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It Is Not Just About Testosterone: Physiological Mediators and Moderators of Testosterone's Behavioral Effects

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Abstract

The inclusion of hormones in social and personality psychology research offers researchers a powerful methodological tool, although careful consideration must be paid to the complex and dynamic biological systems that connect hormones to behavior. Testosterone has been clearly established as a biological fuel for dominance, although behavioral findings have been inconsistent and weak. We suggest a number of physiological systems that affect the influence of testosterone on behavior. Vasopressin synthesis and the aromatization into estradiol both serve to facilitate testosterone's effects. Testosterone's behavioral responses to threats are suppressed by basal cortisol and moderated by allele length on the serotonin transporter gene. Variation in the expression of monoamine oxidase A regulates the levels of neurotransmitters responsible for impulse control, potentially suppressing testosterone's influence over behavior. We argue that researchers must consider these dynamic relationships between hormones and other biological systems when engaging in future social endocrinology research.

As anyone who has observed the behavior of adolescents can safely attest, hormones play a significant role in social behavior. For social and personality psychologists, hormones offer the opportunity to measure a number psychological constructs, both conscious and nonconscious, reliably, validly, and with minimal bias, as well as elucidate biological underpinnings of some social behaviors more easily and cheaply than other social neuroscience methods. For instance, dominance can have a powerful impact on behavior and social functioning and is best measured via testosterone (van Honk, Schutter, Hermans, & Putman, 2004). With regard to stress (which can have a profound effect on physical health), by tracking fluctuations in cortisol, researchers can objectively measure stress uncoupled from subjective experience to gain further understanding of how automatic psychological processes influences physical well-being (Dickerson & Kemeny, 2004).

Testosterone

As the field of social endocrinology progresses and hormone assessment continues to be integrated into social psychological and personality research, it is important for researchers to have a clear understanding of how hormones actually give rise to social behaviors. Within the field of social endocrinology, no hormone has been given greater attention than testosterone, and this review will cover some basic physiological mediators and moderators of testosterone's effects on behavior, covering both those that promote and those than inhibit testosterone's influence.

Testosterone is an androgen, meaning it is a masculinizing steroid hormone. It is produced in both men and women by the gonads, adrenal glands, and within the brain, although because of the fact that the testes produce testosterone in much larger quantities

than any other source, men tend to have roughly seven to ten times as much circulating testosterone as women (Mehta & Josephs, in press). Despite the great sex difference in circulating levels, in many cases testosterone has been shown to exert similar influences on social behaviors across sexes (e.g., Josephs, Sellers, Newman, & Mehta, 2006; Mehta, Jones, & Josephs, 2008; Sellers, Mehl, & Josephs, 2007; Zyphur, Narayanan, Koh, & Koh, 2009). Although most hormones are subject to fluctuations caused by a number of factors, basal (i.e. baseline) steroid hormone levels have been found to stay relatively static over extended periods of time, with test–retest stability of basal testosterone found to be $r = 0.65$ among men and $r = 0.78$ among women over a 2- week span (Liening, Stanton, Saini, & Schultheiss, 2010). This stability of basal testosterone has led many researchers to treat it as a trait-level factor.

If basal hormone levels can be thought of as traits, fluctuations in hormones levels can be conceptualized as transient mood states. Hormones not only influence behavior but are in turn affected by behavior and environmental forces, operating in a reciprocal fashion with the environment (Mazur & Booth, 1998). Specifically, hormones influence behavior, behavior influences the environment, changes in the environment cause temporary fluctuations in circulating hormone levels, and these temporary fluctuations in turn further influence behavior and so on. Hormones act not only as partial causes of behavior but also change in response to behavior.

