PSYCHOSOCIAL AND ENDOCRINE ANTECEDENTS OF RESPONSES TO SOCIAL-EVALUATIVE STRESS

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DISSERTATION ABSTRACT

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Title: Psychosocial and Endocrine Antecedents of Responses to Social-Evaluative Stress

Stress often precedes the onset of physical and mental health disorders, leading to costly and extended morbidity and even increased risk for death. I investigate psychosocial and endocrine precursors to stress responses, specifically examining the causal effects of status-relevant factors that modulate endocrine, affective, and behavioral responses to social-evaluative stressors. For example, while high social status reduces stress responses in numerous species, this stress-buffering effect of status may dissipate or even reverse during times of hierarchical instability. Similarly, some research links testosterone to reduced activity in stress responses systems, but correlational research indicates that higher testosterone is related to increased stress responses in threatening social situations. In each case, the causal influence of these psychosocial (status and hierarchy stability) and endocrine (testosterone) antecedents to stress responses was unclear.

Results from this work reveal that high status in a stable hierarchy buffered stress responses and improved behavioral responses to the stressor, but high status in an unstable hierarchy boosted stress responses and did not lead to better performance. This general pattern of effects was observed across endocrine (cortisol and testosterone), psychological (feeling in control), and behavioral (competence, dominance, and warmth)

responses to the stressor. Further, exogenous testosterone treatment caused increased motivated persistence – which can help persevere through stressful encounters – but, once exposed to a stressor, testosterone caused increased cortisol reactivity, increased negative affect, and decreased motivation in response to social-evaluative stress, especially for individuals high in trait dominance. The reduction in motivation following the stressor was mediated by negative affective responses to the stressor. This work provides evidence of the causal effects of psychosocial and endocrine factors on stress responses and demonstrates the importance of considering these status-relevant precursors when investigating stress within social contexts.

This dissertation includes previously published and unpublished coauthored material.

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In loving memory of:
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CHAPTER I

GENERAL INTRODUCTION

Stress is linked to the onset and duration of a litany of disorders, including coronary heart disease, heart attacks, and strokes (1); certain forms of cancer (2); infectious disease (3); and mental health conditions such as major depressive disorder, anxiety, and substance abuse (4,5). In response to a stressor – any real or imagined physical or psychological threat to an individual (6) – several biological, psychological, and behavioral stress response systems are activated to prepare the body to combat the stressful stimulus and otherwise avoid harm or death (7). But over the course of repeated stress or severe stressors, these same systems can have deleterious effects on physical and mental health (7-9). For example, cortisol, a hormone released from the hypothalamic-pituitary-adrenal axis (HPA axis), helps produce energy necessary for responding to a stressor in the short term but, over extended exposure, cortisol dysregulates immune function (10) and atrophies neural tissue in the central nervous system (11). As such, modulation of the stress response – so that the systems activate to respond to the stressful situation but do not expose an individual to caustic consequences – is an important aspect in determining the downstream effects of stress on health.

Psychosocial and biological systems play an important role in modulating stress response systems, and may therefore determine health and well-being by boosting or attenuating responses to stressors (12). For example, social hierarchies have been extensively studied in their ability to alter cortisol responses to stress, generally showing that high rankings are associated with attenuated cortisol responses to stress and consequently lead to better health than lower rankings (13). These relationships between

social standing, stress, and health have been linked to correlates of human social hierarchies like financial standing, job strain, and subjective experiences of one's position in a hierarchy (13-15). Yet few studies have tested the causal effects of the psychosocial (e.g., subjective social status within a hierarchy) and biological underpinnings (e.g., testosterone) of social hierarchies in order to examine the direction and causality of the relationships between social status and stress responses. This social endocrine approach can provide evidence of the causal impact of psychosocial and biological antecedents of the stress responses that then elucidates new routes by which these psychosocial and endocrine factors may impact health.

The purpose of this dissertation is to examine psychosocial and endocrine antecedents of an array of responses to social stress that have specific consequences for downstream health and well-being. Across three studies, I provide evidence for the effects of experimentally manipulated psychosocial (Chapter 2, social status and hierarchy stability) and endocrine (Chapters 4 and 6, exogenous testosterone) antecedents of the cortisol, testosterone, affect, and behavioral responses to social-evaluative stress. I will also examine the interactive effects of exogenous testosterone and trait dominance, an individual difference factor related to concern for status attainment (16), on stress reactivity (Chapter 4) and on motivated persistence behavior via a handgrip endurance task (Chapter 6).

This work contains previously published and co-authored material. Chapter 2 was previously published in concert with my advisor, Pranjal H. Mehta (17). Chapter 4 contains data and writing that was submitted for publication and was under review at the time of submission (18). My co-authors – Colton B. Christian, Pablo J. Morales, William

T. Harbaugh, Ulrich Mayr, and Pranjal H. Mehta – assisted with study design and data collection and provide edits and commentary on the document that was submitted for publication, although principle analyses and writing are my own. Chapter 6 is data from the same study as Chapter 4 and is associated with the same team of co-authors – Colton B. Christian, Pablo J. Morales, William T. Harbaugh, Ulrich Mayr, and Pranjal H. Mehta – but again analyses and writing are my own.

CHAPTER II

HIERARCHY STABILITY MODERATES THE IMPACT OF STATUS ON STRESS AND PERFORMANCE IN HUMANS

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Introduction

Social status is robustly linked with health outcomes in most human societies. Individuals with higher socioeconomic status live longer, experience increased well-being, and have lower rates of stress-related diseases such as cardiovascular conditions and Type-II diabetes (1,2). These health benefits may be explained in part by the stress-buffering effects of status. High status inhibits responses to acute stressors (3-6), which reduces physiological wear-and-tear and the likelihood of developing stress-linked diseases (2,7). In further support of the hypothesis that status buffers stress, attaining high rank in a hierarchy – such as a leadership position – is related to reduced concentrations of basal cortisol, a hormone released as part of the hypothalamic-pituitary-adrenal (HPA) axis in response to psychological stress (8,9). Despite a growing scientific consensus that high status is related to lower stress in humans, this previous research has focused primarily on stable hierarchies. During times of hierarchical instability when status could change, we propose that high status might boost – not buffer – stress responses. After all, the threat of losing a powerful high-ranking position and the need to defend it may be stressful. Correlational work in nonhuman primates provides initial support for this perspective. In one seminal study of olive baboons (*Papio anubis*), high-ranking males had lower basal cortisol levels compared to low-ranking males when the hierarchy was

stable. However, this effect reversed when the hierarchy was unstable; higher-ranking males had higher basal cortisol levels compared to lower-ranking males (10). Although this correlational evidence from primate research is promising, what we are deeming the *hierarchy instability hypothesis* – that an unstable hierarchy blocks or even reverses the effect of status on responses to acute stressors – is lacking a direct experimental test.

An experimental test of the hierarchy instability hypothesis in humans has public health implications because stress response systems such as the HPA axis impact immune function and overall health (2,7). Evidence in support of the hierarchy instability hypothesis could point to circumstances in which high status may lead to poor health and provide insight into the underlying mechanisms. Testing this hypothesis across multiple aspects of the stress response can further elucidate the consequences of acute stress responses for human behavior in both stable and unstable hierarchies, which to date remain largely unknown. Building on research in nonhuman primates, the present experiment tested the hierarchy instability hypothesis across key hormonal, psychological, and behavioral responses to a social-evaluative stressor.

We tested our hypothesis on cortisol responses to the stressor, but the hierarchy instability hypothesis may extend to testosterone as well. Testosterone is a sex hormone that is theorized to motivate concern for status (11). Thus, concentrations of this hormone may be especially likely to increase under conditions of status threat, such as when high status can be lost. In line with this theorizing, correlational research in nonhuman primates indicates that high-ranking positions in unstable hierarchies are associated with higher basal testosterone levels compared to low-ranking positions in unstable hierarchies, but higher rank is often unrelated to elevated basal testosterone levels in

stable hierarchies (12, 13; cf. 14). Building on this primate research and our hierarchy instability hypothesis, we propose that the threat of losing status for a high-ranking individual in an unstable hierarchy may intensify status-relevant stress and stimulate the desire to protect one's status, leading to elevated testosterone responses to the social-evaluative stressor. In contrast, a high-ranking position in a stable hierarchy may lower status-relevant stress because status cannot be lost and does not require protection, leading to buffered testosterone reactivity to the stressor. Testing the joint influences of status and hierarchy stability on cortisol and testosterone expands prior research on endocrine responses to social-evaluative stressors, which has primarily focused on cortisol as an index of stress and has paid surprisingly little attention to testosterone.

The hierarchy instability hypothesis may also predict behavioral responses to the stressor. Previous research has shown that priming high rank improves performance in social-evaluative situations such as mock job interviews, which leads to better outcomes (e.g., being hired for the job) (15,16). These positive social evaluations are influenced by status-relevant behaviors such as competence, dominance, and warmth (17,18). But again, the causal impact of status on performance in social-evaluative settings has only been tested in stable hierarchies. According to our hierarchy instability hypothesis, high status in a stable hierarchy should lead to positive performance evaluations compared to low status, but hierarchical instability should reduce or reverse these differences.

We also investigated the mechanisms through which status and hierarchy instability impact performance under stress. One likely psychological mechanism is through feeling in control. Powerful high-status positions are associated with greater feelings of control, and perceived control encourages status-relevant behaviors that boost

performance evaluations (19-22). We extend this work by testing whether hierarchy instability blocks the influence of status on performance via reduced feelings of control. In addition to testing this psychological mechanism, we also examined possible endocrine mechanisms. Prior research on acute cortisol responses and performance outcomes in stressful contexts has yielded mixed results (e.g., decision making performance: 23-25), but the consequences of acute testosterone responses for performance under social-evaluative stress have been largely overlooked. There is indirect evidence that elevated basal testosterone concentrations in status-threatening situations (e.g., losing a competition) predicts hyper-vigilance to status cues and impaired cognitive performance (26-28). Extending this prior research to the present study, we explored whether acute cortisol or testosterone responses to the stressor explained the effects of status and hierarchy instability on social-evaluative performance.

To address these open questions regarding status, hierarchy stability, and stress responses, the present study experimentally manipulated status (high or low) and hierarchy stability (stable or unstable) prior to a social-evaluative stressor in a 2 x 2 between-subjects design. We employed the Trier Social Stress Test (TSST), a widely adopted stressor in which participants deliver a speech in front of evaluators that is akin to stressful situations found in professional settings such as job interviews (29,15,16). Fig. 2.1 shows the timeline of the study design. Participants reported their affective states (e.g., feeling in control) before and after the stressor and provided saliva samples at four time points to measure cortisol and testosterone reactivity and recovery to baseline following the stressor. Independent observers without knowledge of the study hypotheses or experimental manipulations later watched the videotaped speeches and rated

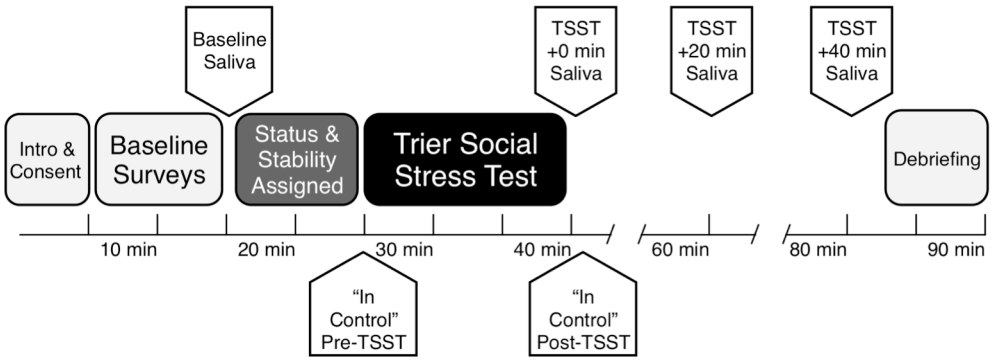

Fig. 2.1: Study timeline depicting experimental manipulations and measurement time of key variables.

participants on behavioral items that capture global performance evaluations (e.g., likelihood of hiring the candidate), competence, dominance, and warmth. We tested the hierarchy instability hypothesis across endocrine, psychological, and behavioral responses to the stressor. Finally, we conducted mediation analyses to investigate the mechanisms through which status and hierarchy stability influenced performance in the social-evaluative task.

Results

Preliminary analyses. For the analyses of endocrine change over time, cortisol and testosterone were natural-log-transformed to correct non-normal distributions; an arbitrary value of 10 was added to transformed cortisol values to ensure scores were positive for ease of interpretation (see Supporting Information). We did not expect differences in baseline hormone concentrations as a function of experimental group because the baseline saliva samples were taken prior to random assignment to experimental conditions. Consistent with this expectation, general linear model (GLM) analyses revealed no main effects or interactions between experimental conditions on baseline cortisol or testosterone concentrations (ps > .05, η^2s < .035). Descriptive

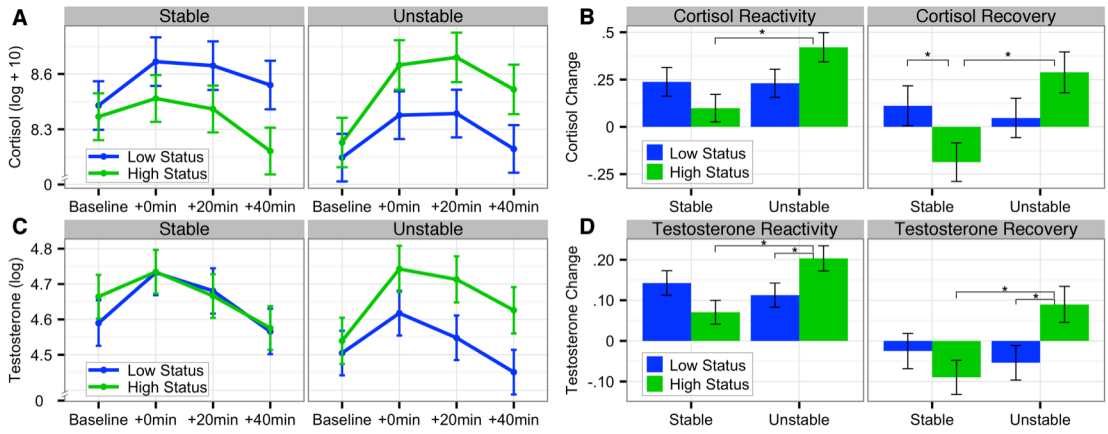

Fig. 2.2. Endocrine stress responses as a function of hierarchy stability and social status **Panel A**. Cortisol concentration (log-transformed plus arbitrary value of 10 added) at four time points: Baseline, +0, +20, and +40 minutes after Trier Social Stress Test (TSST). **Panel B**. Cortisol reactivity and recovery. **Panel C**. Testosterone concentration (log transformed) at four time points, controlling for sex. **Panel D**. Testosterone reactivity and recovery, controlling for sex. All values are estimated marginal means from relevant models; error bars represent standard errors of the means. * = significant uncorrected pairwise comparison at $p < .05$

statistics and conditional means for the main dependent variables are shown in Tables 2.1 and 2.2 (see Supporting Information).

Cortisol. To test the effects of status and hierarchy stability on cortisol responses to the stressor, we conducted a mixed-model GLM analysis with cortisol measurement time as a within-subject factor along with status and hierarchy stability as between-subjects factors. In agreement with our hierarchy instability hypothesis, there was a significant Status x Stability x Time interaction for cortisol ($F(1.82, 192.38) = 3.74$, $p = .029$, $\eta^2 = .034$)[1]. The overall pattern in Fig. 2.2 panel A suggests that higher status in a stable hierarchy buffered cortisol responses to the stressor – including blunted reactivity as well as declining cortisol concentrations during the recovery period. But higher status in an unstable hierarchy increased cortisol responses to the stressor – including enhanced

[1]All mixed-model GLMs for endocrine activity are reported with appropriate Huynh-Feldt corrections. See Supporting Information method section for details.

reactivity as well as sustained elevation of cortisol concentrations during the recovery period.

To confirm this interpretation, we conducted separate GLM analyses for cortisol reactivity and recovery to baseline. Cortisol reactivity was calculated by subtracting baseline cortisol concentrations from cortisol concentrations measured immediately after the stressor. Cortisol recovery to baseline was calculated by subtracting baseline cortisol concentrations from cortisol concentrations measured forty minutes after the stressor. A positive recovery score indicates that cortisol levels were elevated above baseline levels forty minutes following the stressor.

