapidly modulate testosterone concentrations. We then discuss an emerging literature examining the role of competition-induced fluctuations in testosterone in potentiating ongoing and/or future aggressive behavior. In this chapter, we take a comparative approach drawing on both the human and nonhuman literature to provide a sense of the commonalities in the basic neuroendocrine mechanisms underlying complex social behavior.

Basal Testosterone and Aggression

In 1849, Arnold Berthold performed the first formal experiment in behavioral neuroendocrinology (Berthold, 1849). Berthold's experiments consisted of removing the testes of male roosters and observing how this manipulation influenced morphological and behavioral processes. Berthold found that castration essentially eliminated crowing and the development of secondary sex characteristics (e.g., large comb). Moreover, castrated males did not engage in any mating or aggressive behavior—all classic behaviors expressed by male roosters. Berthold then reimplanted the testes into the body cavity of the roosters, and the male phenotype developed, consisting of crowing, mating, and aggression. Since the testes did not form any neural connections after being placed in the body cavity, Berthold concluded that the testes must synthesize and secrete a substance into the bloodstream, ultimately influencing the typical adult male phenotype.

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Today, we know that the substance synthesized and released from the testes is the steroid hormone testosterone. Testosterone is a hormone synthesized by the Leydig cells of the testes in men, the thecal cells of the ovaries and placenta of women, and, to a lesser extent, the adrenal cortex of both men and women. Testosterone synthesis is governed by the hypothalamic–pituitary–gonadal (HPG) axis. Neurons within the hypothalamus secrete gonadotropin-releasing hormone into the hypophyseal portal system, which binds to receptors in the anterior pituitary and stimulates the release of luteinizing hormone and follicular stimulating hormone. Luteinizing hormone travels through the blood stream, binding to receptors on the Leydig cells of the testes and the thecal cells of the ovaries, stimulating the synthesis and secretion of testosterone. Upon crossing the blood–brain barrier, testosterone may interact with receptors located in key brain structures known to mediate various complex social behaviors, including mating and aggression (Newman, 1999).

Several lines of indirect evidence provide support for the idea that testosterone may promote aggressive behavior in humans. A large body of work indicates that men are much more physically aggressive than women (Archer, 2004) and have much higher testosterone concentrations than women (Dabbs, 1990); in addition, at a time when testosterone concentrations peak (ages 21–35), there is an increase in male-to-male aggressive behavior (Daly & Wilson, 1988). Over the years, there has been a major increase in the number of published studies examining the relationship between testosterone and human aggression (see Figure 20.1).

One research strategy aimed at examining links between testosterone and human aggression involves comparing testosterone levels of violent versus nonviolent offenders. This area of research broadly indicates that testosterone concentrations are higher in men and women convicted of violent crimes in comparison to those convicted of nonviolent crimes (for a review see Dabbs, 1993). These findings may suggest that individuals with elevated testosterone concentrations are more likely to commit violent crimes. However, equally plausible is that people who commit violent crimes are also more aggressive while in prison, which may transiently increase testosterone.

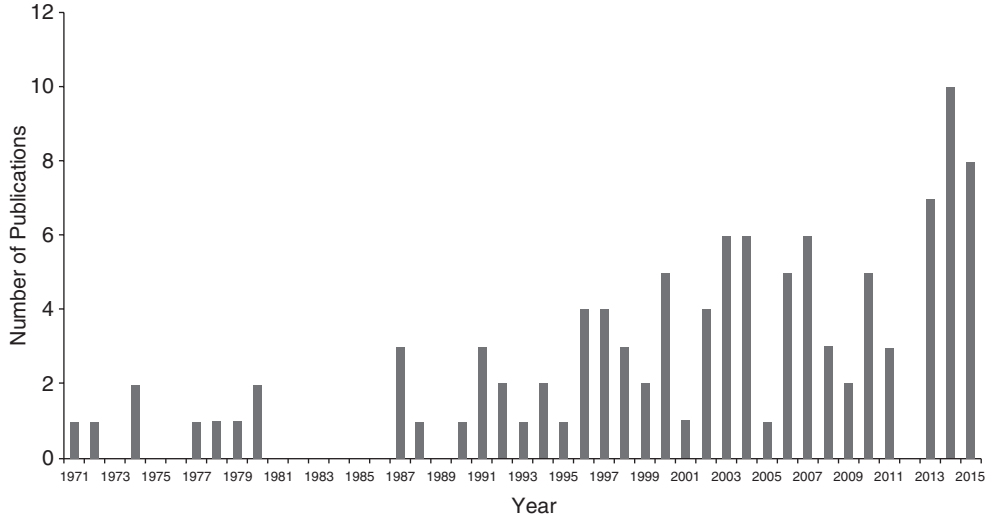

Figure 20.1 Number of publications examining testosterone and aggression, 1971–2015.