Dominance

Although testosterone is responsible for a number of functions (e.g., reproduction, skeletal muscle development), a primary psychological effect of testosterone is dominance. 'Dominance' refers to a motivation for attaining and maintaining high status within a social hierarchy (Mazur & Booth, 1998). As with other personality traits, dominance varies between individuals, with some individuals exhibiting higher levels of dominance than others. Individuals high in dominance are not only motivated to attain high status, but they have also been found to actually succeed in achieving high status (e.g., Anderson & Kilduff, 2009). The pursuit of status can be costly in terms of energy expenditure and the potential for harm, but high status is typically rewarded with social and material benefits not available to those lower on the social hierarchy (Mehta et al., 2008). As such, individuals' dominance play a key role in the formation of social hierarchies, the effectiveness of group functioning, and impact not only the individual but also those above (whose position may be challenged) and those below (who are subordinate) in the social rank (Mazur, 2005).

Research shows that testosterone correlates positively with many behavioral aspects of dominance, such as high self-regard (Cashdan, 1995), self-reports questionnaires (Grant & France, 2001; Sellers et al., 2007), competition (Jones & Josephs, 2006; Josephs et al., 2006; Mehta et al., 2008), and social dominance (Cashdan, 1995; Tremblay et al., 1998). Basal testosterone also correlates with increased sensitivity to status-relevant information (Josephs, Newman, Brown, & Beer, 2003; Josephs et al., 2006; Newman, Sellers, & Josephs, 2005), even when that information is presented outside of conscious awareness (van Honk, Peper, & Schutter, 2005; Wirth & Schultheiss, 2007). The effects of testosterone are situationally dependent, manifesting when dominance is threatened (e.g., Maner, Miller, Schmidt, & Eckel, 2008; Mehta et al., 2008; van Honk et al., 2004; Wirth & Schultheiss, 2007) but not when threat is absent (Newman et al., 2005; Josephs et al., 2006).

It bears mentioning that although testosterone has been linked to aggression, this link is very tenuous (Archer, 2006; Archer, Birring, & Wu, 1998). Dominance and aggression are distinct entities; dominance is a trait-level motivation that drives a wide variety of

behaviors, and aggression is a specific behavior. In most non-human animals, dominance can be established via physical aggression, but generally speaking, human social norms preclude the use of physical aggression as an effective means to obtain high status. In certain circumscribed situations, human dominance may be manifested as physical aggression (e.g., prisons; Dabbs, Carr, Frady, & Riad, 1995), but generally dominance takes the form of more interpersonally nuanced social competitions, such as organized sport, academics, or elections (Mazur, 1973).

Dominant individuals utilize a variety of strategies to obtain high status, including aggression (Archer et al., 1998), coercion (Mazur & Booth, 1998), competition (Mehta et al., 2008), and affiliative engagement (Hawley, Little, & Pasupathi, 2002). As such, dominance can be expressed interpersonally in a number of ways. Postural expansion, or carrying oneself to maximize one's perceived size (Tiedens & Fragale, 2003), maintaining eye contact while speaking to another individual, but not maintaining eye contact while *listening* to another individual (Dovidio & Ellyson, 1982), and lower rates of smiling among women (Mast & Hall, 2004) are all behavioral markers of dominance. Dominant individuals also respond to perceived status threats with anger and aggression (Cohen, Nisbett, Bowdle, & Schwarz, 1996). High dominance has been characterized by actions such as spontaneously and eagerly engaging in interpersonal competition (Mehta & Josephs, 2006), establishing oneself as being in charge of a group (Lord, De Vader, & Alliger, 1986), projecting competence regardless of actual ability (Anderson & Kilduff, 2009), behaving assertively, and controlling the actions of others (Buss & Craik, 1980).

Depending on the types of behavioral and interpersonal strategies used, dominance can have a profound impact, both positive and negative, on social interactions and social behaviors. It is worth noting that individuals generally lack conscious insight into their own dominance levels, because dominance motivations are deeply rooted in physiological systems and generally exist outside of conscious awareness (van Honk et al., 2004). There is limited evidence that dominance behaviors can be predicted using self-reports (e.g., Swann & Hill, 1982), leaving salivary assessment of testosterone as the most accessible means to measure dominance while maintaining validity and predictive utility.