In support of the hierarchy instability hypothesis, there were Status x Stability interactions on both cortisol reactivity ($F(1,106) = 4.82$, $p = .030$, $\eta^2 = .044$) and recovery ($F(1,106) = 6.58$, $p = .012$, $\eta^2 = .058$). As shown in Fig. 2.2 panel B, high-status individuals in an unstable hierarchy exhibited increased cortisol reactivity ($F(1,53) = 8.70$, $p = .005$, $\eta^2 = .141$) and increased cortisol recovery levels (i.e., recovery cortisol levels that remained above baseline; $F(1,53) = 10.56$, $p = .002$, $\eta^2 = .166$) compared to high-status individuals in a stable hierarchy. Low-status individuals in stable versus unstable hierarchies did not differ in their cortisol reactivity ($F(1,53) = 0.01$, $p = .94$, $\eta^2 < .001$) or recovery ($F(1,53) = 0.18$, $p = .673$, $\eta^2 = .003$). Consistent with theories proposing that high status should buffer stress responses in stable hierarchies, high status in a stable hierarchy also significantly reduced cortisol recovery levels compared to low status in a stable hierarchy ($F(1,54) = 4.90$, $p = .031$, $\eta^2 = .083$).

Taken together, these results provide direct empirical support for the hierarchy instability hypothesis across multiple indices of cortisol change. High-status individuals

in a stable hierarchy showed blunted cortisol reactivity to the stressor and declining cortisol concentrations during the recovery period. In contrast, high-status individuals in an unstable hierarchy showed increased cortisol reactivity to the stressor and cortisol concentrations that remained elevated over baseline levels during recovery.

Testosterone. To test the effects of status and hierarchy stability on testosterone responses, we conducted a mixed-model GLM analysis with testosterone measurement time as a within-subject factor, status and hierarchy stability as between-subjects factors, and participant sex as a covariate. This analysis revealed a significant Status x Stability x Time interaction for testosterone ($F(2.52, 264.70) = 4.42$, $p = .008$, $\eta^2 = .040$; see Fig 2.2 panel C). To interpret this interaction, we conducted follow-up GLM analyses on testosterone reactivity and recovery, calculated in the same fashion as the cortisol indices. Status x Stability interactions were found for both testosterone reactivity ($F(1,105) = 7.37$, $p = .008$, $\eta^2 = .066$) and recovery ($F(1,105) = 5.88$, $p = .017$, $\eta^2 = .053$). As shown in Fig. 2.2 Panel D, high status in an unstable hierarchy led to increased testosterone reactivity ($F(1,52) = 10.10$, $p = .002$, $\eta^2 = .163$) and increased testosterone recovery levels ($F(1,52) = 8.11$, $p = .006$, $\eta^2 = .135$) compared to high status in a stable hierarchy. Low-status individuals in stable versus unstable hierarchies did not differ in testosterone reactivity ($F(1,52) = 0.46$, $p = .502$, $\eta^2 = .009$) or recovery ($F(1,52) = 0.219$, $p = .642$, $\eta^2 = .004$).

High-status individuals in an unstable hierarchy also showed increased testosterone reactivity ($F(1,51) = 4.38$, $p = .041$, $\eta^2 = .079$) and increased testosterone recovery levels ($F(1,51) = 5.60$, $p = .022$, $\eta^2 = .099$) compared to low-status individuals in an unstable hierarchy. Collectively, these results generally align with the cortisol

results and suggest that our hierarchy instability hypothesis applies not only to cortisol but to testosterone fluctuations in social-evaluative contexts as well.

Further analyses revealed that the interactions between status and hierarchy stability on endocrine responses (i) showed similar patterns when we adopted alternative strategies for analyzing cortisol and testosterone reactivity (Supporting Information, Tables 2.3 and 2.4, Fig. 2.5 and 2.6) as well as cortisol recovery (Supporting Information, Fig. 2.7); (ii) did not statistically differ between male and female participants (Supporting Information, Table 2.5); and (iii) were robust to additional covariates and to bootstrap bias correction (Supporting Information, Tables 2.6-2.8).

Feeling in Control. To test if our experimental manipulations influenced feeling in control, we conducted a mixed-model GLM analysis with time of measurement as a within-subjects factor along with Status and Hierarchy Stability as between-subjects factors. There was a non-significant Status x Stability x Time interaction ($F(1,103) = 0.001$, $p = .979$, $\eta^2 < .001$), but there was a statistically significant Status x Stability interaction in support of the hierarchy instability hypothesis ($F(1,103) = 4.72$, $p = .032$, $\eta^2 = .044$). Thus, our experimental manipulations modulated feeling in control starting after assignment to experimental conditions and remained after the stressor as well. To interpret the interaction, we averaged feeling in control scores measured before and after the stressor. As shown in Fig. 2.3 Panel A, high status boosted feeling in control scores compared to low status in the stable hierarchy ($F(1,53) = 9.45$, $p = .003$, $\eta^2 = .151$), but high and low status participants were indistinguishable in their feelings of control in the unstable hierarchy ($F(1,50) = 0.047$, $p = .830$, $\eta^2 = .001$). High-status individuals in a stable hierarchy also reported feeling more in control compared to high-status individuals

in an unstable hierarchy ($F(1,52) = 5.47$, $p = .023$, $\eta^2 = .095$). Supplementary analyses revealed that status and hierarchy stability had non-significant effects on global measures of positive and negative affect (Supporting Information, Fig. 2.8). This pattern of results suggests that status and hierarchy stability more robustly influence feeling in control compared to general positive and negative affect, which is consistent with theory linking perceived control to power and status (22).

Behavior During the Social-Evaluative Stressor. Videos of participants' speeches were rated on items that capture performance evaluations (e.g., Would you hire this individual?), competence, dominance, and warmth. Factor analysis indicated that three factors satisfactorily fit the data (Supporting Information, Table 2.9). In line with prior research indicating that appearing competent is a key driver of hiring decisions (17), performance ratings loaded onto the same factor as the competence items; two additional factors emerged for dominance and warmth. Subsequent analyses focused on interview performance (consisting of items that assess competence and performance), dominance,

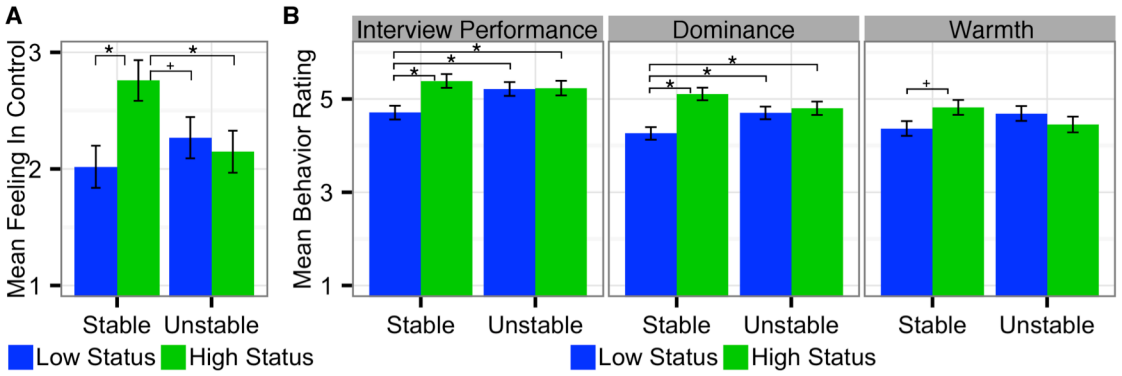

Figure 2.3. **Panel A.** Self-reported feelings of control (average of pre- and post-stress measures) as a function of social status and hierarchy stability. **Panel B.** Observed behavior during job interview speech as a function of social status and hierarchy stability. Values are estimated marginal means and error bars represent standard errors of the means. * = significant uncorrected pairwise comparison at $p < .05$; + = $p < .06$

and warmth; models included sex as a covariate to account for potential sex differences in status-relevant behaviors (30).

In agreement with the hierarchy instability hypothesis, there was a significant Status x Stability interaction on interview performance ($F(1,104) = 4.86$, $p = .030$, $\eta^2 = .045$) (see Fig. 2.3 Panel B). In a stable hierarchy, high-status individuals performed better compared to low-status individuals ($F(1,53) = 9.86$, $p < .003$, $\eta^2 = .157$). But in an unstable hierarchy, high and low status individuals performed equivalently ($F(1,50) = 0.01$, $p = .924$, $\eta^2 < .001$). Status x Stability interactions were found for dominance ($F(1,104) = 7.42$, $p = .008$, $\eta^2 = .067$) and warmth ($F(1,104) = 4.56$, $p = .035$, $\eta^2 = .042$) in the same direction as the effects on performance. High-status individuals in a stable hierarchy exhibited greater dominance ($F(1,53) = 23.08$, $p < .001$, $\eta^2 = .303$) and warmth ($F(1,53) = 3.97$, $p = .051$, $\eta^2 = .070$) compared to low-status individuals in a stable hierarchy. In an unstable hierarchy, there were non-significant differences between high and low status individuals in dominance and warmth ($ps > .32$, η^2s $<.02$).

Follow-up tests revealed that these interactions were driven by low-status participants, who showed better interview performance and increased dominance in the unstable compared to the stable hierarchy ($ps < .029$, $\eta^2 s > .087$; Fig. 2.3B). Overall, this pattern of results extends previous work in which low status in unstable hierarchies increases approach-oriented behaviors such as dominance compared to low status in stable hierarchies (19-21) and suggests further that perceiving a hierarchy as unstable may improve low-status individuals' performance in real-world social evaluations.

The interactions between status and hierarchy stability on feeling in control and behavioral responses to stress showed the same patterns with alternative analytical

approaches (Supporting Information, Table 2.8 and 2.10), and did not statistically differ between male and female participants, with the exception of dominance. For dominance, the joint impact of social status and hierarchy instability, although evident in both sexes, was stronger in men than in women (Supporting Information, Table 2.5).

Mediation Analyses. Next we conducted mediation analyses to investigate the mechanisms through which status and hierarchy stability influenced interview performance. The *PROCESS* macro (v.2.15; 31) was used to determine if the Status x Stability interaction on interview performance was mediated by feeling in control or indices of endocrine reactivity, controlling for sex (see Supporting Information for statistical analysis details, Table 2.11 for partial correlations that control for sex). These

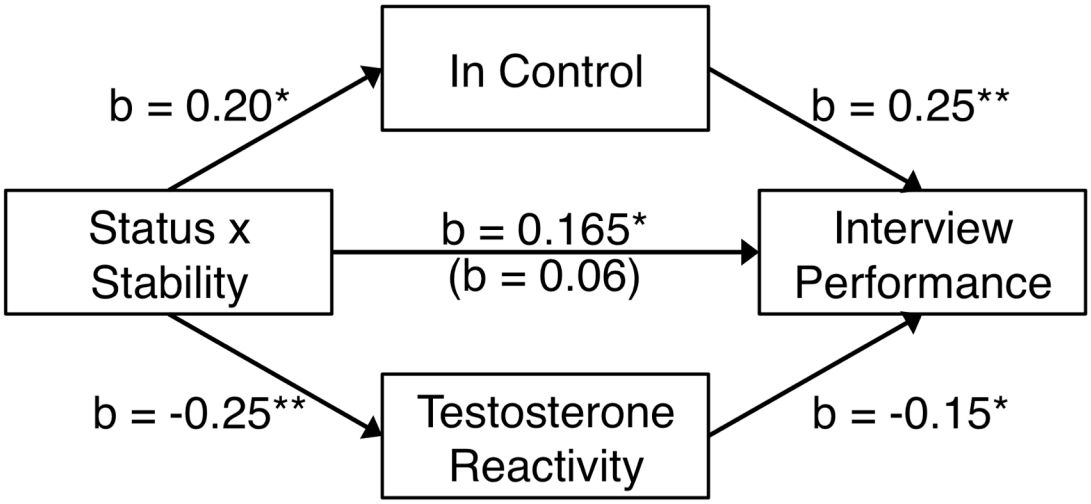

Figure 2.4. Moderated mediation model showing the indirect, interactive effects of Status x Stability on interview performance via feeling in control and testosterone reactivity. Pathway estimates are reported in unstandardized units (31). Sex is a covariate in the model. The model also includes pathways for the main effects of social status and hierarchical instability on the mediators, but these pathways were excluded from these figures. Conditional indirect effects are shown in Supporting Information, Table 2.12. **p < .01 *p <. 05

mediation analyses revealed significant moderated mediations for interview performance via sense of control ($\omega = 0.114$, 95%CI [0.023, 0.275]) and testosterone reactivity ($\omega = 0.087$, 95%CI [0.011, 0.226]) but not cortisol reactivity ($\omega = -0.005$, 95%CI [-0.092, 0.067]; see Supporting Information Table S12 for conditional indirect effects). We tested another model that included both feeling in control and testosterone reactivity to examine if these two factors were independent mediators. As shown in Fig. 2.4, the results suggest that social status and hierarchical instability impacted interview performance through two independent pathways: (i) status and hierarchy stability jointly influenced feeling in control, which predicted better interview performance; and (ii) status and stability interacted to influence testosterone reactivity, which predicted decreased interview performance.[2]

Discussion

The present experiment is the first to test the joint influences of social status and hierarchical stability on endocrine, psychological, and behavioral responses to a social-evaluative stressor. Consistent with the hierarchy instability hypothesis, high status buffered stress responses and improved interview performance in a stable hierarchy, but high status boosted stress responses and carried no performance advantage in an unstable hierarchy. This general pattern was observed across hormonal (cortisol and testosterone), psychological (feelings of control), and behavioral (interview performance, dominance, and warmth) responses to the social-evaluative stressor.

[2]Mediation analyses for dominance and warmth factors are reported in the Supporting Information.

Follow-up mediation analyses suggest that status and hierarchy stability jointly impacted overall interview performance through two independent pathways. First, status and hierarchy stability interactively influenced feeling in control, which was positively related to performance evaluations. This result expands psychological theory of stable hierarchies by revealing that hierarchical instability disrupts the impact of status on behavior via feelings of control (22). Second, status and hierarchical stability interactively influenced testosterone reactivity, which negatively predicted interview performance. This biological pathway extends prior research in which higher basal testosterone levels were related to status-seeking motivation and impaired cognitive performance under conditions of experimentally induced status threat (e.g., defeat in competition; 26-28). Elevated testosterone reactivity in the present study may have led individuals to focus on their threatened status rather than the speech task at hand, disrupting cognitive functioning when delivering the speech and undermining performance evaluations. This testosterone pathway is especially noteworthy because most prior studies on social stressors such as the TSST measure cortisol but rarely measure testosterone responses (32). The current study is the first to demonstrate that the joint influence of status and hierarchy stability on performance is mediated by testosterone responses, but follow-up research is needed to confirm this effect and to specify the underlying mechanisms.

These findings provide direct causal support for the hierarchy instability hypothesis and have applications for devising interventions aimed at reducing stress and improving performance. According to the present results, psychological interventions that alter beliefs about the hierarchy or that use role-playing exercises may improve overall

performance in social-evaluative situations such as job interviews. For example, a low-status individual who "knows" her place in society — that is, who perceives the status hierarchy as stable — may appear less competent in a job interview, reducing her chances of being hired. But merely holding the *belief* that she can rise in the hierarchy — that is, believing that the hierarchy is unstable — may lead to behaviors that signal competence and improve her chances of being hired. The present results also suggest that imagining or acting out a high-status role in a stable hierarchy prior to a real-world stressor such as an interview may reduce endocrine stress responses, increase feelings of control, and improve performance. We look forward to follow-up research that builds upon the present findings to test the efficacy of such hierarchy-relevant psychological interventions.

The current results also inform research on status and health. Correlational studies reveal positive associations between societal-level indicators of status, such as socioeconomic status, and better health outcomes (1,2,4). Dysregulation of stress response systems is theorized to be a mechanism through which lower status confers health risk (1,2,4,5,9), potentially through the joint effects of testosterone and cortisol responses on the immune system (33). However, research on status and human health has generally failed to consider the extent to which the stability of the social hierarchy might alter the relationship between status and health (34; but see 35 for some evidence). According to the hierarchy instability hypothesis, the link between lower status and poorer health may hold only in stable status hierarchies. In unstable hierarchies, *higher* status individuals may show dysregulated stress response systems and worse health outcomes. It should be noted, however, that a single, robust endocrine reaction to a

stressor is not inherently unhealthy. After all, glucocorticoids such as cortisol mobilize energy as part of a healthy response to stress (7). But when these endocrine responses are persistent and repeated over an extended period of time, they may be detrimental to health and well-being. Thus, it will be important to conduct follow-up longitudinal studies in humans in which features of the hierarchy, endocrine stress responses, and health outcomes are tracked over longer periods of time.