Another research strategy has been to examine the relationship between testosterone and human aggression using paper-and-pencil self-report measures. In one study, individual differences in aggressive feelings were positively correlated with serum testosterone concentrations in young men ($r = .49$, $N = 18$; Persky, Smith, & Basu, 1971). Despite this early report, studies with much larger sample sizes (e.g., $N = 100–250$) have failed to find any association between baseline testosterone and self-report measures of aggression (e.g., Archer, Birring, & Wu, 1998; Monti, Brown, & Corriveau, 1977; Popma et al., 2007). Nevertheless, a meta-analysis by Archer, Graham-Kevan, and Davies (2005) reported a positive relationship between testosterone and various self-report measures of aggression ($r = .08$).

A third research strategy has been to examine the relationship between baseline testosterone and behavioral measures of aggression. One study reported positive correlations (average $r = .38$) between precompetition testosterone concentrations and indices of behavioral aggression in male judo fighters (Salvador, Suay, Martinez-Sanchis, Simon, & Brain, 1999). Other studies have used well-validated laboratory paradigms. Berman, Gladue, and Taylor (1993) reported that baseline testosterone concentrations were positively correlated ($r = .41$) with aggressive behavior on the Taylor Aggression Paradigm. The Taylor Aggression Paradigm is a laboratory paradigm in which participants compete against a fictitious opponent on a reaction time task. Prior to each trial, participants are required to set a shock (or noise blast) intensity that will be administered to their fictitious opponent if the opponent loses the trial. Aggressive behavior in this task is defined as the average shock (or noise blast) intensity that participants deliver to their opponent on win trials (Giancola & Parrott, 2008).

Some recent studies have reported that testosterone concentrations are positively correlated with the extent to which participants reject unfair offers in the ultimatum game (Burnham, 2007; Mehta & Beer, 2010; but see Eisenegger, Naef, Snozzie, Heinrichs, & Fehr, 2010). The ultimatum game is a task whereby a "proposer" is given a sum of money and has the opportunity to offer as much or as little money to a "receiver." Once the offer is made, the receiver has the choice to either accept or reject the offer. If the offer is accepted, both participants receive their split of the money. If the receiver rejects the offer, both participants leave with no money. Economic theory predicts that receivers should accept any offer greater than zero. However, numerous studies indicate that proposals that are below 20% of the sum are generally rejected (Camerer & Thaler, 1995). Rejection behavior on the ultimatum game can be considered a form of aggression as it is committed with the intent to cause harm to another individual (i.e., financial harm), who, in turn, is motivated to avoid such treatment.

Other researchers have used the Point Subtraction Aggression Paradigm (PSAP; Cherek, 1981) to examine the association between baseline testosterone concentrations and aggression (Gerra et al., 1997). The PSAP is a computer task in which participants are paired with a fictional opponent and the main goal of the task is to gain as many points as possible—the more points earned, the more money participants receive. During the task, participants have points taken from them by their fictitious opponent (actually a computer program). Participants have three response options available to them: (1) reward response, (2) steal response, and (3) protection response. Gerra and colleagues (1997) reported a positive association ($r = .41$) between baseline testosterone concentrations and aggressive behavior in young men ($N = 30$; Gerra et al., 1997). Using an experimental approach, Pope, Kouri, and Hudson (2000) reported that 6 weeks of testosterone treatment increased aggressive behavior in men tested on the PSAP. It is important to note that the authors found no change in self-reported aggression following testosterone treatment, indicating that the behavioral measure of aggression was more sensitive to the effects of testosterone treatment.