Overview of Testosterone Moderation

Despite considerable evidence for testosterone's connection to dominance, research on testosterone's effect on human social behavior has been frustratingly inconsistent. Although many studies have found an association between testosterone and behavior, many others have found weak or nonexistent effects. These null findings range from competitive behaviors (Gonza′lez-Bono, Salvador, Serrano, & Ricarte, 1999; Maner et al., 2008; Schultheiss et al., 2005) to aggression, both physical and nonphysical (Archer et al., 1998; Carré & McCormick, 2008; Carré, Putnam, & McCormick, 2009).

One explanation for these inconsistent findings is the presence of psychological moderators. For instance, research has found that the experience of simply winning or losing does not necessarily elicit expected fluctuations in testosterone. When an individual attributes their victory to external factors (e.g., luck), their response mimics that of losing (Gonza′lez-Bono et al., 1999). Additionally, a victory does not mean the same thing to an individual with no interest in exerting influence over others that it does to one motivated by interpersonal influence (Schultheiss et al., 2005). Psychological moderators such as cognitive appraisals (Gonza′lez-Bono et al., 1999), motivation, (Schultheiss et al., 2005), and social anxiety (Maner et al., 2008) all work to facilitate or suppress the relationship between situational forces, testosterone, and social behavior.

Recent research suggests yet another explanation for the inconsistencies in the literature. Testosterone's influence on behavior may be moderated not only by psychological processes, but by a number of other biological systems as well. For the purposes of the present review, it will help to breakdown testosterone's behavioral effects into two broad, overlapping categories. The first is testosterone's promotion of approach behaviors toward goals related to dominance and status. The second is the reactionary behaviors fueled by testosterone in response to social threats. These two categories do overlap (e.g., threats to established high status), but they will provide a helpful context for understanding testosterone's broad psychological influence. We will also discuss two classes of physiological mechanisms influencing testosterone's effects: mediators that promote testosterone's influence and moderators that suppress it. As more researchers include social endocrinology methods in their research, it is important to account for key physiological systems that, if ignored, could potentially wash out existing effects.

Mediated Promotion of Testosterone Effects

Vasopressin

Vasopressin is a peptide hormone produced in the central nervous system via androgen-dependent synthesis. Because of vasopressin's dependence on androgens, its effects are often sexually dimorphic; male behavior is more influenced by vasopressin than female behavior, which is more heavily influenced by oxytocin (Carter, 2007). Given that vasopressin synthesis is dependent on androgens, vasopressin and testosterone are closely linked in the types of social behaviors they influence. Specifically, vasopressin promotes testosterone-fueled behaviors by influencing social perceptions and facilitating preparation for defensive social aggression (Figure 1).

In a pair of studies (Thompson, George, Walton, Orr, & Benson, 2006; Thompson, Gupta, Miller, Mills, & Orr, 2004), administration of exogenous vasopressin was found to