We experimentally manipulated social status in the present study, but our manipulation also contained aspects of social power. Status, which is also referred to as prestige, can be defined as social standing that is granted to individuals for superior skills, success, or knowledge (18). Power is defined as asymmetrical control over resources and tends to be positively correlated with status in real-world hierarchies (36,37). In line with other experimental designs (21,22), our manipulation therefore included features of social status and power in order to emulate real-world hierarchies. The few studies to date that differentiated power and status suggest that they sometimes lead to different outcomes; for instance, status often promotes – whereas power reduces – justice toward others (38). But both power and status are plausible explanations for the interactions between social rank and stability seen in the present study. For example, unstable high-ranking positions lead to behaviors aimed at protecting one's high rank through social motives closely linked to power (39). Yet other evidence suggests that losing a prestigious high-status position is more aversive than losing a powerful position because status is more closely related to an individual's self-concept (40). Additional research will be needed to clarify the extent to which social status and power contribute to the influence of hierarchical

rank on acute stress responses and social-evaluative performance in stable and unstable hierarchies.

We provide initial evidence suggesting that status and hierarchy stability influence behavior via acute testosterone reactivity to the stressor. This proposed causal pathway is consistent with rapid, non-genomic effects of steroid hormones on neural functioning and behavior that occur over the course of minutes or seconds (41). But our study design measured naturally occurring hormonal and behavioral stress responses, precluding us from making strong claims about causal direction. It is plausible that the causal direction goes the other way as well, from behavior to hormone changes, which is consistent with theorizing that hormones and behavior influence each other in reciprocal feedback loops (11). Future research can provide greater insight into causality by pharmacologically inhibiting or increasing testosterone concentrations during social-evaluative stressors.

This study measured salivary hormone concentrations with enzyme immunoassay (EIA), a common technique due to its convenience and cost effectiveness. Methods like liquid chromatography tandem mass spectrometry (LC-MS/MS) are thought to provide more valid measurements compared to EIAs, but the logistical and financial requirements of LC-MS/MS methods have limited their widespread use. Prior research indicates high correspondence between EIAs and LC-MS/MS for salivary cortisol but only moderate correspondence for salivary testosterone (42-44). This moderate correspondence is likely due to known sources of measurement error in EIAs, such as cross-reactivity, particularly in the low range of measurement (e.g., testosterone levels in women; 43). These sources of measurement error likely obscure relationships that exist rather than promote

relationships that do not exist (43). Hence, we suspect that the hormonal evidence for the hierarchy instability hypothesis found in the present experiment will be stronger in future LC-MS/MS studies. We look forward to replications that adopt LC-MS/MS methods.

In conclusion, this experiment provides evidence that the influence of status on stress responses and performance depends on the stability of the hierarchy. This knowledge has applications in domains such as business, education, politics, the arts, and medicine. For example, the results can inform hierarchy-based interventions for improving performance in social-evaluative contexts as job interviews, presentations, auditions, and political debates. Because stress is a risk factor for disease and poor well-being (1,2), the findings also have implications for the influence of hierarchy on health.

Materials and Methods

We briefly report methods here and describe full methods and statistical analysis details in the Supporting Information. We tested our predictions by experimentally manipulating social status and hierarchy stability in undergraduate participants (n = 118; 57.3% female; Age: M = 19.8) who were recruited for course credit. Participants were told that, based on their responses to pre-laboratory questionnaires, they had been assigned to complete an upcoming puzzle-building task as either a "manager" (high status) or "builder" (low status) and that another participant (actually a confederate) would perform the unassigned role (20,22). Participants were told specifically that the assignment was based on their "leadership skills and experience" in order to connect the role assignment to prestige (18). In reality, roles were randomly assigned. Participants were also told that the manager would be in charge of directing subordinates in the

building process and would evaluate the "builder" at the end of the task to determine how to split bonus money.

Next all participants were asked to complete the TSST, a five-minute speech about one's qualification for a job and a five-minute serial subtraction math task, in front of a panel of observers. In order to manipulate hierarchy stability, participants were told that their role (manager/builder) could change based on the speech/math task (unstable hierarchy) or that their performance on the task would not affect their role assignment (stable hierarchy). A five-minute preparation period was completed in the presence of a gender-matched confederate in order to increase the salience of the manipulations. Panelists and confederates were blind to participants' assigned conditions. Participants provided informed consent to participate in a group activity and perform a speech task. The University of Oregon's Institutional Review Board approved all methods.

Hormones were assayed from saliva collected via passive drool approximately 10 minutes after arriving at the laboratory (baseline), as well as 0, 20, and 40 minutes after the TSST. Participants responded to a prompt asking how "in control" they felt after assignment to status and stability conditions and after the TSST, which was included as a separate item in a broader measure of self-reported affect. Three independent observers rated videos of each participant's speech for status-relevant behaviors and two items that assessed overall interview performance (Supporting Information, Table 2.9).

Missing Data and Outliers. Three participants did not complete the social stress task, and four did not correctly identify the manager or builder role to which they were assigned, which left 111 participants for the main analyses. One participant did not produce enough saliva to assay, leaving 110 participants for hormone analyses. The

remaining hormone data were examined for outliers. One cortisol value and three testosterone values were Winsorized to 3 SD above the means of each offending sample's time point's mean. Two participants' videos were not recorded due to technical difficulties, leaving 109 participants for behavioral analyses.

Manipulation checks. Participants completed manipulation check items ("How do you perceive the status of your role compared to the other role?" and "Do you think your position might change?") and were asked to describe which role they were assigned. Participants assigned to the manager role perceived their role as higher status compared to participants assigned to the subordinate role ($F(1,105)=35.6$, $p<.001$, $\eta^2= .18$). Participants in the unstable hierarchy were more likely to report that their role could change compared to participants in the stable hierarchy (χ^2 (1) = 8.32, p = .004, *Cramer's* V = .276).

Supporting Information

Open Data and Materials

Data and materials for this publication can be found online at: https://osf.io/js2x4

Supporting Materials and Methods

Participants. The ethnic breakdown of participants in the study was approximately 70% European-American, 13% Asian or Asian-American, 7% Hispanic/Latino, 5% Pacific Islander, and 3% or less African-American, Middle Eastern, and Native American. Sample size was estimated *a priori* via power analysis with G*Power3 (45), which assumed four groups with four repeated measures, power ß= 0.80, a small effect size F = 0.15, α = .05, correlation among repeated measures = 0.65, and non-sphericity correction = 0.75.

	Full Sample	*Males*	*Females*
	Cortisol (µg/dL)		
Baseline	0.244 (.020)	0.232 (.022)	0.252 (.031)
TSST +0’	0.322 (.023)	0.333 (.032)	0.313 (.033)
TSST +20’	0.342 (.028)	0.39 (.043)	0.307 (.037)
TSST +40’	0.284 (.024)	0.313 (.039)	0.262 (.031)
Reactivity[a]	0.243 (.039)	0.32 (.064)	0.185 (.046)
Recovery[b]	0.058 (.054)	0.19 (.081)	-0.041 (.071)
	Testosterone (pg/ml)		
Baseline	112.8 (6.19)	171.3 (8.36)	69.1 (2.64)
TSST+0’	129.0 (7.05)	195.3 (9.48)	79.6 (3.28)
TSST+20’	124.0 (7.18)	192.0 (9.65)	73.3 (3.07)
TSST+40’	113.1 (6.45)	175.9 (8.20)	66.2 (2.75)
Reactivity[a]	0.13 (.015)	0.128 (.023)	0.132 (.021)
Recovery[b]	-0.023 (.022)	0.019 (.028)	-0.054 (.033)
	Pre-stress Self-report		
In Control	2.37 (.10)	2.77 (.16)	2.08 (.11)
Positive Affect	2.27 (.08)	2.57 (.11)	2.04 (.09)
Negative Affect	2.06 (.07)	2.02 (.11)	2.09 (.08)
	Post-stress Self-report		
In Control	2.25 (.10)	2.78 (.17)	1.85 (.11)
Positive Affect	2.04 (.07)	2.33 (.11)	1.83 (.09)
Negative Affect	2.09 (.08)	2.05 (.12)	2.12 (.10)
	Behavior During Stressor		
Interview Performance	5.15 (.08)	5.00 (.14)	5.26 (.09)
Dominance	4.71 (.07)	4.77 (.11)	4.66 (.10)
Warmth	4.61 (.08)	4.40 (.14)	4.77 (.09)

Table 2.1. Mean (SE) for main study variables. [a]Reactivity is log-transformed hormone concentrations immediately after the social-evaluative stressor minus baseline concentrations (i.e., log[TSST+0] – log[baseline]). [b]Recovery is log-transformed hormone concentrations forty minutes after the social-evaluative stressor minus baseline concentrations (i.e., log[TSST+40] – log[baseline]).

Procedure

Pre-Lab and Arrival. Prior to arriving at the laboratory session, participants responded to personality questionnaires online, which were used as part of the status manipulation. Participants were instructed to abstain from eating, drinking, exercising, and smoking for two hours before their scheduled experimental session. To account for diurnal variability in endocrine activity, all sessions occurred in the afternoon between 1300 and 1730 hrs. After arriving at the laboratory, participants were seated in an individual testing room where informed consent was obtained to participate in a group activity and perform a speech task. Saliva-sampling and demographic questionnaires were administered for approximately 10 minutes before baseline saliva was collected via passive drool.

Status Manipulation. Participants were then told that based on their responses to

	Stable Hierarchy		*Unstable Hierarchy*	
	High Status	*Low Status*	*High Status*	*Low Status*
		Cortisol		
Reactivity	.10 (.07)	.24 (.07)	.42 (.09)	.23 (.07)
Recovery	-.19 (.09)	.11 (.10)	.29 (.12)	.05 (.11)
		Testosterone		
Reactivity	.07 (.03)	.14 (.03)	.20 (.03)	.11 (.03)
Recovery	-.09 (.04)	-.02 (.04)	.09 (.04)	-.05 (.04)
		Affect and Behavior		
Feeling in control	2.76 (.17)	2.02 (.16)	2.17 (.17)	2.27 (.21)
Interview Performance	5.40 (.14)	4.72 (.16)	5.26 (.14)	5.23 (.15)
Dominance	5.10 (.12)	4.26 (.13)	4.79 (.15)	4.69 (.14)
Warmth	4.83 (.15)	4.28 (.18)	4.49 (.19)	4.72 (.14)

Table 2.2. Conditional means (SEs) for main dependent variables.

pre-laboratory questionnaires, they had been assigned to complete an upcoming puzzle task as either the "manager" (high status) or "builder" (low status) while another participant (actually a confederate) would perform the unassigned role. Participants were told specifically that the assignment was based on their "leadership skills and experience" in order to connect this manipulation to prestige (expertise, skills). In actuality, status was randomly assigned and there was no puzzle task. All participants were told that the participant in the role of manager would be in charge of directing subordinates, would decide how to structure the process for building the tasks, and would evaluate the "builder" at the end of the task in order to determine how to split $10 of bonus money.

Stability Manipulation and Social-Evaluative Stressor. Next, all participants were asked to complete a "speech task in front of a panel of observers" who were "trained in behavioral observation and social competency" in order to "see how [the participant] interact[s] with others." This task is actually the TSST, a well-validated social-evaluative stressor that involves delivering a five-minute speech about one's qualifications for one's ideal job and doing five minutes of serial subtraction in front of two evaluators. The panel of evaluators consisted of a college-aged man and woman (i.e., approximately the same age range as the participants) who were trained to maintain neutral facial expressions and generally be non-reactive. Participants were told that their role (manager/builder) could change based on the speech/math task (unstable hierarchy) or that their performance on the task will not affect their role assignment (stable hierarchy). A five-minute preparation period (but not the speaking portion) was completed in the presence of a gender-matched confederate in order to increase the

salience of the manipulations. Panelists and confederates were blind to participants' randomly assigned conditions.

Following completion of the TSST, the participants then recovered for forty minutes while filling out additional demographic questionnaires and performing unrelated tasks not included in the present report. Subsequent saliva samples were collected at 0, 20, and 40 minutes post-TSST for a total of four saliva samples, including baseline.

Affective States. After assignment to status and stability conditions and after the TSST, participants responded to a prompt asking how "in control" they felt, which was included as a separate item in a broader measure of self-reported affect. This item was analyzed separately using GLMs because theory suggests that status and hierarchy stability may influence feeling in control specifically, but not necessarily influence general positive or negative affect (46).

Saliva Sampling and Assays. In order to collect saliva, participants were instructed to drool approximately 2 mL of saliva into plastic centrifuge tubes, which was immediately frozen in a -20 °C freezer and then transported to a -80 °C freezer for long-term storage. Consistent with standard published procedures (47), saliva samples were later thawed and centrifuged at 3500 rpm for 10 minutes at room temperature. The remaining fluid was then aliquoted into 250 μL samples and frozen again before being thawed and analyzed for cortisol and testosterone in duplicate using enzyme immunoassay kits (Salimetrics, LLC; State College, PA). The average intra-assay coefficients of variation (CVs) were 5.59% (cortisol) and 6.00% (testosterone); the inter-

assay CVs were 8.22% (cortisol) and 8.10% (testosterone) averaged across low and high control samples.

Behavioral Ratings. Three trained research assistants (2 female), who were naïve to each participant's experimental condition and the purpose of the study, watched the first 2.5 minutes of each participant's speech. They then rated how much they agreed that twenty-nine variables were present in the video, on a scale from 1 – extremely disagree, to 8 – extremely agree. These variables were inspired by previous theory and research on behavioral responses to status and stress (15-53) and represented behavioral components of each participant's competence (e.g., intelligent, competent, etc.), dominance (e.g., confident, dominant, etc.), and warmth (e.g., warm, friendly, etc.) – three theorized behavioral routes to status attainment (18,54). The research assistants answered two additional questions regarding the participant's overall interview performance on separate scales: "How good was this interview?" (1 – extremely bad to 8 – extremely good) and "If you were in charge of hiring, how likely would you be to hire this individual?" (1 – extremely unlikely, to 8 – extremely likely). In order to reduce the potential for gender stereotypes to influence ratings, all male participants' videos were watched and rated in random order before female participants' videos were watched and rated in random order.

The research assistants' responses (average inter-rater reliability across all variables: $\alpha = .665$) were submitted to a factor analysis. A three-factor solution with varimax rotation was investigated and found to account for 66.4% of variance (see Table 2.9).

Statistical Analyses

Data Transformation. Two-tailed Kolmorogov-Smirnov tests of normality revealed non-normal distributions for cortisol and testosterone concentrations at multiple time points (*p*s < .03). We corrected this non-normality by natural-log-transforming cortisol and testosterone concentrations and used these transformed scores in analyses that examined the effects of the experimental manipulations on changes in endocrine concentrations over time. The scale that cortisol is measured on (e.g., Baseline concentration, M = .244 μg/dL, SE = .020, range = [.07, 1.84]) results in negative values for many of the log-transformed cortisol data. Thus, an arbitrary value of 10 was added to each transformed cortisol value to ensure that all values were positive for ease of interpretation. Testosterone's scale (e.g., Baseline concentration, M = 112.8, SE = 6.19, range = [30.4, 343.2]) does not result in negative transformed values and so did not require an arbitrary linear transformation.

Endocrine Analyses. To analyze the overall endocrine response patterns, 2 (High vs. Low Status) x 2 (Stable vs. Unstable Hierarchy) x 4 (Time) repeated measures GLM were used. Mauchly's test of sphericity revealed violations of sphericity, so Huynh-Feldt corrections were applied. For follow-up analyses of acute reactivity, each hormone's change from baseline to immediately after the stressor (TSST+0) was calculated. Similarly for recovery, endocrine change from baseline to forty minutes after the stressor was calculated. This index of recovery measures the extent to which individuals were exposed to a given hormone during a period in which hormones should decline following initial reactivity to the stressor (55). Larger, positive values indicate a hormone did not return to baseline during the forty minutes of recovery; a zero or negative value indicates a hormone did return to baseline (or sub-baseline levels consistent with circadian decline

in hormone concentrations). These values were regressed on status, hierarchical stability, and their interaction (in addition to participant sex for testosterone analyses) in separate univariate GLMs.