In summary, studies of the relationship between baseline testosterone concentrations and aggression in people have yielded small and inconsistent effects. Importantly, competitive and aggressive interactions are known to potentiate testosterone release (Oliveira, 2009; Wingfield, Hegner, Dufiy, & Ball, 1990), suggesting that the relationship between testosterone and aggressive behavior is much more complex than previously thought. In fact, testosterone concentrations are highly responsive to competitive interactions in a number of taxa including birds (Wingfield et al., 1990), fish (Oliveira, 2009), nonhuman primates (Bernstein, Rose, & Gordon, 1974), humans (Archer, 2006), and insects (Scott, 2006). The following section will present an overview of this literature, beginning with a description of the challenge hypothesis and the biosocial model of status, two of the main theoretical models guiding current research on the bidirectional relationship between testosterone and aggressive behavior.

Competition-Induced Changes in Testosterone

The challenge hypothesis was originally developed to explain intra- and interspecies variation in testosterone secretion in birds. Wingfield et al. (1990) noted that testosterone concentrations fluctuate around three levels during the season: constitutive baseline (level A), breeding baseline (level B), and physiological maximum (level C). In monogamous male birds that provide paternal care, testosterone concentrations remain relatively low (level A) during the nonbreeding season. Concentrations increase (level B) at the start of the breeding season as a means to initiate spermatogenesis: the expression of secondary sex characteristics and the full display of male reproductive behavior. Finally, concentrations may reach peak levels (level C) in response to intrasexual competitive interactions as a means to facilitate aggression and territorial behavior. When intrasexual competition decreases, testosterone concentrations return to the constitutive baseline. It has been proposed that the costs associated with maintaining elevated testosterone concentrations throughout the season (e.g., decreased paternal care, increased risk for physical injury/death, depressed immune function, increased energetic demands) may have led to a highly flexible endocrine system capable of rapidly adjusting testosterone concentrations in response to changes in the social environment (Wingfield, Lynn, & Soma, 2001).

The biosocial model of status (Mazur, 1985) is a conceptually similar theoretical model to the challenge hypothesis, with the additional prediction that testosterone concentrations during competition will vary as a function of the outcome of the competitive interaction: increasing in winners and decreasing in losers. Mazur (1985) speculated that winners of competitive interactions may face additional challenges for status and the increase in testosterone may serve to promote competitive and aggressive behaviors aimed at maintaining and defending one's status. In contrast, the decrease in testosterone in response to defeat serves to promote submissive behaviors aimed at avoiding further loss of status and/or physical injury.

Most studies testing hypotheses derived from the biosocial model of status come from sport competitions (Archer, 2006; Salvador, 2005). The first study in humans to demonstrate the effect of competition outcome on testosterone reactivity patterns was based on a small sample of male varsity tennis players. The authors reported that men had an increase in testosterone after a victory and a decrease in testosterone after a defeat (Mazur & Lamb, 1980). A similar effect was observed in another small study of male varsity wrestlers in which winners had elevated postcompetition testosterone concentrations relative to losers (Elias, 1981). The importance of the objective outcome in modulating testosterone release is corroborated by research

in male cichlid fish. In that study, the authors took advantage of the fact that cichlid fish cannot recognize their own image in a mirror, and, as a result, attack their image as if the image were that of another fish. The authors reasoned that, if aggressive behavior is the driving force behind the rise in testosterone, then these fish should have an increase in testosterone in the "mirror-challenge" condition (Oliveira, Carneiro, & Camirio, 2005). In contrast, if the outcome of the interaction is critical to modulating testosterone release, then no testosterone change would be expected based on the fact that there was no clear outcome. These researchers' results were consistent with the latter hypothesis; testosterone concentrations were nonresponsive to the mirror-elicited challenge, despite the finding that the fish increased their rate of aggressive behavior during the testing period (Oliveira et al., 2005).