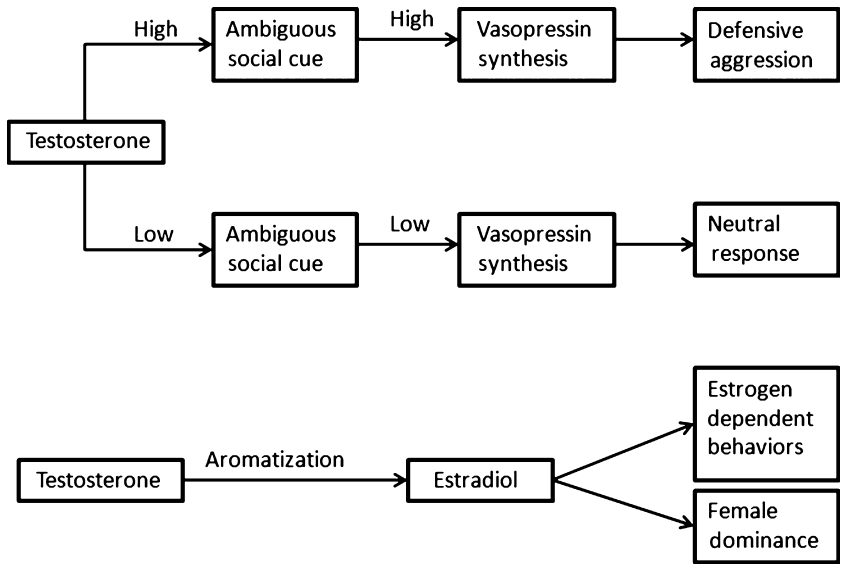

Figure 1 Facilitation of testosterone's behavioral effects (arrows are not meant to imply causation).

increase agonistic responses in men to emotionally neutral faces. The exogenous administration of vasopressin mimics endogenous elevations of vasopressin that would necessitate high levels of androgens for proper synthesis. It is worth noting that women were found to experience the opposite effect, showing increased affiliative responses (Thompson et al., 2006). The researchers argue that these sexually dimorphic responses reflect Taylor et al.'s (2000) 'tend and befriend' hypothesis. Namely, men respond to potential threats aggressively, but women respond affiliatively.

Estradiol

Estradiol is an estrogen, or feminizing steroid hormone, that can be metabolized from testosterone via aromatization. Research has shown that some of testosterone's effects are completely or partially mediated by properly aromatized estradiol (e.g., aggression: Trainor, Kyomen, & Marler, 2006; reproduction: Roselli, 2007; cognition: Janowsky, 2006). Additionally, most endocrine models of social behavior are derived from animal models that focus primarily on male behavior. The limited research on human dominance that has focused specifically on female behavior suggests a powerful role of estradiol in female dominance (Figure 1). Stanton and Schultheiss (2007) found that among women basal estradiol was positively correlated with implicit need for power, a personality trait closely related to dominance. Estradiol was also found to fluctuate in response to competitive outcomes, just as testosterone has been found to do in both men and women.

Moderated Suppression of Testosterone Effects

While vasopressin and estradiol both work to facilitate the behavioral approach effects of testosterone, there are a number of moderators that work to suppress testosterone's effect. Within this category of moderators, there are two basic effects that are disrupting testosterone's influence on behavior: threat response and impulse control.

Threat response

As seen with the effects of vasopressin, testosterone is often implicated in preparation for responding to a threat. Generally, testosterone prepares an individual under threat to respond defensively. There are two key moderators that control how testosterone-fueled threat response is manifested: cortisol and the serotonin transporter gene.

Cortisol. Cortisol, like testosterone, is another steroid hormone, and basal levels have been found to be positively, albeit weakly correlated with basal testosterone in the range of $r = 0.2$ to $r = 0.3$ (Mehta & Josephs, 2010). Cortisol is known primarily for its role in the physiological stress response. When an organism experiences stress, the hypothalamic–pituitary–adrenal axis (HPA-axis) is activated. The HPA-axis consists of the series of communications between the hypothalamus, the pituitary gland, and adrenal glands, culminating in the release of cortisol, as well as other hormones such as epinephrine and norepinephrine. The stress response causes physiological changes that include increased respiration, heart rate, blood pressure, and blood glucose levels. These changes are all intended to prime the organism to physically respond to stressors by facilitating fight or flight behaviors. Unlike epinephrine and norepinephrine, cortisol is able to pass through the blood–brain barrier, establishing itself as a primary influence over behavior during active stress responses. As such, cortisol has profound physiological *and* psychological effects.