Behavioral Analyses. We conducted separate GLM analyses on interview performance, dominance, and warmth with status, stability, and their interaction as between subject variables with participant sex as a covariate.

Moderated Mediation Analyses. Using the *PROCESS* Model 8 template in *SPSS* (v. 22, IBM Corp.), our primary moderated mediation models were produced with interview performance as the outcome variable; social status as the independent variable; feeling in control, testosterone reactivity, or cortisol reactivity as the mediator; sex as a covariate; and hierarchy stability as a moderator. We also produced similar moderated mediation models with dominance or warmth as outcome variables. Bootstrap analyses were used to calculate bias-corrected 95% confidence intervals for the indirect effects of each putative mediator (n = 1000 subsamples). Indices of reactivity were based on standardized residuals that were produced from regressing TSST+0 concentrations on baseline concentrations. Residuals for recovery were calculated from regressing TSST+40 endocrine concentrations on baseline endocrine concentrations. For testosterone, these residual values were normally distributed when produced from raw testosterone concentrations. Raw cortisol concentrations resulted in skewed residuals, and so log-transformed cortisol concentrations were submitted to residual calculation and used in the moderated mediation models. These metrics of endocrine reactivity and recovery were employed in the correlational analyses (Table 2.11) and in the moderated mediation analyses.

Supplementary Analyses

In the sections below, we report supplemental analyses for (a) endocrine reactivity and recovery; (b) the moderating effects of sex; (c) robustness checks for the main analyses; (d) behaviors during the stressor; (e) moderated mediation analyses; and (f) positive and negative affect.

Supplementary Endocrine Analyses

Alternative Methods for Measuring Acute Endocrine Reactivity. The main text reports analyses for hormone reactivity to the stressor as change scores from Baseline to TSST + 0. We confirmed that the Status x Stability effects on endocrine reactivity extend to the following alternative methods for modeling acute reactivity: percent change scores; unstandardized residuals calculated by regressing endocrine concentrations at TSST+0 on the baseline concentrations; and area-under-the-curve with respect to increase (AUC_I; 56). AUC_I was calculated as:

$$AUC_i = \left(\frac{Baseline + TSST40}{2}\right) + TSST0 + TSST20 - (4-1) * Baseline$$

Distinguishing it from the other three indices of reactivity, AUCi takes into account all four samples and thus represents change in endocrine concentrations across the reactivity and recovery period. Tables 2.3 and 2.4 (and Figures 2.5 and 2.6) show results for cortisol and testosterone, respectively, across these different analyses.

	Raw Change			Percent Change			Unstandardized Residuals			AUCi		
	B	CI	p	B	CI	p	B	CI	p	B	CI	p
(Intercept)	0.25	0.17 – 0.32	**<.001**	0.03	0.02 – 0.04	**<.001**	0.00	-0.07 – 0.08	.907	0.52	0.29 – 0.75	**<.001**
Social Status	0.01	-0.06 – 0.09	.734	0.00	-0.01 – 0.01	.800	0.01	-0.06 – 0.09	.725	0.02	-0.21 – 0.24	.877
Hierarchy Stability	-0.08	-0.15 – -0.00	**.039**	-0.01	-0.02 – -0.00	**.034**	-0.07	-0.15 – 0.00	.060	-0.24	-0.47 – -0.01	**.037**
Status x Stability	-0.08	-0.16 – -0.01	**.030**	-0.01	-0.02 – -0.00	**.030**	-0.08	-0.16 – -0.01	**.025**	-0.25	-0.48 – -0.02	**.031**
Observations		110			110			110			110	
R^2 / adj. R^2		.082 / .056			.080 / .054			.077 / .051			.035 / .008	

Table 2.3. Cortisol reactivity modeled in several ways. Each column represents a separate GLM with effects coded variables (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1).

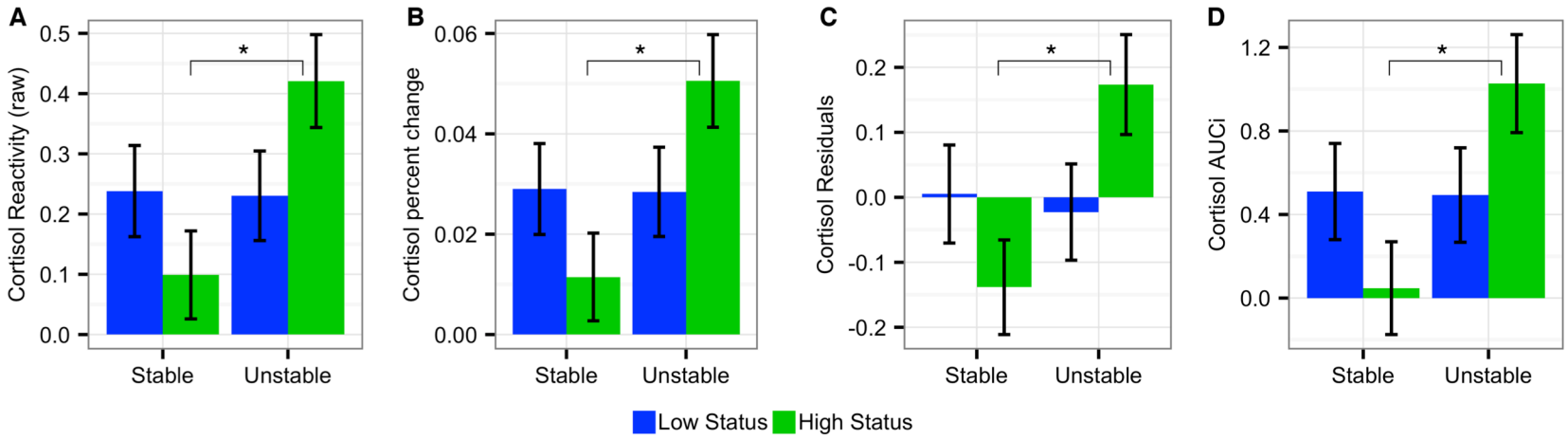

Figure 2.5. Cortisol reactivity modeled in several ways. **A**. Raw difference **B**. Percent change **C**. Residual change **D**. AUC_i * = significant uncorrected pairwise comparison at $p < .05$

	Raw Change			Percent Change			Residuals			AUCi		
	B	CI	p	B	CI	p	B	CI	p	B	CI	p
(Intercept)	0.13	0.10 – 0.16	**<.001**	0.03	0.02 – 0.04	**<.001**	0.00	-0.03 – 0.03	.814	0.21	0.13 – 0.28	**<.001**
Social Status	0.00	-0.03 – 0.03	.761	0.00	-0.01 – 0.01	.751	0.01	-0.02 – 0.03	.725	0.03	-0.05 – 0.10	.507
Hierarchy Stability	-0.03	-0.06 – 0.00	.090	-0.01	-0.01 – 0.00	.101	-0.02	-0.05 – 0.01	.107	-0.08	-0.15 – -0.00	**.048**
Sex	-0.00	-0.03 – 0.03	.987	-0.00	-0.01 – 0.00	.418	0.01	-0.02 – 0.04	.454	0.05	-0.03 – 0.13	.195
Status x Stability	-0.04	-0.07 – -0.01	**.008**	-0.01	-0.02 – -0.00	**.011**	-0.04	-0.07 – -0.01	**.008**	-0.12	-0.20 – -0.05	**.002**
Observations		110			110			110			110	
R^2 / adj. R^2		.090 / .055			.090 / .056			.090 / .055			.131 / .097	

Table 2.4. Testosterone reactivity modeled in several ways. Each column represents a separate GLM with effects coded variables (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1; Sex: Male = 1, Female = -1).

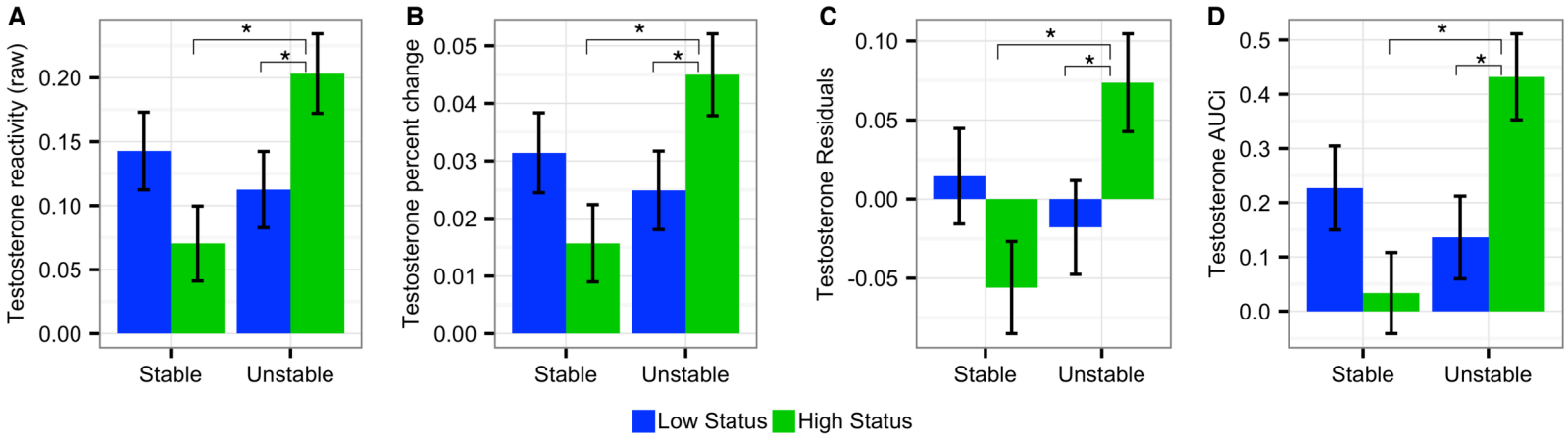

Figure 2.6. Testosterone reactivity modeled in several ways. **A**. Raw difference **B**. Percent change **C**. Residual change **D**. AUC_i * = significant uncorrected pairwise comparison at $p < .05$

Endocrine Recovery

Slope. The main text reports hormone recovery as change scores from Baseline to TSST +40. We also examined the effect of status and hierarchy instability on recovery slope. To calculate recovery slope, we used the lme4 package in R (57) to extract Empirical Bayes estimates of the linear slope that connects the three post-stressor samples (TSST+0, TSST+20, TSST+40) for each participant. This recovery slope represents a bias-corrected measurement of the rate at which participants' hormone concentrations changed over the three post-stressor samples and is appropriate for between-person comparisons of endocrine recovery (58, 59). Within this measure, more negative numbers represent a quicker reduction (steeper slope) following activation, and less negative numbers represent a more prolonged recovery period (flatter slope).

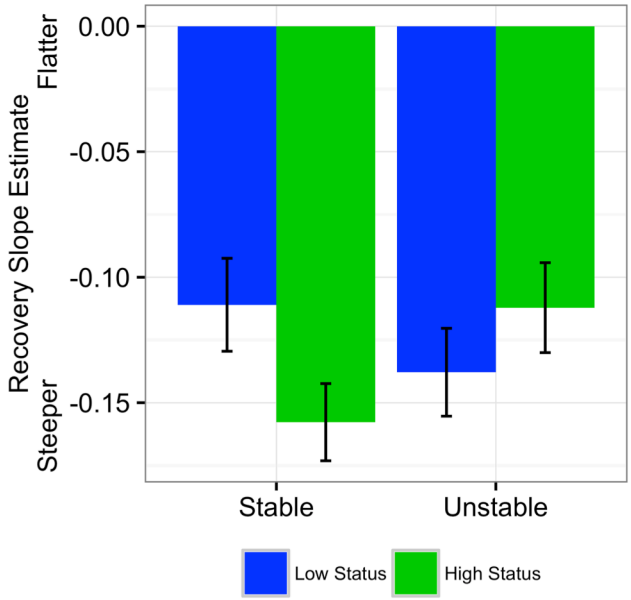

Figure 2.7. Cortisol recovery slope, calculated by extracting linear slope from TSST+0 to TSST+40

A GLM found a significant Status x Stability interaction on the recovery slope for cortisol ($F(1,106) = 4.38$, $p = 0.039$, $\eta^2 = 0.040$; Figure S3) but not testosterone ($F(1,105) = 0.564$, $p = 0.454$, $\eta^2 = 0.005$). The pattern of the interaction for cortisol indicates that high status individuals in a stable hierarchy exhibited steeper slopes, indicative of a quicker recovery following activation of the HPA axis. But in the unstable hierarchy, high status individuals had flatter slopes, indicative of extended activation of the HPA

axis. The opposite pattern was observed for low status individuals in stable versus unstable hierarchies, although none of the pairwise comparisons were significant.

Moderating Effect of Participant Sex. We explored sex as a moderator of the effects of status and hierarchy stability on all dependent variables reported in the main document (Table 2.5). Consistent with previous research (60), a Time x Sex effect was found for cortisol wherein men showed stronger reactivity to the stressor compared to women ($F(1.91, 194.57) = 6.28$, $p = 0.003$, $\eta^2 = 0.058$). However, the Time x Sex x Status x Stability interactions were non-significant for cortisol and testosterone (p-values > 0.16, $\eta^2 < 0.17$), suggesting that endocrine responses to social status and hierarchical instability did not depend on participant sex.

There was a Sex x Status x Stability interaction on dominance behavior (see Table 2.5). The pattern of the interaction revealed that the Status x Stability interaction on dominance was stronger in men than in women. There were non-significant interactions between status, stability, and sex for all other dependent variables reported in the main text. Collectively, these analyses revealed that the status x stability interactions on our primary dependent variables generally showed similar effects in men and women.

	Cortisol reactivity		Testosterone reactivity		Feeling in control		Interview Performance		Dominance		Warmth	
	B	P	B	p	B	P	B	P	B	p	B	p
(Intercept)	0.26	**<.001**	0.13	**<.001**	2.35	**<.001**	5.14	**<.001**	4.71	**<.001**	4.59	**<.001**
Status	0.01	.713	0.00	.853	0.17	**.041**	0.18	**.016**	0.23	**.001**	0.04	.624
Hierarchy Stability	-0.08	**.032**	-0.03	.068	0.08	.325	-0.09	.232	-0.03	.713	0.00	.972
Sex	0.07	.052	0.00	.945	0.38	**<.001**	-0.12	.103	0.05	.448	-0.19	**.024**
Status x Stability	-0.09	**.022**	-0.04	**.005**	0.21	**.011**	0.17	**.023**	0.20	**.004**	0.18	**.029**
Status x Sex	-0.01	.772	-0.02	.172	0.11	.205	0.12	.117	0.01	.923	-0.11	.165
Stability x Sex	-0.01	.894	-0.01	.349	0.02	.789	-0.01	.856	0.05	.496	-0.08	.303
Status x Stability x Sex	-0.03	.434	-0.02	.141	0.06	.503	0.11	.146	0.14	**.047**	0.03	.741
Observations	110		110		111		109		109		109	
R^2 / adj. R^2	.123 / .063		.125 / .065		.260 / .210		.166 / .108		.202 / .146		.117 / .056	

Table 2.5. Status x Stability x Sex GLMs showing generally null moderating effects of participant sex. Status, stability, and sex are effects coded (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1; Sex: Male = 1, Female = -1).

Additional robustness checks. We conducted two additional robust checks:

1) We tested the extent to which the endocrine results remained robust when controlling for covariates relevant to endocrine function (participant sex, time since awakening, and hours of sleep prior to the experimental session; Table 2.6) and socioeconomic status (subjective social status via the "ladder" survey (61); mother's and father's education; and family income; Table 2.7). These analyses revealed statistically significant status x hierarchy stability interactions across all models.

2) We examined models with bias-corrected bootstrapped estimates. Bias-corrected bootstrap estimates of the status x stability interaction term were produced for the six main GLMs (endocrine reactivity, sense of control, and the three behavioral factors) using the "boot" library in R (62, 63). The models were replicated 1000 times and the bias-corrected and accelerated (BCa) bootstrap estimates of the 95% confidence intervals were extracted. Effects were considered robust if the 95% confidence intervals for the status x stability interaction term did not contain zero. For each model, the social status x hierarchy stability interaction term was robust to bootstrap bias correction (Table 2.8).