Some studies suggest that the effect of competition outcome on testosterone release may occur in individuals not directly involved in the competitive interaction. Bernhardt, Dabbs, Fielden, and Lutter (1998) obtained pre- and postgame saliva samples from male spectators attending college basketball and professional soccer games. Supporters of the winning and losing teams demonstrated an increase and a decrease in testosterone, respectively. In a more recent series of studies, male varsity hockey players watched videos of themselves engaged in a victory, a defeat, or a neutral documentary film. Carré and Putnam (2010) reported that testosterone concentrations increased significantly after watching a video of a previous victory, but not after a video depicting a previous defeat or a neutral video. A similar "spectator effect" has been observed in male cichlid fish. In their experiment, Oliveira, Lopes, Carneiro, and Camirio (2001) had fish watch two isolated conspecific male neighbors through a one-way mirror. After a period of habituation, the opaque partition separating the two neighbors was removed, allowing the bystander fish to observe their neighbors engaged in an aggressive interaction. Results indicated that the experimental bystanders exposed to an aggressive interaction had significantly higher testosterone and 11-ketotestosterone (a metabolite of testosterone) concentrations relative to the control bystanders not exposed to an aggressive interaction. These studies provide compelling support for the idea that simply watching competition can have similar effects on the neuroendocrine system to those observed when one actually engages in competition.

In summary, the literature reviewed in this section suggests that testosterone is highly responsive to competitive interactions and that winners typically have elevated testosterone concentrations relative to losers. Most of these results have been interpreted from a functional perspective whereby acute changes in testosterone may serve to promote competitive and aggressive behaviors.

Context-Dependent Changes in Testosterone and Aggressive Behavior

Several authors have speculated that changes in testosterone during competition may enable an organism to adjust its ongoing and/or future social behavior according to changes in the social environment (Archer, 2006; Mazur, 1985; Oliveira, 2009; Wingfield et al., 1990). In one of the first studies to test this functional hypothesis, Klinesmith, Kasser, and McAndrew (2006) randomly assigned men to interact with a toy gun or with a board game. The authors hypothesized that interacting with a toy gun would represent a social challenge and that, in accordance with research on the effects of social challenge (Archer, 2006; Wingfield et al., 1990), this would produce an acute increase in testosterone concentrations. After interacting with the toy gun (or board game), participants were given a cup of water and were instructed

to add as much or as little hot sauce as they wished to the cup, which would later be consumed by another participant. The amount of hot sauce placed in the cup served as the primary measure of aggression. As predicted, men who interacted with the toy gun demonstrated a robust increase in testosterone concentrations and were much more aggressive than participants who interacted with the board game. Critically, the relationship between interacting with the toy gun and aggressive behavior was mediated by testosterone responses to the task.

In another experiment, Carré, Putnam, and McCormick (2009) had men and women compete in same-sex dyads on a rigged laboratory competition wherein half were randomly assigned to a win condition and half to a loss condition. After the competitive interaction, participants performed the PSAP with the same opponent. For men, a rise in testosterone during the competitive interaction predicted increased aggression in the subsequent interaction (Carré et al., 2009). In another study, the same research group reported that changes in testosterone in response to social inclusion (but not social exclusion) were positively correlated with subsequent reactive aggression among men tested on the PSAP (Geniole, Carré, and McCormick, 2011). In more recent work, competition was modeled using an Xbox Kinect video game (Carré, Campbell, Lozoya, Goetz, & Welker, 2013). A large sample of men and women ($N = 237$) were randomly assigned to experience a string of victories or defeats in either a boxing or a volleyball game. The results indicated that male winners had elevated testosterone concentrations and aggressive behavior compared to male losers. Moreover, the effect of winning on subsequent aggressive behavior was statistically mediated by heightened testosterone concentrations after the victory (Carré et al., 2013).

Finally, it was recently demonstrated that a long-term intervention program designed to curtail antisocial behavior in "at-risk" youth was successful, in part, because it dampened testosterone responses to social provocation. This intervention was implemented in preschool, and the children assigned to the intervention condition received social–cognitive–behavioral therapy while those assigned to the control condition received no such treatment. When tested 20 years later, the intervention group demonstrated less aggression on the PSAP and decreased testosterone reactivity to social provocation compared to the control group. Notably, the association between assignment to the intervention condition and decreased aggression was statistically mediated by decreased testosterone reactivity to provocation (Carré, Iselin, Welker, Hariri, & Dodge, 2014). Collectively, these findings are consistent with the idea that acute fluctuations in testosterone within the context of human competition may have important effects on current and/or future social behavior. The effects of testosterone dynamics on aggression in the studies reviewed here were found exclusively in men (Carré et al., 2013). Because testosterone concentrations were not experimentally manipulated, it is not possible to make stronger causal claims concerning testosterone's role in modulating competitive and aggressive behavior.

