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Exogenous Testosterone Rapidly Increases Aggressive Behavior in Dominant and Impulsive Men

Justin M. Carré, Shawn N. Geniole, Triana L. Ortiz, Brian M. Bird, Amber Videto, and Pierre L. Bonin

ABSTRACT

BACKGROUND: Although traditional wisdom suggests that baseline levels of testosterone (T) promote aggressive behavior, decades of research have produced findings that have been largely weak and inconsistent. However, more recent experimental work suggests that exogenous administration of T rapidly potentiates amygdala and hypothalamus responses to angry facial expressions. Notably, these brain regions are rich in androgen receptors and play a key role in modulating aggressive behavior in animal models.

METHODS: The present experiment extends this work by examining whether acutely increasing T potentiates aggressive behavior in men. In a double-blind, placebo-controlled, between-subject design, healthy adult men (n = 121) were administered either T or placebo, and subsequently engaged in a well-validated decision-making game that measures aggressive behavior in response to social provocation. In light of prior correlational research, we also assessed the extent to which T's effects on aggressive behavior would depend on variability in trait dominance and/or trait self-control.

RESULTS: Exogenous T on its own did not modulate aggressive behavior. However, T's effects on aggression were strongly influenced by variation in trait dominance and trait self-control. Specifically, T caused an increase in aggressive behavior, but only among men scoring relatively high in trait dominance or low in trait self-control.

CONCLUSIONS: These findings are the first to demonstrate that T can rapidly (within 60 minutes) potentiate aggressive behavior, but only among men with dominant or impulsive personality styles.

Keywords: Aggression, Competition, Hormones, Self-control, Testosterone, Trait dominance

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Research in animal models indicates that testosterone (T) plays an important role in modulating aggressive behavior (1). However, evidence for a role of T in promoting human aggression has been inconsistent (2). Importantly, T concentrations are not static, but rather fluctuate rapidly in the context of competitive interactions (3). It has been speculated that acute changes in T during competition may serve to fine-tune ongoing or future aggressive behavior (4–6). In support of this hypothesis, a rise in T after winning a competitive interaction is required to potentiate subsequent aggression in male California mice (7–9). Other research in male cichlid fish indicates that winning a competition increases one's probability of winning subsequent interactions—an effect that is eliminated when blocking the competition-induced rise in T (10). Complementing this work are studies in humans demonstrating that an acute rise in T concentrations during competition (but not baseline levels of T) predicts increased competitive motivation (11,12) and aggressive behavior (13–16). These findings are consistent with theoretical models suggesting that changes in T may serve to adaptively regulate ongoing or future dominance-related behavior (4,17). However, a major limitation of this research is that it is correlational, and thus the extent to which an acute increase in T plays a causal role in modulating competitive or aggressive behavior is not clear.

Pharmacological challenge research indicates that a single administration of T increases threat-related amygdala, hypothalamic, and periaqueductal gray reactivity to angry facial expressions in healthy men (18). These findings parallel evidence in women in which a single administration of T increases amygdala and hypothalamic reactivity to angry facial expressions (19–21). Notably, these subcortical brain structures are rich in both androgen and estrogen receptors (22–24) and play a key role in potentiating reactive aggression in animal models (1,25). More recently, a single application of T increased men's perception of their own physical dominance (26), suggesting that T may increase men's perception of their own formidability. Collectively, this research suggests that acutely increasing T concentrations rapidly modulates neural and psychological processes relevant to human aggression.

It has been proposed that social-contextual or individual difference factors may moderate the effect of T on human aggression (3,27). In particular, correlational and experimental

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work suggests that trait dominance may play a role in moderating relationships between T and human dominance behavior. People with dominant personality styles tend to behave in assertive, forceful, and self-assured ways (28) to achieve or maintain high social status. In one study, a rise in T after winning a competition predicted increased aggressive behavior in a subsequent task, but only among men scoring high in trait dominance (13). Also, baseline T concentrations were positively correlated with men's dominance behavior during a mate competition, but only for men scoring high in trait dominance (29). Finally, a single administration of T to women increased their competitive motivation after a victory, but only for those scoring high on trait dominance (30).