	Model 1		Model 2		Model 3	
A: ΔCortisol	*B*	*p*	*B*	*p*	*B*	*p*
Intercept	0.25	**<.001**	0.26	**<.001**	0.44	.159
Social Status	0.01	.741	0.01	.724	0.02	.675
Hierarchy Stability	-0.08	**.041**	-0.08	**.034**	-0.08	**.041**
Status x Stability	-0.08	**.031**	-0.09	**.025**	-0.08	**.031**
Sex			0.07	.052	0.08	.053
Time since awakening					-0.01	.530
Hrs of sleep					-0.01	.697
Observations	109		109		108	
R^2 / adj. R^2	.080 / .054		.117 / .083		.119 / .066	

	Model 1		Model 2	
B: ΔTestosterone	*B*	*p*	*B*	*p*
Intercept	0.13	**<.001**	0.13	.307
Social Status	0.00	.797	0.01	.705
Hierarchy Stability	-0.03	.102	-0.03	.063
Sex	0.00	.973	0.00	.909
Status x Stability	-0.04	**.007**	-0.04	**.008**
Time since awakening			0.01	.449
Hrs of sleep			-0.01	.606
Observations	109		109	
R^2 / adj. R^2	.090/.055		.102/.048	

Table 2.6. Cortisol and testosterone reactivity to stress controlling for biosocial variables. **A.** Log-transformed cortisol change (TSST+0 – baseline) regressed on the listed variables. **B.** Log-transformed testosterone change regressed on the listed variables. All categorical variables are effects coded (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1; Sex: Male = 1, Female = -1.

	Model 1		Model 2		Model 3		Model 4	
A: ΔCortisol	B	p	B	p	B	p	B	p
Intercept	0.25	**<.001**	0.44	**.006**	0.18	.148	0.41	.110
Social Status	0.01	.741	0.02	.569	0.02	.687	0.03	.503
Hierarchy Stability	-0.08	**.041**	-0.08	**.048**	-0.08	**.039**	-0.08	**.043**
Status x Stability	-0.08	**.031**	-0.09	**.034**	-0.08	**.033**	-0.09	**.033**
Mother's Education			-0.01	.751			-0.01	.774
Father's Education			-0.01	.666			-0.01	.664
Family Income			-0.01	.454			-0.02	.471
Subjective SES					0.02	.565	0.01	.780
Observations	109		105		107		103	
R^2 / adj. R^2	.080 / .054		.102 / .047		.085 / .049		.107 / .041	

	Model 1		Model 2		Model 3		Model 4	
B: ΔTestosterone	B	p	B	p	B	p	B	p
Intercept	0.13	**<.001**	0.20	**.003**	0.05	.307	0.06	.559
Social Status	0.00	.797	0.01	.675	0.00	.863	0.01	.728
Hierarchy Stability	-0.03	.102	-0.03	.071	-0.02	.128	-0.03	.076
Sex	0.00	.973	0.00	.848	0.00	.752	0.00	.807
Status X Stability	-0.04	**.007**	-0.04	**.008**	-0.04	**.008**	-0.04	**.010**
Mother's Education			-0.01	.486			-0.01	.568
Father's Education			0.00	.798			0.00	.656
Family Income			-0.01	.451			-0.00	.929
Subjective SES					0.02	.080	0.02	.114
Observations	109		105		107		103	
R^2 / adj. R^2	.088 / .053		.105 / .060		.112 / .048		.122 / .048	

Table 2.7. Cortisol and testosterone reactivity, controlling for relevant socioeconomic status variables. **A.** Log-transformed cortisol change (TSST+0 – baseline) regressed on the listed variables. **B**. Log-transformed testosterone change (TSST+0 – baseline) regressed on the listed variables. All categorical variables are effects coded (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1; Sex: Male = 1, Female = -1).

	$B_{original}$	B_{robust}	95%CI
Cortisol Reactivity	-0.0827	-0.0832	(-0.158, -0.007)
Testosterone Reactivity	-0.0415	-0.0418	(-0.072, -0.013)
Sense of Control	0.209	0.210	(0.04, 0.386)
Interview Performance	0.170	0.171	(0.025, .317)
Dominance	0.193	0.189	(0.067, 0.338)
Warmth	0.169	0.170	(0.015, 0.326)

Table 2.8. Bias-corrected bootstrap estimates (r = 1000 subsamples) of the status x stability interaction term and its 95% confidence interval (extracted via BCa method) from each analysis. Each row represents a GLM used in the main document to analyze the DV listed in the first column.

Supplementary Behavioral Analyses

Table 2.9 reports factor loadings and inter-rater reliabilities. Bold numbers indicate that the item loaded on a single factor at > 0.5 and therefore was included in that factor[3].

The inter-rater reliabilities for individual items are generally in line with other research on status-relevant behaviors (e.g., 18). We also examined the inter-rater reliabilities for each behavioral factor (bottom row, Table 2.9). This metric of inter-reliability is appropriate because our statistical analyses employed these aggregated factors (64, 65). Each rater's scores were averaged into the interview performance, dominance, and warmth behavioral factors prior to calculating Cronbach's α for inter-rater reliability. Doing so revealed higher inter-rater reliabilities (Interview Performance:

[3] Observers also rated participants on six additional items that did not satisfactorily on any one of the factors and were excluded from statistical analyses. These items were: Fidgets with hands, bodily motion, etc.; fidgets with items like a pencil, study equipment, etc.; likeable; maintains eye contact; talks fast; and stumbles over words.

$\alpha = 0.835$; Dominance: $\alpha = 0.834$; Warmth: $\alpha = 0.769$), suggesting that the raters generally agreed on the aggregate measures of behavior.

Despite achieving high inter-reliability at the behavioral factor level, the inter-rater reliabilities for some of the individual items indicated low to moderate agreement among raters. Thus, we tested whether the status x stability interaction on behaviors during the stressor would show the same general pattern after excluding items with inter-rater reliabilities of Cronbach's $\alpha < .60$. This cutoff removed six items – five items from the dominance factor (nervous, stressed, awkward, strong posture, and dominant appearance), one from the warmth factor (humorous), and none from the interview performance factor. This subset of items raised the average inter-rater reliability to $\alpha = 0.714$. We then used GLMs to regress the new behavioral factors on social status, hierarchy instability, and their interaction (controlling for participant sex). As shown in Table 2.10, the interaction between status and stability remained statistically significant for dominance and was marginally significant for warmth, though the effect was in the same direction and magnitude as the original analysis. Overall, these new analyses reveal the same pattern of results as the main analyses.

	Interview Performance	Dominance	Warmth	Inter-rater Reliability (Cronbach's α)
How good was this interview?	**0.832**	0.364	0.267	0.827
Competent	**0.830**	0.353	0.231	0.640
Intelligent	**0.825**	0.180	0.212	0.635
How likely would you hire person?	**0.824**	0.360	0.285	0.808
Engaged	**0.775**	0.280	0.378	0.674
Coherent	**0.747**	0.446	0.089	0.702
Bored	**-0.742**	-0.055	-0.427	0.612
Creative	**0.546**	0.312	0.337	0.668
Leader-like	0.282	**0.819**	0.303	0.778
Confident	0.345	**0.818**	0.307	0.827
Nervous	-0.185	**-0.817**	0.132	0.525
Follower-like	-0.108	**-0.814**	-0.138	0.649
Dominant sounding	0.115	**0.808**	0.258	0.724
Stressed	-0.147	**-0.758**	0.132	0.574
In control	0.556	**0.735**	0.140	0.795
Awkward	-0.161	**-0.711**	0.051	0.543
Strong posture	0.144	**0.705**	0.074	0.587
Quiet	-0.089	**-0.686**	-0.517	0.727
Dominant appearing	-0.112	**0.670**	0.292	0.574
High Status	0.220	**0.500**	0.380	0.650
Warm	0.456	0.079	**0.799**	0.679
Happy	0.301	0.166	**0.863**	0.774
Friendly	0.379	0.081	**0.839**	0.677
Smile-y (smiled a lot)	0.147	0.053	**0.810**	0.716
Humorous	0.118	0.178	**0.696**	0.350
Inter-rater Reliability of aggregate behavior (Cronbach's α)	**0.835**	**0.834**	**0.769**	

Table 2.9. Factor loadings and inter-rater reliabilities for behavioral items, as assessed by independent observers' ratings of the videotaped social-evaluative stressor.

	Dominance				Warmth			
	Original		New (reduced)		Original		New (reduced)	
	B	p	B	p	B	p	B	p
(Intercept)	4.73	**<.001**	4.76	**<.001**	4.59	**<.001**	4.83	**<.001**
Social Status	0.25	**.001**	0.26	**.001**	0.05	.566	0.06	.476
Hierarchy Stability	-0.06	.421	-0.07	.344	0.01	.907	0.02	.868
Sex	0.07	.321	0.06	.437	-0.18	**.039**	-0.21	**.023**
Status x Stability	0.19	**.008**	0.23	**.004**	0.17	**.041**	0.16	.074
Observations	106		106		106		106	
R^2 / adj. R^2	.182 / .149		.181 / .148		.081 / .044		.082 / .045	

Table 2.10. Comparison of GLMs for behavior factors with and without items that had lower inter-rater reliability. Interview performance is not displayed because it did not contain items with lower inter-rater reliability. Status, stability, and sex are effects coded (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1; Sex: Male = 1, Female = -1).

Supplemental Moderated Mediation Analyses

We report moderated mediation analyses for interview performance in the main document; here we report the partial correlations among the main variables controlling for sex (Table 2.11), the conditional indirect effects for those analyses (Table 2.12), as well as moderated mediation results for dominance and warmth. The Status x Stability interaction on dominance was mediated by feeling in control ($\omega = 0.098$, 95%CI [0.028, 0.205]) but not testosterone or cortisol reactivity (95% CI's overlapped with zero). These results extend prior research (46) by showing that hierarchical instability disrupts the effect of high status on dominance behaviors via reduced feelings of control. The Status x Stability interaction on warmth was not significantly mediated by feeling in control,

	1	*2*	*3*	*4*	*5*	*6*	*7*
1.Cortisol Reactivity							
2. Cortisol Recovery	0.75**						
3. T Reactivity	0.52**	0.40**					
4. T Recovery	0.20*	0.43**	0.38**				
5. In-Control	-0.09	-0.13	-0.17†	0.03			
6. Interview Performance	-0.004	-0.08	-0.23*	-0.01	0.35**		
7. Dominance	-0.05	-0.14	-0.07	-0.05	0.37**	0.61**	
8. Warmth	0.05	-0.02	-0.10	-0.07	0.15	0.55**	0.41**

Table 2.11. Partial correlations (controlling for sex) between the primary dependent measures. Reactivity and recovery are calculated by regressing endocrine concentrations at TSST+0 or TSST+40 (respectively) on baseline endocrine concentrations. **p < 0.001; *p < 0.05; †p < 0.10

testosterone reactivity, or cortisol reactivity (95% CIs overlapped with zero). Testosterone and cortisol recovery were not found to mediate any of the behaviors (95% CIs overlapped with zero). These non-significant mediations suggest that other psychological and biological factors that were not measured in the present experiment may explain the effects of the hierarchy on warmth (e.g., progesterone changes, which have been linked to affiliation motivation, 66). Additional studies will be required to identify the mechanisms through which the social hierarchy influences warmth behavioral responses to stress.

Our primary correlational and mediation analyses revealed that greater sense of control was positively related to interview performance, whereas testosterone reactivity was negatively related to interview performance. Additional analyses revealed non-significant Sex x Testosterone reactivity and Sex x Feeling in control interactions on

interview performance (*p*s> .10, η^2s < .026). These results suggest that the pathways between these mediators and interview performance did not statistically differ between male and female participants.

Supplemental Analyses for Self-Reported Affect

Participants responded to thirteen items related to their momentary positive and negative affect on a 1 to 5 scale, from "Not at all" to "Extremely." These questions were administered after having status and stability assigned and immediately after the stressor. Positive affect: Interested, excited, happy, strong, enthusiastic, proud, and self-confident (Cronbach's α =0.89). Negative affect: Distressed, upset, sad, irritable, ashamed, and

	In Control		Testosterone Reactivity	
	Stable	Unstable	Stable	Unstable
Interview Performance	.11 [.040, .232]	-.004 [-.09, .067]	.045 [.000, .139]	-.043 [-.12, -.004]
Dominance	.099 [.045, .183]	-.003 [-.07, .06]	---	---
	Low Status	High Status	Low Status	High Status
Interview Performance	-.037 [-.154, .021]	.077 [.022, .188]	-.013 [-.081, .013]	.020 [.000, .151]
Dominance	-.033 [-.118, -.015]	.069 [.029, .141]	---	---

Table 2.12. The conditional indirect effects (ω) for each significant moderated mediation with bias-corrected bootstrapped 95% confidence intervals. Status and hierarchy stability are effects coded (Status: High = 1, Low = -1; Stability: Stable = 1, Unstable = -1); all analyses include participant sex as a covariate (Males = 1; Females = -1). The top section of the table shows conditional indirect effects with hierarchy stability as the moderator of the influence of status on interview performance and dominance. The bottom section of the table shows the conditional indirect effects with status as the moderator of the influence of hierarchy stability on interview performance and dominance. We include both sets of conditional effects to inform follow-up research.

nervous (Cronbach's α =0.82). The aggregated positive and negative affect scores were submitted to separate 2 (Time) x 2 (High vs. Low Status) x 2 (Stable vs. Unstable Hierarchy) mixed GLM analyses. There was a marginally significant Status x Stability x Time interaction on positive affect ($F(1,104) = 3.50$, $p = 0.064$, $\eta^2 = 0.033$) but not negative affect ($F(1,104) = 0.958$, $p = 0.330$, $\eta^2 = 0.01$). As shown in Figure 2.8, the pattern of the interaction aligns with the hierarchy instability hypothesis. The stronger effects for feeling in control reported in the main text compared to the results reported here are consistent with social hierarchy theories, which posit that social rank influences behavior through perceived controllability as opposed to global positive or negative affect (46).

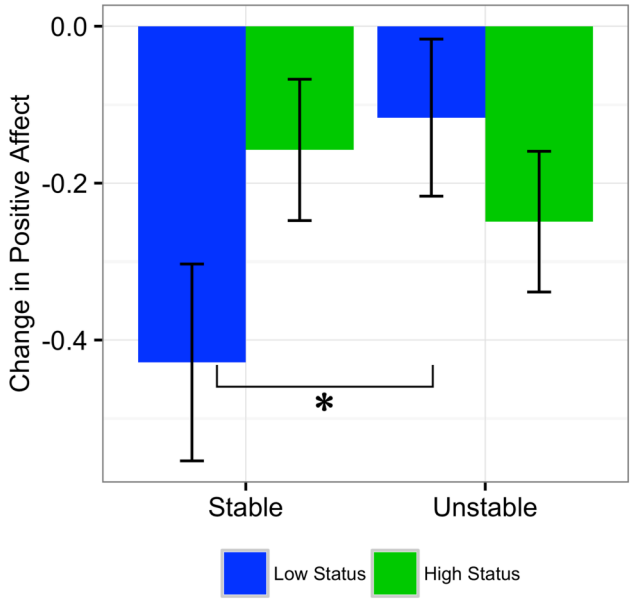

Figure 2.8. Change in self-reported positive affect from pre- to post-stress. Error bars represent standard errors of the mean. * = significant uncorrected pairwise comparison at p < .05

CHAPTER III

BRIDGING THE GAP OF TESTOSTERONE'S CAUSAL EFFECTS ON STRESS RESPONSES

The prior chapter demonstrates the causal effects of social status and hierarchy stability on responses to stress. This work suggests that the beneficial effects of high rank on social status depend on the stability of that high rank. Of particular interest, unstable high ranking caused an increased testosterone response to the stressor, which was then found to mediate differences in behavioral responses to the stressor. The preceding chapter hypothesized that testosterone might lead an individual to focus on his or her threatened social position, resulting in disrupted cognitive processes and poor behavioral performance.