Basal cortisol has also been linked to approach and avoidance behaviors, and much like testosterone, basal cortisol levels remain relatively static over time; test–retest stability has been found to be $r = 0.93$ and 0.73 for men and women, respectively, over 2 weeks (Liening et al., 2010). Approach and avoidance motivations are broadly defined as 'systems that motivate behavior toward desirable outcomes (approach) and away from undesirable outcomes (avoidance)' (Foster & Trimm, 2008, p. 1005). Essentially, high approach motivation is characterized by appetitive behaviors and sensitivity to reward. Avoidance motivation is characterized by avoidant behaviors and sensitivity to punishment. High levels of cortisol have been found to correspond to behavioral inhibition, shyness, and introversion, all avoidance behaviors (Kagan, Reznick, & Snidman, 1987; Kalin, Larson, Shelton, & Davidson, 1998a; Kalin, Shelton, Rickman, & Davidson, 1998b; Nunez, Ferre, Escorihuela, Tobena, & Fernandez-Teruel, 1996; Smider et al., 2002). Low levels of cortisol, on the other hand, have been found to correspond to more approach-motivated behaviors, such as extraversion and disinhibition (Shoal, Giancola, & Kirillova, 2003; Virgin & Sapolsky, 1997).

Recently, basal cortisol has been hypothesized to moderate testosterone's effect on behavior, suppressing the influence of testosterone (Figure 2; Mehta & Josephs, 2010; Popma et al., 2007). For instance, among 17- and 18- year-old offenders, high basal testosterone predicted aggressive behaviors, but *only* if basal cortisol was low (Dabbs, Jurkovic, & Frady, 1991). In a more recent study, basal cortisol and basal testosterone interacted to significantly predict individual differences in overt aggression among adolescent men, such that testosterone and aggression were related *only* when the individual also had low levels of cortisol. When cortisol was high, testosterone and aggression were unrelated (Popma et al., 2007). Much like the disinhibition model of impulsive psychopathy, high levels of avoidance motivation are manifested as inhibition and work to suppress behavior (Wallace, Malterer, & Newman, 2009). Testosterone will always fuel a

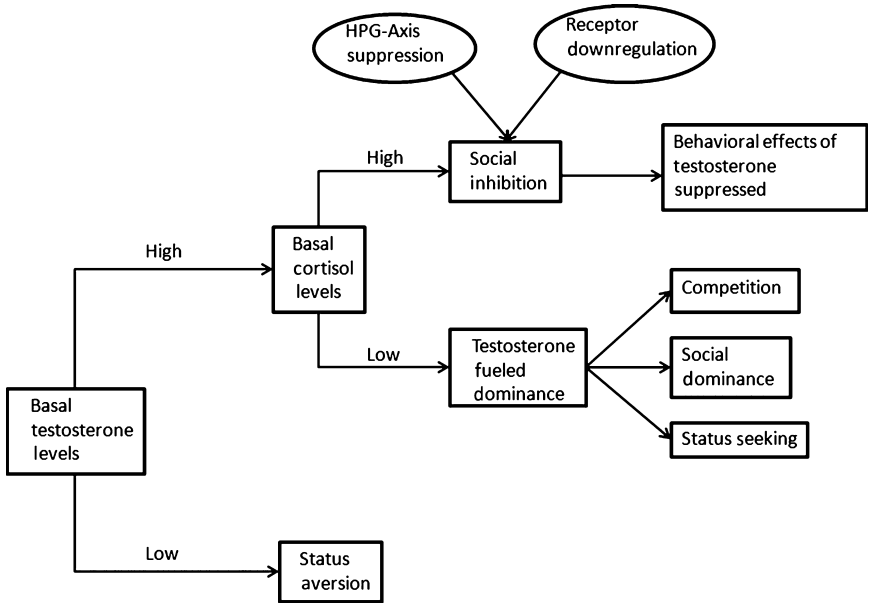

Figure 2 Cortisol's moderation of testosterone's effect on dominance (arrows are not meant to imply causation).

desire for status; but an individual also needs an approach-motivated behavioral style that only low levels of cortisol allow to act on that desire.