Animal models are particularly useful for testing causal mechanisms resulting in complex social behavior. In experiments, administration of testosterone to male California mice after they experienced a victory produced increased aggressive behavior in subsequent interactions (Fuxjager, Oyegbile, & Marler, 2011; Gleason, Fuxjager, Oyegbile, & Marler, 2009; Trainor, Bird, & Marler, 2004) and increased the probability of them winning subsequent interactions (Fuxjager et al., 2011; Gleason et al., 2009). In addition, Oliveira, Silva, and Canario (2009) examined the role of testosterone in mediating the "winner" and "loser" effects in male tilapia. In control fish, winners of a first aggressive interaction were more likely to win a subsequent aggressive interaction (88% won the second fight), whereas losers were more likely to lose subsequent interactions (87% lost the second fight). However, winners treated with an

antiandrogen drug, which prevented the normal increase in testosterone in response to competitive interactions, were less likely to win a subsequent aggressive interaction (relative to control males). In contrast, losers treated with 11-ketotestosterone were not more likely to win a subsequent aggressive interaction. These findings indicate that the winner effect depends critically on acute fluctuations in testosterone. Going beyond the role of circulating testosterone concentrations, Fuxjager and colleagues (2010) reported that the winner effect is due to an up-regulation of androgen receptors in several key brain regions involved in reward and motivation (e.g., nucleus accumbens and ventral tegmental area) as well as social aggression (bed nucleus of the stria terminalis). Together, the studies discussed in this section provide support for the role of competition-induced testosterone dynamics in mediating ongoing and/or future social behavior.

Future Directions and the Dual-Hormone Hypothesis

Despite evidence linking testosterone to human aggression and/or dominance behaviors, these relationships are either weak or inconsistent. Consequently, some researchers have shifted focus toward the interaction of testosterone with other hormones—namely, cortisol. In a seminal paper, Dabbs, Jurkovic, and Frady (1991) examined the relationship between testosterone, cortisol, and violent crime among 113 incarcerated violent male offenders. The findings from this study showed that testosterone was positively correlated with inmates' previous violent crime, but this was only true among those with relatively low levels of cortisol. This modulating effect of cortisol has seen growing attention in what is now commonly termed the "dual-hormone hypothesis" (Mehta & Josephs, 2010), which posits that the extent to which testosterone plays a role in status-relevant behavior (e.g., aggression) will depend critically on variability in circulating concentrations of cortisol (see Mehta & Prasad, 2015, for a review). Specifically, testosterone should be positively related to aggression, but only among individual with low levels of cortisol.

In support of the dual-hormone hypothesis, Popma et al. (2007) found that, among 103 young males in a delinquency diversion program, testosterone was positively related to self-reported overt aggression, but only among those with low levels of cortisol. A separate study found that, among female athletic intercollegiate competitors, testosterone was positively related to status as rated by teammates, but only among individuals low in cortisol (Edwards & Casto, 2013). Recent behavioral studies also show similar findings to those using self- or other-reported measures. For instance, Pfattheicher, Landhäußer, and Keller (2014) found that individuals were more likely to choose an option to punish others in a public goods game when their testosterone levels were high, an effect that was found exclusively among those with low cortisol levels. Most recently, Mehta, Welker, Zilioli, and Carré (2015) found that, across two studies, basal testosterone was positively associated with risk taking in various forms, but only among individuals with low levels of cortisol.

These early studies suggest that cortisol may function antagonistically in the link between testosterone, on the one hand, and human social dominance and aggressive behavior, on the other. Mechanisms accounting for this effect occur through cortisol down-regulation of androgen receptor levels, joint hormonal influence on neural activity in specific brain areas (e.g., amygdala, frontal lobes), and global psychological conceptualizations such as cortisol's influence on stress and behavioral inhibition (Mehta & Josephs, 2010). Future investigations are needed to fully understand the biological and psychological mechanisms underlying the

dual-hormone hypothesis. Further, it may also benefit researchers to investigate more complex interactions between cortisol, testosterone, and personality variables, as one study provides preliminary evidence to suggest that the dual-hormone hypothesis can predict parent-rated externalizing problems, but only at high levels of disagreeableness and emotional instability (Tackett, Herzhoff, Harden, Page-Gould, & Josephs, 2014).