Despite this mediating effect of testosterone, the causal direction of the relationship between testosterone and behavioral responses to social stressors cannot be determined. Further, a large body of work indicates that testosterone modulates cortisol (1-3) and affective responses (4,5) to stressors as well. In order to examine the causal effects of testosterone on the cortisol, affective, and behavioral stress responses, the next chapter experimentally manipulates testosterone levels prior to a social-evaluative stressor. This work extends findings in the previous chapter by providing clarity to the causal effects of testosterone on endocrine, affect, and behavioral responses to social-evaluative stressors.

CHAPTER IV

EXOGENOUS TESTOSTERONE ENHANCES CORTISOL AND AFFECTIVE RESPONSES TO SOCIAL-EVALUATIVE STRESS IN DOMINANT MEN

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Introduction

Stress is a leading contributor to poor health and mortality: Exposure to chronic or severe stress predicts increased risk for cardiovascular disease, psychiatric conditions such as depression and substance use disorders, and infectious disease (1-3). Cortisol, a steroid hormone released as part of the hypothalamic-pituitary-adrenal (HPA) axis response to stress, is a key mechanism through which stress influences physical and mental health via alterations in immune system activity and neurotoxic effects within the central nervous system (1,4,5). Stress also heightens negative affect, which is an independent pathway that predicts poor mental and physical health (2,6).

Prevailing theories propose that the sex hormone testosterone should reduce stress responses, but the causal effect of testosterone on stress reactivity in humans remains unclear. In animal (e.g., rodent) models of stress, testosterone reduces cortisol reactivity to stress (7) and reduces fear behavior (8). Consistent with this stress-buffering account, in humans, testosterone suppresses cortisol responses to pharmacological stimulation of the HPA axis (9), and reduces unconscious attention to fearful faces (10). Yet other studies indicate that testosterone correlates with *increased* cortisol and negative affect in response to situations that threaten social status, like losing a competition (11) or being relegated to a low-ranking social position (12). Testosterone reactivity to a stressor has also been found to mediate decreases in behavioral performance during a stressor

following a status threat manipulation (13). This correlational evidence is convergent with theorizing that testosterone directs the pursuit and maintenance of social status (14) and suggests that testosterone may enhance acute stress responses when the stressor relates to social status, like during a social evaluation for a high-status job. Despite this correlational evidence, the causal effects of testosterone on responses to status-relevant social stress have not been adequately tested in healthy young adults[4]. Further, the few correlational studies that have examined testosterone and social stress have provided mixed evidence linking testosterone levels to both increased (14) and decreased (16) cortisol responses to stress. In order to clarify this inconsistent correlational evidence, we provide a direct causal test of testosterone's impact on cortisol and negative affect responses to stress using a status-relevant social stressor. We also explore what effect testosterone has on causing behavioral responses to social-evaluative stress. Moreover, given the increasing rate at which testosterone is prescribed (17), administering testosterone prior to a social-evaluative stressor will provide much needed insight into testosterone's potential impact on stress and health.

Prior work also indicates that testosterone's causal effects on status-relevant behavior depend on trait dominance, an individual difference factor relevant to concern for status attainment. High levels of trait dominance – that is, an individual's propensity to use force, fear, or intimidation to gain high-ranking positions within social groups (19) – accentuate the behavioral effects of testosterone in status-relevant situations. For example, exogenous testosterone increases aggression after provocation (20) and

[4] But *cf.* 18, for a small sample of older men that found no difference in cortisol response to an acute stressor several days after a single dose of testosterone vs. placebo.

increases competitive behavior after winning a contest (21), but only in individuals high in self-reported trait dominance. These behavioral results suggest that trait dominance may also exacerbate the influence of high testosterone levels on responses to status-relevant stressors.

Methods and Materials

Participants

Male participants (n = 120) between the ages of 18-40 (Mean: 22.50 years; SE: 0.33; Max: 39.6) were recruited via emailing campus listservs and by placing flyers on and near campus. All interested parties were screened for medical conditions that would prevent participation in the study, including immune, endocrine, neurological, or mental health conditions, and alcohol or drug abuse. The University of Oregon's Institutional Review Board approved all methods.

Protocol

Once screening had been passed, the participant chose a day (Monday – Saturday)

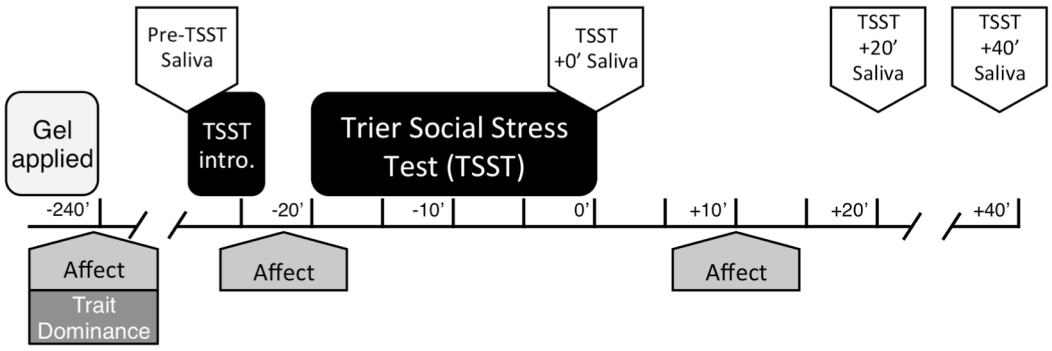

Figure 4.1: Timeline of the experimental protocol. All timings are approximate and are represented in minutes relative to the end of the social-evaluative stressor. "TSST intro." represents the instructions for the social-evaluative stress procedure.

to attend an approximately six-hour laboratory session, which began between 9:00 AM and 11:00 AM. These start times were chosen so that the social-evaluative stressor would occur between 1:00 – 3:00 PM to control for diurnal endocrine variation. Participants were asked to refrain from eating or drinking anything other than water or brushing their teeth 1.5 hours prior to the start of the laboratory session. Upon arrival at the laboratory session, experimenters obtained informed consent from the participant and collected a basal saliva sample (see **Figure 4.1** for a study timeline of saliva sampling and self-report data collection).

Topical Gel Application and Blinding. Participants were informed that they would be given a gel containing testosterone or placebo as well as a note in a sealed envelope. Participants were further told that the note would either reveal whether the gel was testosterone or placebo – a single-blind condition – or simply tell him that he had an equal chance of receiving testosterone or placebo – a double-blind condition. The single- and double-blind conditions were implemented in order to be able to control for the expected effects (i.e., the 'conventional wisdom') of receiving testosterone (22). A laboratory member who was not involved in data collection prepared the envelopes prior to the start of data collection in the study, thus the experimenter in the laboratory session never knew whether the vial contained testosterone or placebo, or whether the participant was in the single- or double-blind condition. All analyses control for blinding.

Under the supervision of the experimenter, the participant wore rubber gloves (in order to prevent cross contamination of laboratory equipment) and rubbed small increments of the lotion onto his own shoulders and upper arms. The participant was then given several minutes to read the contents of the envelope, during which the gel dried.

The testosterone (or placebo) was allowed to absorb into the body for 3 hours while the participant filled in baseline questionnaires.

Pharmacological Manipulation. The testosterone gel (Androgel, AbbeVie, Inc., North Chicago, IL) consisted of a 150-mg dose of testosterone in addition to pharmacologically inactive ingredients (i.e., carbomer 980, ethanol 67.0%, isopropyl myristate, purified water, and sodium hydroxide). The placebo gel contained the same inactive ingredients as the testosterone gel; the lack of testosterone was the only difference between the testosterone and placebo gels. The testosterone dose and time course for the research protocol was based on prior topical testosterone administration research that showed that serum testosterone concentrations peaked 3 hours after a 150-mg testosterone gel dose (23). Prior research has also shown physiological effects and neural reactivity 3-6 hours after testosterone administration (24,25). In order to execute our protocol during probable peak concentrations and conclude data collection within a 6-hour time period, this project utilized a 150-mg dose of testosterone approximately three hours prior to beginning the data collection portion of the study, and approximately four hours (Mean = 3.98 hours, SE = 0.015 hours) prior to the social-evaluative stressor.

Social-Evaluative Stressor. The social-evaluative stressor consisted of the Trier Social Stress Test (TSST; 26), in which participants were asked to complete a mock job interview for a high-status, managerial position followed by a verbal mental math task. The job was outlined in a printed job posting that was designed to be representative of an early career position consisting of managing a small team (i.e., “up to twelve student employees”) in a campus business office. Two panelists wearing white lab coats conducted the interview. These panelists were trained to maintain neutral affect and

behavior throughout and were provided with verbal responses for interactions with participants.

Other tasks. The study protocol contained two decision-making paradigms prior to the social-evaluative stressor in order to maximize the data obtained from each participant undergoing exogenous testosterone administration. These included (i) a competition task and (ii) a pro-social decision-making task; both of which will be analyzed and reported elsewhere. A second saliva sample (analyzed in the *Supplemental Materials*) was collected prior to the start of the competition task (3 hours after the gel application). The social-evaluative stress protocol commenced after these decision-making tasks.

Questionnaires

Dominance and Prestige Scale. The measure for trait dominance was taken from a scale that measures both the dominance aspects of status-seeking motivation, related to obtaining status via force, fear, or intimidation, as well as prestige, which is related to obtaining status via competence, adept social skills, or respect (19). The survey consists of 17 items related to dominance (e.g., "I try to control others rather than permit them to control me.") and prestige (e.g., "Members of my peer group respect and admire me.") on a scale from 1 (not at all) to 7 (very much). Dominance (Cronbach's $\alpha = .68$) and prestige items (Cronbach's $\alpha = .83$) were averaged and normalized within each subscale.

Positive and Negative Affect. The PANAS-X general negative affect and general positive affect subscales were used to measure affect responses. Participants responded on a 1 (not at all or very little) to 4 (quite a bit) scale. Negative (average Cronbach's $\alpha = 0.89$) and positive (average Cronbach's $\alpha = 0.84$) items were averaged for each time point

according to published guidelines (27). We also conducted exploratory analyses on the lower-order subscales that underlie general negative affect (fear, hostility, guilt, sadness) and general positive affect (joviality, self-assurance, attentiveness; see *Supplementary Materials*).

Saliva Sampling and Endocrine Assays

The cortisol response to stress was determined from saliva samples collected once before and +0, +20, and +40 minutes after the stressor (see *Supplementary Materials* for analyses of additional samples that occurred prior to the stressor, **Figures 4.5** and **4.6**). In order to collect saliva, participants were instructed to drool approximately 2 mL of saliva into polypropylene centrifuge tubes. Saliva was chosen for this study (instead of e.g. serum measures) because the length of the study (approximately 6 hours) would have required the use of an indwelling catheter, which was deemed an unnecessary added risk. Salivary measures also avoid the probable confounds of increased stress of collecting serum samples (i.e., venipuncture and/or indwelling catheter could induce a confounding stress response). Saliva samples were immediately frozen in a -20 °C freezer and then transported to a -80 °C freezer for long-term storage. Consistent with standard published procedures (28), saliva samples were later thawed and centrifuged at 3500 rpm for 10 minutes at room temperature. The remaining fluid was then aliquoted into 250 μL samples and frozen again before being thawed and analyzed for cortisol and testosterone in duplicate using enzyme immunoassay (EIA) kits (DRG, Germany). The average intra-assay coefficients of variation (CVs) were 4.68% (cortisol) and 6.55% (testosterone, for samples within range); the inter-assay CVs were 14.8% (cortisol) and 16.1% (testosterone) averaged across low and high control samples.

Because of the exogenous testosterone administration, testosterone levels were too high to be read on the commercial EIA kits available in 34.4% of samples within the exogenous testosterone group (17% of all samples). As a conservative estimate of the testosterone concentrations, we replaced these unknown values with the maximum value for the EIA kit, 5250 pg/mL. If one of the two duplicate samples was within the kit's range, we calculated the average of that known value and the 5250 pg/mL maximum. This conservative data replacement strategy provides a low-end estimate of the average testosterone concentration for the individuals administered exogenous testosterone.

Status-relevant Behavior During Social-Evaluative Stressor

Four trained research assistants, who were naïve to each participant's treatment condition, viewed the first three minutes of each performance and rated the performance on a series of 27 variables on a scale from 1 (extremely disagree) to 8 (extremely agree). These variables were selected to approximate three routes by which status is often earned (13), including competence (e.g., intelligent, coherent, bored (reverse scored)), dominance (e.g., dominant posture, leader-like), and warmth (e.g., friendly, happy). The videos were also rated for interview performance (i.e., "How good was this interview?" and "Would you hire this person?").

Analytical Plan

Multi-level models were constructed to examine Time x Testosterone vs. Placebo (T/P) and Time x T/P x Dominance effects on the cortisol response to social-evaluative stress. The Time x T/P model at the first level consisted of within-participant, polynomial-contrasted effects of time and, at the second level, a cross-level interaction of T/P with time (see *Supplementary Materials* for full models). T/P condition was effects

coded (Testosterone = 1, Placebo = -1). The linear and quadratic effects of time were chosen *a priori* to model the linear rate of change (linear effects) and the curvature of the growth trajectory (quadratic effects; 29). All analyses controlled for participant blinding condition.

In order to confirm interpretations of the multilevel models of the cortisol response, we conducted separate GLM analyses on measures of cortisol reactivity and recovery to the social-evaluative stressor. Reactivity was calculated by subtracting pre-stress cortisol concentration from cortisol concentration twenty minutes after the stressor (the +20 sample); recovery was calculated by subtracting pre-stress cortisol concentration from cortisol concentration forty minutes after the stressor (the +40 min sample). A positive recovery score indicates that cortisol levels had not returned to baseline forty minutes after the end of the stressor; a negative recovery score indicates that cortisol levels had fallen below the baseline levels. A more general measure of cortisol reactivity that takes into account all four samples, area-under-the-curve with respect to increase (AUC_I), was also calculated according to published recommendations (30).

All analyses were run in R (ver. 3.3.1, 31) using the lme4 package for multilevel models (32). The 95% confidence intervals for effect estimates were calculated via the sjPlot package (33) and all graphs were produced in ggplot2 (34).

Results

Preliminary Analyses

Four participants (n = 2 from testosterone treatment group) did not complete the social-evaluative stressor and were excluded from analyses. Two additional participants were missing a single sample – one participant left the laboratory prior to completing the

TSST+40 sample and one participant's sample was improperly aliquoted during the assay process – but these participants were left in the analyses as multilevel models are generally able to account for singular missing data points. Outliers (>3 SD) for negative affect were found at each time point (Baseline: n = 2; Pre-TSST: n = 3; Post-TSST: n = 1); these values were Winsorized to a score at 3 SD above the mean for each time point.

As expected, the testosterone gel substantially increased testosterone concentrations (mean of post-gel testosterone concentrations, Testosterone group: M = 2959.87 pg/mL, *95%CI*[2472.46, 3447.28]; Placebo group: M = 164.00 pg/mL, *95%CI*[122.99, 205.01]; see *Supplementary Materials*, **Figure 4.7**). No differences in baseline cortisol or affect were found between treatment groups or in exploration of interactions between treatment group and trait dominance (see *Supplementary Materials*).

Cortisol Response to Stress

Examining the impact of exogenous testosterone on cortisol concentrations across time revealed a significant Time x Testosterone/Placebo (T/P) condition interaction, such that exogenous testosterone increased cortisol responses to the social-evaluative stressor compared to placebo (Time x T/P: $B = 0.020$, *95%CI*[0.001, 0.038]; **Figure 4.2A**, **Table 4.1**, *Supplementary Materials*). As predicted, this Time x T/P interaction was moderated by trait dominance (Time x T/P x Dominance: $B = 0.020$, *95%CI*[0.002, 0.038]; $Time^2$ x T/P x Dominance: $B = -0.021$, *95%CI*[-0.039, -0.003]; **Figure 4.2B**). To decompose this interaction, we split the data at the median dominance rating and examined Time x T/P interactions for high and low trait dominance individuals. For individuals high in trait dominance, testosterone significantly increased the cortisol response to the social evaluative stressor compared to placebo (Time x T/P: $B = 0.044$, *95%CI*[0.018, 0.070];

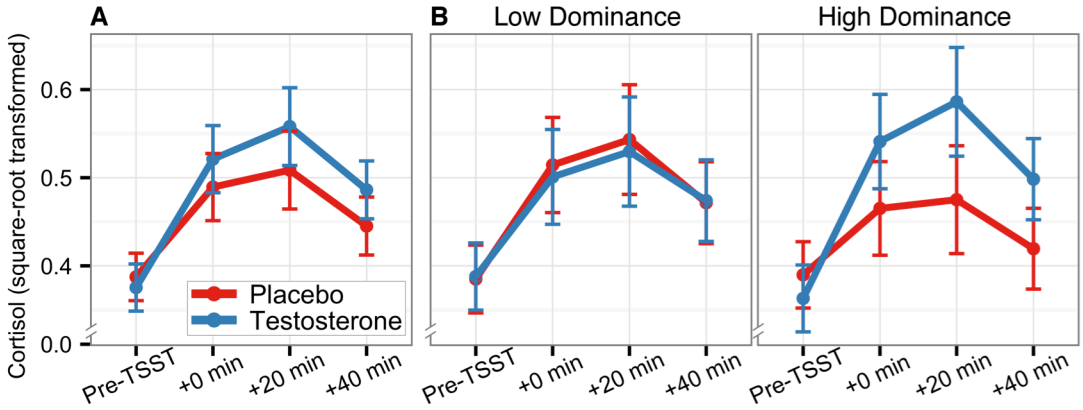

Figure 4.2: Cortisol response to social-evaluative stress. All values are estimated marginal means from relevant models and all error bars are 95% confidence intervals. A. Time x Testosterone vs. Placebo (T/P) effect on cortisol response. B. Time x T/P effect on cortisol response graphed at ±1 SD trait dominance.