An individual's ability to exert self-control under affectively charged situations might also mitigate the effect of T on aggression. Some research indicates that individuals scoring high on trait-based measures of self-control are more efficient at inhibiting aggressive impulses during social provocation (31). Other research indicates that tasks designed to bolster self-control decreased participants' subsequent aggression, whereas those designed to disrupt or temporarily reduce self-control increased participants' subsequent aggression (32,33). According to one theoretical model of aggression (34), instigating triggers, such as provocation, and impelling forces, such as T, may promote aggressive impulses, but these impulses may not manifest behaviorally among individuals high in trait self-control because these individuals are better equipped to override such impulses. Thus, T's effects on aggression may be reduced among those high in self-control but pronounced among those low in self-control.

In this experiment, we employed a double-blind, placebo-controlled, between-subjects design to investigate the causal role of T in promoting aggression in healthy young men. We predicted that T would increase aggressive behavior. Also, in light of previous correlational and experimental work (13,29,30), we predicted that T's effects on aggressive behavior would be most robust among men scoring relatively high on trait dominance. Also, we predicted that exogenous T would have no effect on aggressive behavior for people with strong impulse control (i.e., elevated trait self-control), Instead, T would increase aggressive behavior among men with weak impulse control. Collectively, such findings would suggest that individual differences in trait dominance or trait self-control may confer differential sensitivity to the acute effects of T on men's aggressive behavior.

METHODS AND MATERIALS

Participants

Our sample consisted of 121 healthy men between the ages of 18 and 35 (mean age = 25.27 years, SD = 4.98 years). Subjects were recruited from advertising on local media sites, through medical research participant databases, and through local colleges and universities. Prior to enrollment in the study, each prospective participant was interviewed to determine his eligibility. Exclusion criteria for participants included the following: currently receiving prescription medication affecting hormone concentrations (e.g., glucocorticoids, androgens), current diagnosis of a psychiatric disorder, diagnosed heart condition, drug or alcohol dependency, and membership on a sports team or organization where T is a banned substance. Participants who qualified for the protocol consented to providing blood samples for hormonal assay, as well as to having their T levels temporarily manipulated. The study was approved by the Nipissing University Research Ethics Board. Participant ethnicities were self-reported as follows: 77.5% Caucasian, 13.1% First Nations, 4.1% Asian, 1.7% Latin American, and 3.3% other.

Procedure

Testing occurred in a single session (see Figure 1). Participants reported to the laboratory at one of two times, 10 AM or 1 PM. Upon arrival, participants completed informed consent. Next, participants completed a battery of online self-report questionnaires assessing basic demographic information and individual differences in personality (see later). After the completion of the online questionnaires, a phlebotomist drew 10 mL of participants' blood to assess hormone concentrations (see the Supplement for details on the assay). Participants were then randomly assigned to either the drug or the control group. Drug condition (AndroGel [AbbVie Inc.,

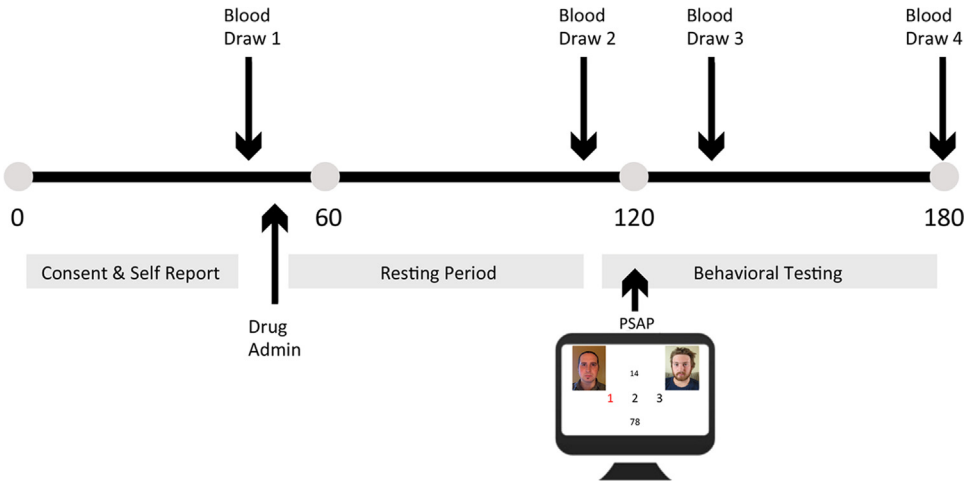

Figure 1. Experimental timeline for the entire protocol. PSAP, Point Subtraction Aggression Paradigm.