Androgen Receptor Gene: CAG Repeat Polymorphism

The biological actions of testosterone are in part mediated by testosterone's binding to the androgen receptor. Recent work suggests that the relationship between testosterone and aggression or dominance may be moderated by variability in the number of CAG repeats upstream of the androgen receptor. The degree of androgen sensitivity appears to be inversely related to the length of CAG repeats, and, as a result, it should be expected that testosterone's effects on physiological and behavioral processes will be more pronounced among those with relatively shorter CAG repeats within the androgen receptor (Chamberlain, Driver, & Miesfeld, 1994; Choong, Kemppainen, Zhou, & Wilson, 1996). Indeed, some recent evidence suggests that this may be the case. For instance, Rajender and colleagues (2008) found that, when comparing violent offenders (i.e., those convicted of murder and/or rape) to a control group, the offenders had significantly shorter CAG repeats within the androgen receptor. Additionally, Vermeersch, T'sjoen, Kaufman, and Vincke (2010) found that, in a large sample of adolescent boys ($N = 300$), testosterone was positively related to aggressive risk taking, nonaggressive risk taking, and dominance behaviors, but only among those with relatively shorter CAG repeats within the androgen receptor. Given this preliminary evidence, and that human phenotypic displays may depend on androgen receptor sensitivity, CAG repeat length appears to be an important consideration for future research examining links between testosterone and human dominance or aggressive behavior.

Personality Traits, Testosterone Reactivity, and Aggression

Evidence suggests that individual differences in personality traits may moderate the relationship between testosterone and aggressive behavior. In one study, a rise in testosterone among winners of a competitive interaction positively predicted subsequent aggression, but only among men scoring relatively high on trait dominance (Carré et al., 2009). In two other studies (Norman, Moreau, Welker, & Carré, 2015), a rise in testosterone during competition was associated with heightened aggressive behavior, but only among men (not women) scoring relatively low in trait anxiety. These findings are notable in the light of animal research showing that trait anxiety modulates neuroendocrine function and aggression. Specifically, male rats selectively bred for low anxiety showed a heightened testosterone response to social threat and aggressive behavior relative to male rats bred for high anxiety (Veenema, Tomer, Blume, Beiderbeck, & Neumann, 2007). Collectively, these preliminary findings suggest that it will be critical to examine the role of individual difference factors in moderating hormone–behavior relationships in humans.

Neural Mechanisms Through Which Testosterone May Modulate Aggression

An important area for future research will be to examine the neural mechanisms through which testosterone may modulate human aggressive behavior. A large body of evidence from

animal models indicates that several interconnected cortical and subcortical structures within the social behavior network (Newman, 1999) are involved in the modulation of reactive aggression (Nelson & Trainor, 2007). One specific model that has received support from lesion and electrical/chemical stimulation experiments (mainly in rodents and cats) indicates that the medial amygdala, medial hypothalamus, and periaqueductal gray positively modulate reactive aggression (Siegel, Bhatt, Bhatt, & Zalcman, 2007). Specifically, the medial amygdala provides excitatory input to glutamatergic neurons in the medial hypothalamus, which exert excitatory drive on periaqueductal gray neurons, ultimately mediating a reactively aggressive response (Siegel et al., 2007). Research in humans has generally focused on the role of the orbitofrontal cortex (OFC), reporting that individuals with localized lesions to the OFC engage in heightened reactive aggression (see Siever, 2008, for a review). Given the extensive projections from the OFC to limbic structures such as the hypothalamus and amygdala, it has been proposed that the propensity to engage in reactive aggression may emerge from impaired regulatory control of the OFC over these subcortical structures (Nelson & Trainor, 2007).