Time^2 x T/P: *B* = -0.034, *95%CI*[-0.060, -0.007]). But for individuals low in trait dominance, the testosterone and placebo groups did not significantly differ in their cortisol responses (estimates of Time x T/P 95%CIs contain zero; **Table S2**). Trait prestige levels did not interact with T/P to predict cortisol levels (all 95%CIs contain zero; **Tables S3**).

Follow-up analyses on cortisol reactivity and recovery confirmed the multilevel models, revealing significant T/P x Dominance interactions for reactivity (B = .038, 95%CI[0.006, 0.070]; **Figure 4.3A**), recovery (B = .026, 95%CI[0.002, 0.049]; **Figure 4.3B**), and AUC_I (B = .154, 95%CI[0.025, 0.282] ; **Figure 4.3C**). Thus these analyses confirm that high dominant men given testosterone showed increased reactivity to the stressor and reduced propensity to return to baseline (i.e., increased recovery scores) after the stressor compared to men given placebo.

Positive and Negative Affect Responses

Testosterone increased negative affect in anticipation of the stressor compared to placebo (Time x T/P: *B* = 0.050, *95%CI*[-0.010, 0.111]; Time^2 x T/P: *B* = -0.044, *95%CI*[-0.088, -0.001]; **Figure 4.4A**). Testosterone's causal increase of negative affect across time was also moderated by trait dominance (Time x T/P x Dominance: *B* = 0.080, *95%CI*[0.021, 0.139]; Time^2 x T/P x Dominance: *B* = -0.036, *95%CI*[-0.079, 0.007]; **Figure 4.4B**; **Table 4.4**, *Supplementary Materials*). For individuals high in trait dominance, testosterone increased negative affect in anticipation of the stressor and remained elevated after the stressor compared to placebo (Time x T/P: *B* = 0.098, *95%CI*[0.013, 0.182]; **Table 4.5**, *Supplementary Materials*). But for individuals low in trait dominance, testosterone did not significantly increase negative affect compared to placebo (95%CIs contain zero). Thus, testosterone enhances negative affect in response to a forthcoming social stressor and sustains this negative response to the stressor for individuals high in trait dominance.

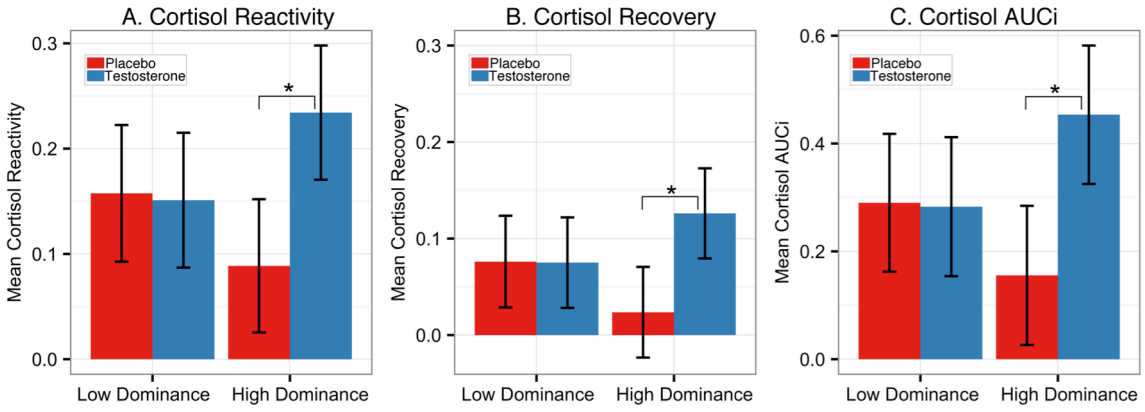

Figure 4.3: Follow-up analyses of cortisol response to social-evaluative stress. All values are estimated marginal means from relevant models and all error bars are 95% confidence intervals. Panel A: Cortisol reactivity, calculated by subtracting cortisol levels at baseline from cortisol levels 20 minutes after the end of the social-evaluative stressor. Panel B: Cortisol recovery, calculated by subtracting baseline cortisol levels from cortisol levels 40 minutes after the stressor. Panel C: Cortisol AUCi.

Follow-up analyses revealed that the effects of testosterone among high-dominance men were also seen on two specific subscales of negative affect, fear and hostility (**Table 4.6, Figure 4.8**). Trait prestige levels were not found to moderate the effect of testosterone on negative affect in response to stress (**Table 4.7**).

Positive affect decreased in anticipation of and in response to the social-evaluative stressor (Time: B = -0.437, *95%CI*[-0.503, -0.371]), but neither testosterone nor the interaction of testosterone and trait dominance moderated this effect (all 95%CIs contain zero; **Table 4.8**).

Behavioral Response

The behavioral variables were submitted to a confirmatory factor analysis with promax rotation that revealed good fit across the three *a priori* factors (68% of variance was explained by the three factors). As with previous work (13), the performance variables (e.g., "How good was this interview?") loaded heavily on the competence

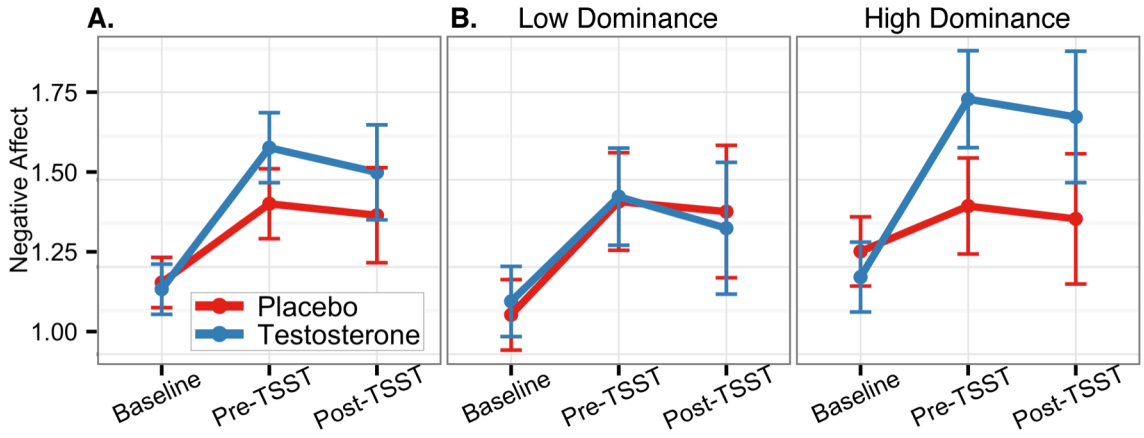

Figure 4.4: Negative affect response to social-evaluative stress. All values are estimated marginal means from relevant models and all error bars are 95% confidence intervals. A. Time x T/P on negative affect. B. Time x T/P effect on negative affect graphed at ±1 SD trait dominance.

factor, which we therefore renamed "overall performance" (**Table 4.9**). Across the three factors, the raters demonstrated good reliability (average Cronbach's alpha = 0.71).

Testosterone did not causally alter behavioral responses to the social-evaluative stressor (Overall performance: B = -0.077, 95%CI[-0.222, 0.068]); Dominance: B = -0.007, 95%CI[-0.157, 0.144]; Warmth: B = -0.113, 95%CI[-0.266, 0.039]), though the effects were generally consistent with previous work showing testosterone related to reduced performance and reduced warmth (13). Trait dominance did not moderate the effects of testosterone on behavioral responses to the stressor (Overall performance: B = 0.023, 95%CI[-0.123, 0.168]; Dominance: B = 0.066, 95%CI[-0.085, 0.217]; Warmth: B = 0.094, 95%CI[-0.059, 0.247]).

Discussion

This study provides causal evidence that exogenous testosterone increases cortisol concentrations and negative affect in response to a social-evaluative stressor, especially for individuals with high trait dominance. For an individual high in trait dominance – who is already predisposed to being concerned with status and accustomed to wielding social or even physical force to obtain it – exogenous testosterone administration motivates strong concern for his status, making him vigilant for cues to potential threats. Indeed, testosterone increases neural and cardiovascular reactivity to threatening interpersonal cues that may signal an impending social challenge, like angry faces (35-39). This increased concern for status during a social evaluation may be driving the dominant, testosterone-administered participant to feel more negative affect and elicit a stronger physiological response to the stressor. The present work therefore advances theory on testosterone and social status (14) and challenges medical assumptions of

testosterone's stress-suppressant effects (9,16) by showing that testosterone's influence on susceptibility to status threat extends to acute social-evaluative stress. Future work that investigates the effects of testosterone on stress responses must (i) consider the social context in which the stressor exists and (ii) account for individual differences in relevant psychosocial constructs like trait dominance.

In addition to these psychosocial explanations, careful consideration must be given to the potential biological mechanisms by which testosterone increases stress responses. Testosterone has been linked to increased activity in brain areas sensitive to cues indicative of threatening situations, such as the amygdala (39). In animal research the amygdala is a key component of the neural regulation that promotes HPA responses to stress (40), with limited work suggesting it may influence human responses to social stressors as well (41). Testosterone is also associated with reduced connectivity within frontal-limbic neural circuitry, a pattern thought to indicate decreased neural regulation of affect and behavioral responses to threat (42,43). Although currently untested, an individual with high testosterone levels who is high in trait dominance in the midst of a social evaluation may therefore experience increased activation and reduced regulation of these neural threat responses as part of an exacerbated response to the stressor.

Testosterone did not significantly influence status-relevant behavioral responses to the stressor, although the directions of the effects were consistent with prior work showing that increased testosterone reactivity to a stressor mediated reduced performance during the stressor (13). These null results may also be explained by evidence indicating that testosterone correlates more robustly with status-seeking behaviors in unstable hierarchies than in stable hierarchies (44). In the present work, participants were asked to

perform a mock-job interview for a high status position, but this contrasts with prior work that explicitly manipulated social status and/or hierarchy stability via competitive outcomes (21) or unambiguous descriptions of hierarchy instability (13). In order to clarify testosterone's causal role in directing behavioral responses to social-evaluative stress, exogenous testosterone (vs. placebo) should be administered with an explicit manipulation of social status and hierarchy stability.

Although the present study was limited to male participants, future research must consider the extent to which the effects of testosterone and dominance on stress occur in women. Prior work has shown that neural responses to threat are similar for men and women given exogenous testosterone (35,39) and, more broadly, that the interactive effects of exogenous testosterone and trait dominance alter women's status-relevant behavior (21). But women also generally react less robustly to social-evaluative stress compared to men (16) and women's dominance motivations may be more dependent on other steroid sex hormones, like estradiol (50). Future work must therefore investigate what role testosterone and status-relevant individual differences play in altering responses to social-evaluative stressors in women.

These results have important implications for understanding testosterone's role in stress and health. Stress often precedes the onset of psychiatric conditions, like depression and substance use disorders (2,3). Individuals high in trait dominance with high testosterone levels may therefore be susceptible to stress-linked disorders due to an increased reactivity to social stress. In support of this inference, one large-scale study found that above average levels of testosterone predicted increased depressive symptomatology, though controlling for protective factors like marriage and employment

status attenuated this relationship (45). Similarly, dominance motivations have been theorized to share common etiology for externalizing psychopathologies with known links to stress exposure, such as drug and alcohol abuse (46). Although the present work provides initial evidence that testosterone and trait dominance influence stress responses with implications for mental health, future work must determine the exact causal pathways by which these biological and psychosocial factors affect the onset and course of mental health conditions.

In terms of physical health, recent evidence indicates that exogenous testosterone, increasingly prescribed to treat hypogonadism (17), may boost the risk of non-fatal heart attacks (47, but *cf.* 48 for alternative explanations of these findings). To date, none of this work has considered the potential ramifications testosterone may have for stress-related health conditions when considered within a psychosocial context. Our findings show that in response to a social stressor, testosterone increases cortisol levels and negative affect, which are both theorized pathways for downstream negative consequences of stress such as poor cardiovascular health (1,4-6) and increased risk for acute cardiac events such as a heart attack (49). It should be noted that an acute stress response in itself is not unhealthy. For example, cortisol and negative affect could provide metabolic energy and motivation to gain a high status position within the stressful social setting. But in the long term, over repeated stressors, testosterone's causal increase of these stress responses may represent a liability to health and well-being (1), particularly for dominant individuals. These results advocate strongly for the inclusion of psychosocial variables like trait dominance in future biomedical and clinical studies on the effects of testosterone on stress and stress-linked health outcomes.

Supplementary Materials

Supplementary Methods

Analytical Plan. In the main document, we discuss the basic analytical plan; here we report the full multilevel model and results. Thus the model for Time x T/P was analyzed as follows (Equation 4.1):

$$\text{Level I:} \quad Cortisol_{ij} = \beta_{0j} + \beta_{1j}Time_{ij} + \beta_{2j}Time_{ij}^2 + r_{ij}$$

$$\text{Level II:} \quad \beta_{0j} = \gamma_{00} + \gamma_{01}TP_j + \gamma_{02}Blinding_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}TP_j + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}TP_j + \mu_{2j}$$

Similarly, the Time x T/P x Dominance analyses consisted of the following model (Equation 4.2):

$$\text{Level I:} \quad Cortisol_{ij} = \beta_{0j} + \beta_{1j}Time_{ij} + \beta_{2j}Time_{ij}^2 + r_{ij}$$

$$\text{Level II:} \quad \beta_{0j} = \gamma_{00} + \gamma_{01}TP_j + \gamma_{02}Dominance_j + \gamma_{03}TP\ x\ Dominance_j + \gamma_{04}Blinding_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}TP_j + \gamma_{12}Dominance_j + \gamma_{13}TP\ x\ Dominance_j + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}TP_j + \gamma_{22}Dominance_j + \gamma_{23}TP\ x\ Dominance_j + \mu_{2j}$$

For each set of models, we also explored models that included the two, earlier cortisol samples. In these models, time consisted of six epochs that were polynomial contrasted up to a quartic comparison (i.e., Time^4).

Models for the affective responses to social-evaluative stress were similar to the models for cortisol, with random intercepts and random effects of linear time for each participant. For example, the Time x T/P x Dominance model consisted of the following (Equation 4.3):

$$\text{Level I:} \quad Affect_{ij} = \beta_{0j} + \beta_{1j}Time_{ij} + \beta_{2j}Time_{ij}^2 + r_{ij}$$

Level I: $\beta_{0j} = \gamma_{00} + \gamma_{01}TP_j + \gamma_{02}Dominance_j + \gamma_{03}TP\ x\ Dominance_j + \gamma_{04}Blinding_j + \mu_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}TP_j + \gamma_{12}Dominance_j + \gamma_{13}TP\ x\ Dominance_j + \mu_{1j}$

$\beta_{2j} = \gamma_{20} + \gamma_{21}TP_j + \gamma_{22}Dominance_j + \gamma_{23}TP\ x\ Dominance_j$

Separate models for positive and negative affect were analyzed for the main document. We explore the lower-level positive and negative subscales within these supplementary materials.

Supplementary Analyses

Supplementary Preliminary Analyses.