This moderation effect extends beyond aggressive behaviors within a population already at risk for physical aggression (i.e., delinquent adolescents). In a pair of recent studies, the effects of testosterone on competition and leadership were *only* expressed when basal cortisol was low (Mehta & Josephs, 2010). In the first study, when cortisol was low, basal testosterone predicted whether or not an individual would choose to continue competing or withdrawal from competition after having been defeated. When cortisol was high, the effects of testosterone following the defeat were not just suppressed but were reversed. In the study on leadership style, basal cortisol again moderated the effect of testosterone, such that when cortisol was low, high basal testosterone predict more dominant behavior as determined by independent raters. This effect disappeared among those with high basal cortisol.

Preliminary research on the physiological interaction of testosterone and cortisol has suggested two possible mechanisms for how cortisol is able to moderate the relationship between testosterone and behavior (see Figure 2). Just as cortisol is tied to the HPA-axis, testosterone is tied to the hypothalamic–pituitary–gonadal axis (HPG-axis), which cues the release of testosterone in preparation for reproductive behaviors. These two axes have an antagonistic relationship; that is, when one axis is activated, the other is suppressed (Viau, 2002). It is possible that the stress response is suppressing testosterone's effects until an organism's physiology returns to baseline. Research has also shown that cortisol, as well as other glucocorticoids, can have an impact on the expression of androgen receptors. Specifically, high levels of glucocorticoids downregulate receptors' expression (Burnstein, Maiorino, Dai, & Cameron, 1995; Smith, Syms, Nag, Lerner, & Norris, 1985; Welch & Diamond, 2001). Thus, high levels of cortisol might not affect testosterone levels directly, but rather the number of available androgen receptor sites.

Serotonin transporter gene (5-HTTLPR). Because cortisol is released by the HPA-axis in response to stressful events and stimuli, transient elevations in cortisol (but not basal measurements) are a common way to measure the degree to which an individual experiences stress (Dickerson & Kemeny, 2004). Previous work on cortisol reactivity has implicated serotonergic systems, specifically the polymorphic region of the serotonin transporter gene (5-HTTLPR), in moderating the response to stressful and anxiety-provoking experiences (Hariri & Holmes, 2006).

Recent research has found that the interaction of basal testosterone and allele length of 5-HTTLPR predicted cortisol reactivity when individuals were 'defeated' by an impossible experimental task (Rivers, Josephs, Beevers, & McGeary, 2010). It was found that among individuals with at least one short allele, there was a positive relationship between basal testosterone and cortisol reactivity, such that those individuals with high basal testosterone experienced higher levels of cortisol reactivity. The experience of being defeated by the task elicited the same negative reaction among high testosterone individuals as the experience of being defeated by an actual competitor, activating a general physiological mechanism that extends beyond testosterone and dominance.

Relative to those individuals with a pair of long 5-HTTLPR alleles, individuals with short alleles have been found to show exaggerated stress responses, hyperactivation of the amygdala in response to emotionally provocative stimuli, and a weaker functional coupling between the amygdala and the orbitofrontal cortex, suggesting weaker cortical control of emotion (Hariri & Holmes, 2006). Furthermore, fMRI research shows that these same neural circuits are activated in those individuals with high testosterone who

are exposed to emotionally threatening stimuli (van Wingen, Mattern, Verkes, Buitelaar, & Fernandez, 2010; van Wingen et al., 2009). Thus, it appears that short allele length on the 5-HTTLPR amplifies the effects of high testosterone, whereas long allele length suppresses it (see Figure 3).

Impulse control

Another means by which testosterone's effects are suppressed is through impulse control. Testosterone has been linked to a number of social approach behaviors (both desirable and undesirable), but there are neurological mechanisms that work to counteract testosterone and ultimately suppress its behavioral effects.

Monoamine oxidase A (MAO-A). Monoamine oxidase A (MAO-A) plays a crucial role in regulating the release of the neurotransmitters serotonin and norepinephrine, which play a crucial role in impulse control and the regulation of aggressive behavior (Buckholtz & Meyer-Lindenberg, 2008). MAO-A levels are regulated by the expression of the MAO-A promoter gene (MAOA-LPR). When this gene is underexpressed, MAO-A occurs in lower levels in the neural regions associated with impulse and aggression regulation (Sjöberg et al., 2008).