One research strategy for investigating neural correlates of aggressive behavior has examined behavioral and neural responses to angry facial expressions. Angry facial expressions represent clear affective signals of threat and, depending on the dominance relationship between sender and receiver, these threat stimuli may elicit fight-or-flight responses from the receiver. For instance, dominant individuals may perceive an angry facial expression as a challenge to their status, whereas submissive individuals may perceive the angry facial expression as an enforcement of the prevailing relationship, thus promoting approach and avoidance behaviors, respectively (Van Honk & Schutter, 2007). Behavioral and neuroimaging studies have found that individuals prone to anger and reactive aggression (e.g., intermittent explosive disorder, borderline personality disorder) display attentional biases, enhanced amygdala reactivity, and decreased OFC–amygdala coupling during processing of angry facial expressions, suggesting that such processes may represent a neurobiological marker for one's propensity to engage in reactive aggression (Siever, 2008). Studies in healthy men and women have reported that even normal variation in constructs linked to reactive aggression (e.g., approach motivation, trait anger, trait anxiety) positively map onto variability in amygdala reactivity to angry facial expressions (Beaver, Lawrence, Passamonti, & Calder, 2008; Carré, Fisher, Manuck, & Hariri, 2012). Collectively, these findings converge on a model in which relatively increased amygdala reactivity and/or decreased coupling of prefrontal regions (ventral anterior cingulate cortex, OFC) with the amygdala during processing of threat-related stimuli may bias one's propensity toward engaging in reactive aggression.

Notably, androgen and estrogen receptors are widely distributed throughout the neural circuitry underlying reactive aggression (Newman, 1999), suggesting that testosterone and/or its metabolites (e.g., estradiol) may directly modulate this circuitry by interacting with intracellular androgen or estrogen receptors, which affect gene transcription, protein expression, and ultimately cell function. In addition to this slow genomic mode of action (i.e., it takes several minutes to hours for testosterone to influence gene transcription and subsequent protein formation), testosterone may also influence physiological processes within seconds through rapid nongenomic mechanisms such as activation of G-protein-coupled membrane-bound androgen or estrogen receptors and/or direct modulation of voltage- and ligand-gated ion channels (Michels & Hoppe, 2008). A growing body of neuroimaging data provide support for the idea that testosterone may influence threat-related neural processes. For instance, individual differences in baseline testosterone concentrations correlate positively with amygdala reactivity to facial expressions of anger and fear (Derntl et al., 2009; Manuck et al.,

2010) and correlate negatively with OFC responses to perceived provocation (Mehta & Beer, 2010). Finally, exogenous administration of testosterone has been found to increase amygdala reactivity and decrease amygdala–OFC coupling during processing of angry facial expressions (Goetz et al., 2014; Hermans, Ramsey, & Van Honk, 2008; Van Wingen et al., 2008; Van Wingen, Mattern, Verkes, Buitelaar, & Fernández, 2010). Thus, to the extent that enhanced behavioral and neural responses to social threat bias reactive aggression, the findings reviewed in this section converge on a model in which testosterone may modulate the expression of reactive aggression by enhancing amygdala reactivity and/or decreasing ventral ACC– or OFC–amygdala coupling during processing of social threat (e.g., angry facial expressions, social provocation).

Conclusion

In this chapter we reviewed the current literature on the relationship between testosterone and human aggression. In general, there is a weak and inconsistent positive relationship between baseline testosterone concentrations and various indices of human aggression. More robust is the finding that testosterone concentrations change rapidly in the context of human competition—and that such changes in testosterone concentrations positively predict ongoing and/or future human aggression. We suggest that future research consider the potential role of other hormones (e.g., cortisol) and personality traits as moderators of the relationship between testosterone and human aggression.

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ABSTRACT

One of the most widely studied biological correlates of aggressive behavior is the steroid hormone testosterone. Although traditional wisdom might suggest that individuals with more testosterone are more likely to be aggressive, research over the past several decades has identified important contextual, individual difference, and methodological variables that are key moderators of any such effect. In this chapter, we review literature examining how aggression is linked with baseline levels of testosterone, how testosterone fluctuates rapidly within the context of human competitive behavior, and how such competition-induced hormonal fluctuations serve to potentiate ongoing and/or future aggressive behavior. The neuroendocrine mechanisms underlying such complex social behavior are discussed from research conducted within humans as well as nonhuman species, providing comparative clues as to the adaptive nature of such intricate systems.

KEYWORDS

aggression, biosocial model of status, challenge hypothesis, competition, dual-hormone hypothesis, hormones, neuroendocrine function, social neuroendocrinology, testosterone, violence