If human displays of dominance are constrained by social norms, that is dominance is typically expressed via socially nuanced behaviors rather than overt aggression, one would expect that a genotype associated with the underexpression of the MAO-A promoter gene to lead to lower levels of MAO-A, a lack of impulse control, and ultimately to testosterone-driven dominance manifesting itself as aggression (Figure 4). A study by Sjöberg et al. (2008) found this exact effect. Among men with diagnosed antisocial personality disorder (ASPD), a population at risk for physically aggressive behavior, testosterone was positively correlated with aggression, but *only* in those men with the low activity MAOA-LPR. On the other hand, there was no correlation between testosterone and

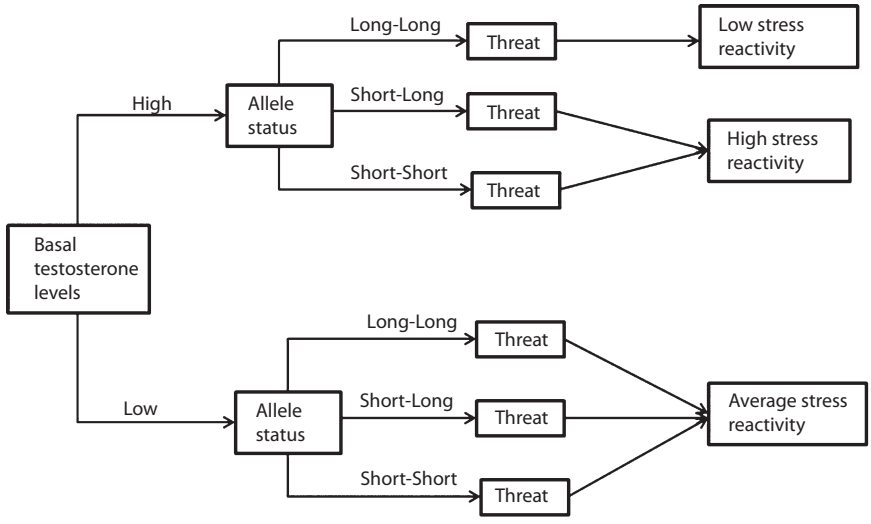

Figure 3 5-HTTLPR's moderation of testosterone's effect on threat response (arrows are not meant to imply causation).

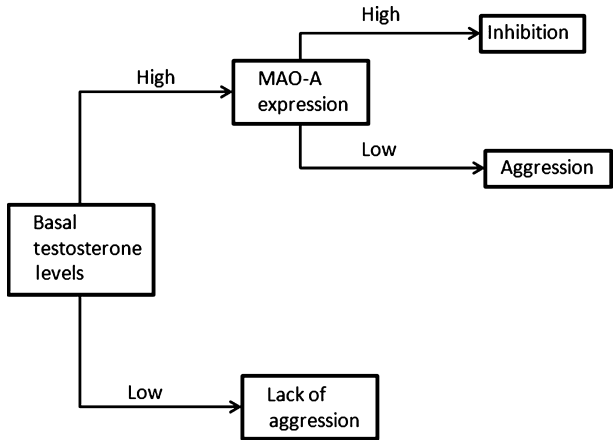

Figure 4 MAO-A's moderation of impulse control (arrows are not meant to imply causation).

aggression in men with ASPD and the high activity MAOA-LPR. It is worth noting, however, that neither testosterone nor the MAO-A gene (alone or interactively) predicted aggression in nonpathological controls not typically at risk for physical aggression.

Serotonin. Serotonin has been found to inhibit the effects of androgen-dependent dominance. Low levels of serotonin have been associated with decreased impulse control because of dysfunction in the prefrontal cortex, which increases the amount of impulsive aggression (Seo, Patrick, & Kennealy, 2008). In terms of testosterone-driven dominance behaviors, empirical research has found that across a variety of species, serotonin is negatively associated with aggression and social status (e.g., Bonson, Johnson, Fiorella, Rabin, & Winter, 1994; Larson & Summers, 2001). Additionally, if low levels of serotonin decrease self-control over testosterone-driven behaviors, it would follow that a combination of high testosterone-driven threat response and an underexpression of serotonin (i.e., short 5-HTTLPR) would lead to the increased cortisol reactivity outlined in the previous section.

Dopamine and norepinephrine. Dopamine and serotonin are both implicated in self-control, but their roles and relative levels are inversely related. Although serotonin inhibits impulsive behavior, dopamine facilitates aggression and social dominance by decreasing behavioral inhibition (van Erp & Miczek, 2000). Much like dopamine, norepinephrine also appears to facilitate dominance behaviors in social interactions (van der Vegt, Lieuwes, Cremers, de Boer, & Koolhass, 2003). Following dominance contests, dopamine and norepinephrine both increase in winners, suggesting a crucial role in the neural mechanisms that facilitate aggression. Thus, dysfunctional regulation of neurotransmitters responsible for impulse control (e.g., serotonin) and dominance-driven aggression (e.g., dopamine, norepinephrine) because of the underexpression of MAO-A gives rise to increased behavioral expression of testosterone-driven dominance.

Conclusions and Future Directions

Social endocrinology research has established that hormones play an important role in social behavior. Some social behaviors, such as dominance, are driven by deeply rooted,

nonconscious physiological mechanisms, making direct measurement of physiology preferable to traditional methods of psychological assessment, such as self-reports (van Honk et al., 2004). Yet physiological systems are often interdependent, and a single physiological measure in isolation may be inadequate for predicting behavior.

We have outlined a number of physiological mediators and moderators of testosterone's influence on behavior. Broadly, there are those elements that facilitate testosterone's effect on behavior, such as vasopressin and estradiol, as well as elements that work to suppress testosterone's behavioral effects, such as cortisol and serotonin. For the field of social endocrinology to progress, researchers must take into account these additional physiological elements. Studying only testosterone severely limits a researcher's ability to account for significant and sizable portions of variance in behavior. As previous work has demonstrated (Archer et al., 1998; Archer, Graham-Kevan, & Davies, 2005), the effects of testosterone on behavior can very easily be washed out when these factors are not accounted for by researchers.

Future researchers are strongly advised to consider this when conducting social endocrinology research. Although it may help to conceptualize basal hormone levels as a personality trait and hormonal fluctuations as transient mood states, researchers should not lose sight of the fact that hormones such as testosterone are merely one element (and in some cases a fairly proximal one) in a complex and dynamic series of physiological and neurobiological mechanisms that ultimately give rise to behavior. Some of the aforementioned physiological elements are more easily measured (e.g., cortisol, estradiol, 5-HTTLPR) than others (e.g., vasopressin, serotonin), but whether or not researchers choose to measure them, it is important to have an understanding of the dynamic processes between the input of hormones and the output of behavior.

Short Biographies

Scott H. Liening's research focuses on how individual differences interact with status differences to affect small groups' ability to cooperate effectively. He is primarily interested in how biological factors, specifically hormones such as testosterone and cortisol, influence behavior in cooperative situations that feature status differences among group members. This research incorporates social psychology, personality psychology, and social endocrinology. He received his B.A. in Psychology from the University of Michigan, and is in the process of obtaining his Ph.D. in Psychology from the University of Texas at Austin.

Robert A. Josephs is a product of the New York State public school system, remaining within that system from K through college (he received a B.A. in biological psychology from Cornell University, the largest of the Ivy's and the only 'hybrid' Ivy combining private and public colleges within one University). After graduating in 1983, he remained true to his public school roots, obtaining an M.S. in 1986 from the University of Washington under the supervision of Claude Steele. When Steele relocated to the University of Michigan in 1987, Josephs followed him to Ann Arbor, receiving the Ph.D. in social psychology in 1990. He was hired that same year by the Psychology Department at the University of Texas-Austin, and has been there ever since.

Endnote

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