Full model estimates. We report estimates and confidence intervals of the interactions of interest in the main document; here we report the full results for each model of cortisol (**Table 4.1** and **4.2**) and affective responses to social-evaluative stress (**Tables 4.5, 4.6,** and **4.10**).

***Testosterone concentrations*.** Salivary testosterone concentrations were found to be non-normally distributed and, like cortisol concentrations, were submitted to square-root transformation. We did not expect baseline differences in testosterone levels between the testosterone and placebo groups prior to gel application; GLM testing of transformed testosterone concentrations confirmed testosterone concentrations were equivalent at baseline (T/P: B = 0.55, 95%CI[-0.34, 1.44]; see **Figure 4.7** for full-day testosterone concentrations).

Tests for baseline differences in self-report measures and cortisol. We examined if the T/P or blinding conditions or their interaction altered responses to the self-report measures via GLM analyses. Self-report trait dominance was not altered by T/P (B = -

0.008, 95%CI[-0.19, 0.18]), blinding (B = 0.097, 95%CI[-0.09, 0.28]), or the T/P x blinding interaction (B = -0.004, 95%CI[-0.19, 0.18]). Negative affect at baseline was not altered by testosterone administration (B = -0.009, 95%CI[-0.05, 0.03]), blinding (B = -0.010, 95%CI[-0.05, 0.03]), or the T/P x blinding interaction (B = -0.010, 95%CI[-0.05, 0.03]). Positive affect at baseline was not altered by T/P (B = 0.07, 95%CI[-0.05, 0.18]), blinding (B = 0.005, 95%CI[-0.11, 0.12]), or the T/P x Blinding interaction (B = -0.02, 95%CI[-0.14, 0.09]).

We also explored if cortisol differed just before the social-evaluative stressor due to testosterone treatment or blinding conditions. The pre-TSST cortisol sample was not altered by T/P (B = -0.005, 95%CI[-0.023, 0.014]), blinding, (B = 0.003, 95%CI[-0.015, 0.022]) or the T/P x blinding interaction (B = 0.002, 95%CI[-0.017, 0.021]).

Similarly, we used general linear regression models with T/P, blinding, and trait dominance to investigate if trait dominance and the T/P x Trait Dominance interaction predicted baseline (pre-TSST) cortisol or affect. We found that trait dominance and the T/P x Dominance interaction did not predict differences in pre-TSST cortisol ($B_{Dominance}$ = -0.006, 95%CI[-0.024, 0.013]; $B_{T/P \times Dom}$ = -0.005, 95%CI[-0.024, 0.014]) or positive affect ($B_{Dominance}$ = 0.076, 95%CI[-0.040, 0.191]; $B_{T/P \times Dom}$ = 0.059, 95%CI[-0.056, 0.174]). Trait dominance did relate to increased negative affect at baseline ($B_{Dominance}$ = 0.064, 95%CI[0.024, 0.104]), but did not interact with T/P ($B_{T/P \times Dom}$ = -0.028, 95%CI[-0.068, 0.012]).

Exploratory Analyses

Cortisol changes prior to the social-evaluative stressor. In keeping with prior research on acute stress responses (51), our primary analyses examined salivary cortisol

changes from immediately before the TSST to 0, 20, and 40 minutes after the TSST. Here we confirmed that T/P did not influence salivary cortisol changes across three pre-stressor samples: A basal sample collected soon after participants arrived in the laboratory, a sample collected approximately three hours after gel administration, and the Pre-TSST sample. Multilevel models revealed a main effect of time on cortisol measured before the TSST consistent with circadian decline (Time (linear): B = -0.123, 95%CI[-0.144, -0.101]), but there were no significant effects of T/P or T/P x trait dominance on salivary cortisol changes examined before the TSST. These results indicate that the effect of T/P can be attributed to cortisol responses to the social-evaluative stressor but not cortisol changes prior to the TSST.

Additional exploratory multilevel models provided evidence that testosterone marginally increased the cortisol response to social-evaluative stress even when including the all six samples, from across the full laboratory protocol ($Time^2$ x T/P: B = 0.016, 95%CI[-0.002, 0.034]; $Time^3$ x T/P: B= -0.021, 95%CI[-0.046, 0.004]). The interactive effects of testosterone and trait dominance were also found to impact cortisol responses when including the earlier samples (Time x T/P x Dominance: B = 0.032, 95%CI[0.003, 0.061]; $Time^3$ x T/P x Dominance: B = -0.035, 95%CI[-0.059, -0.011]). Visual inspection of the results supports the analyses in the main document, indicating that these differences were most readily apparent in the cortisol responses to the social-evaluative stressor (**Figures 4.5** and **4.6**).

Exploratory Analyses of Affect Subscales. The main document focused on the higher-order positive and general negative affect scales, but the PANAS-X also contains lower-order subscales that distinguish the specific affective content of the general

positive and negative mood states (52). Here we explore the interactive effects of testosterone and trait dominance on the lower-order subscales that make up general negative affect (fear, hostility, guilt, sadness) and general positive affect (joviality, self-assurance, attentiveness). Within the negative subscales, significant three-way Time x T/P x Trait Dominance interactions were found for fear and hostility, but not guilt or sadness (**Table 4.6**). Testosterone increased fear and hostility in anticipation of the social-evaluative stressor in high trait dominance participants; no differences between testosterone and placebo were found in the low trait dominance participants **(Figure 4.8**). These effects on fear suggest that the threat to one's status inherent in social-evaluative stress is accentuated in men high in trait dominance who were given exogenous testosterone. Further, the results related to hostile affect extend prior work on dominance, testosterone, and aggression (53) by showing that the interactive effects of trait dominance and exogenous testosterone impacts self-reported hostility. No Time x T/P x Trait Dominance effects were found for any of the lower-order positive affect subscales.

Prestige. The main document analyzes interactive effects of trait dominance and T/P in a Time x T/P x Dominance interaction. Here we show that trait dominance's complement, trait prestige, does not predict cortisol (**Table 4.3**) or negative affect responses (**Table 4.7**) to the social-evaluative stressor. This is in keeping with prior work suggesting that trait dominance, and not prestige, is associated with enhanced responses to status-threatening situations (54).

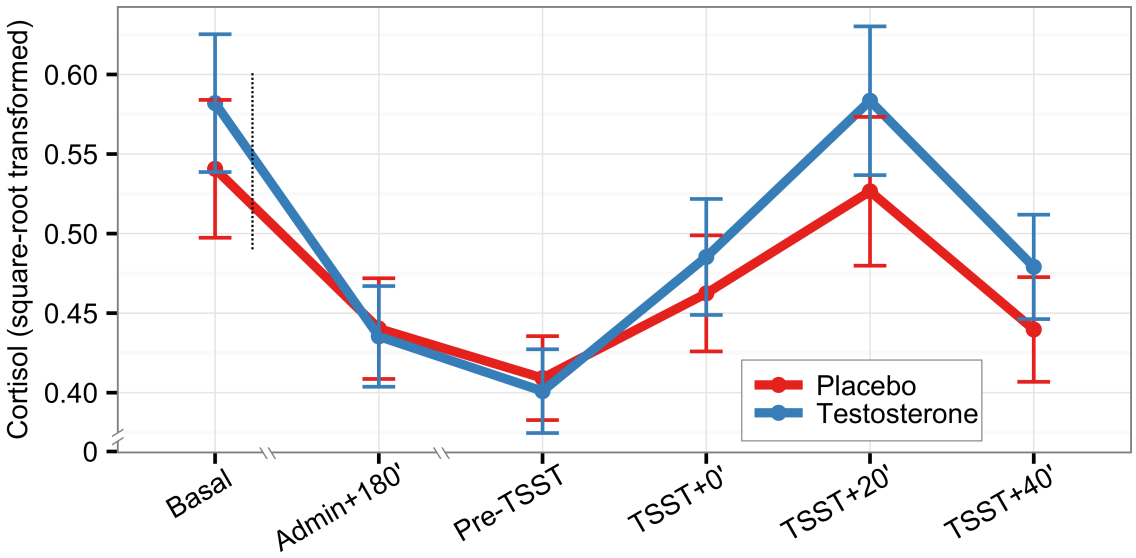

Fig. 4.5

Estimated marginal means of cortisol concentrations across lab day. The dashed line represents gel administration. The second saliva sample, "Admin+180,'" was collected 3 hours after gel administration. Error bars are 95%CIs.

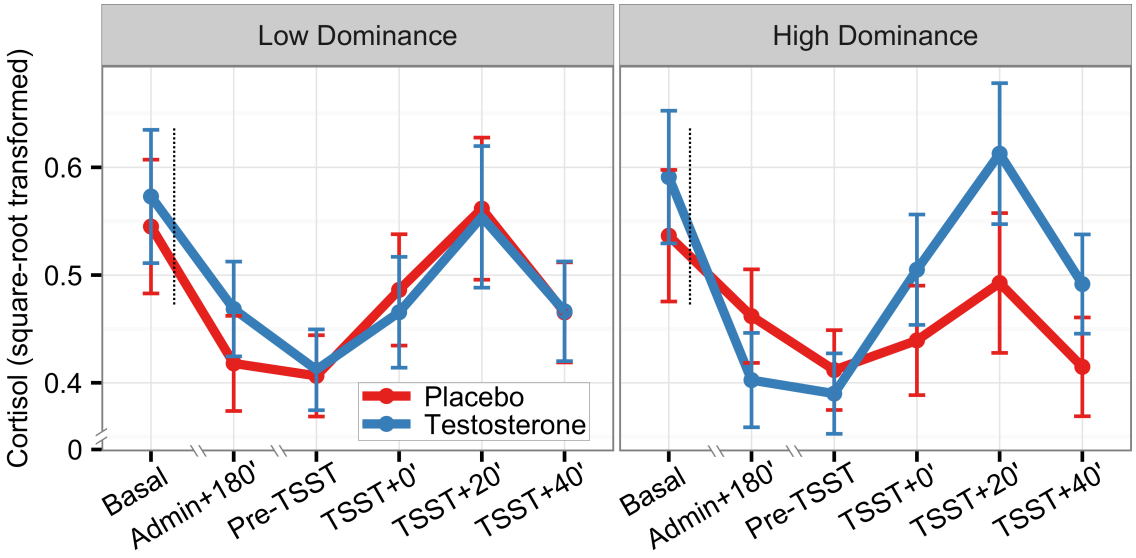

Fig. 4.6

Estimated marginal means of cortisol concentrations across lab day plotted at +/-1SD of trait dominance. The dashed line represents gel administration. The second saliva sample, "Admin+180,'" was collected 3 hours after gel administration. Error bars are 95%CIs.

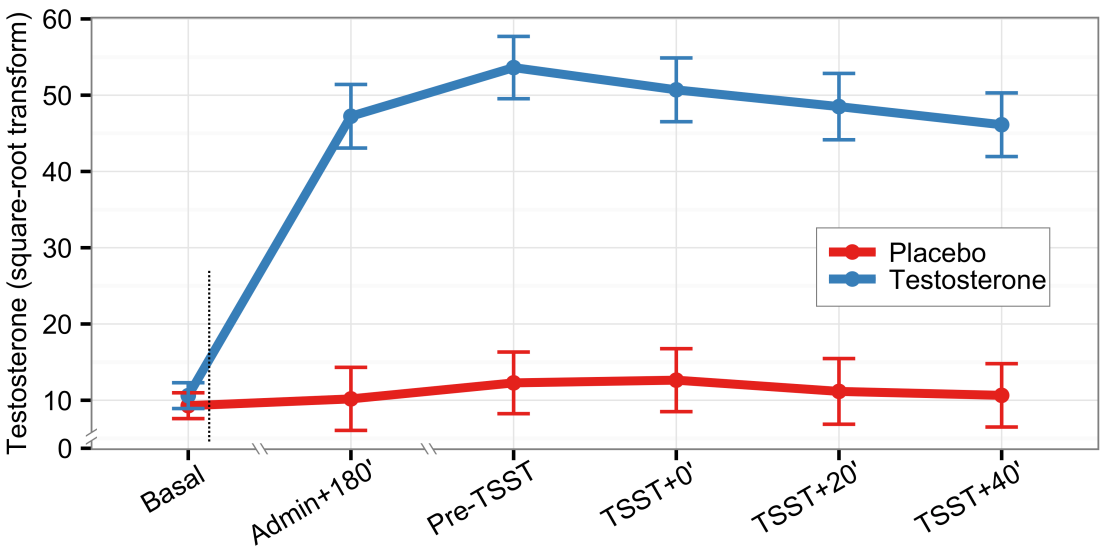

Fig. 4.7

Estimated marginal means of testosterone concentrations after exogenous testosterone or placebo application. The dashed line represents gel administration. The second saliva sample, "Admin+180,'" was collected 3 hours after gel administration. Error bars are 95%CIs.

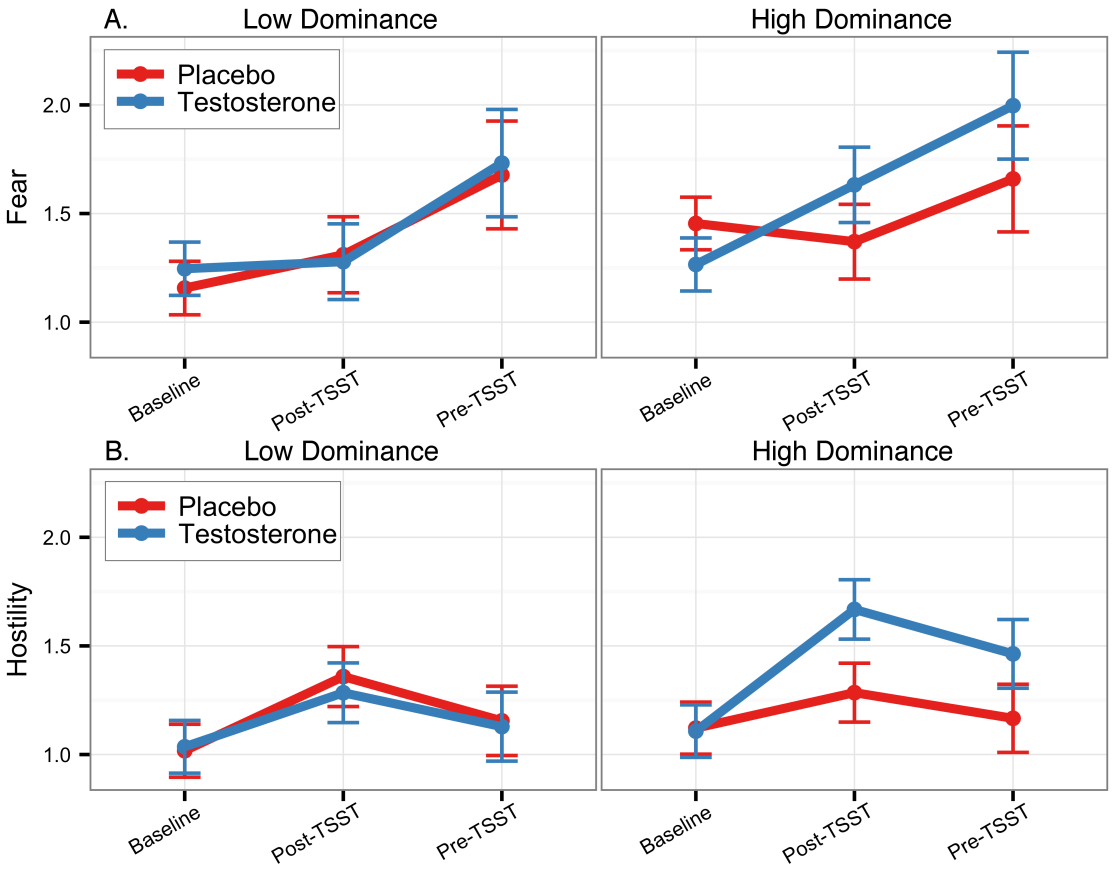

Fig. 4.8

Estimated marginal means from exploratory analyses of the Time x T/P x Dominance effects on the fear and hostility subscales of the PANAS-X. "Pre-TSST" was measured after giving instructions for the social-evaluative stress task but before beginning the task. Error bars represent 95%CI. Panel A. Fear subscale. Panel B. Hostility subscale.

Table 4.1.

Time x T/P (Model 1) and Time x T/P x Dominance (Model 2) effects on cortisol response from four time points (pre-stressor and +0, +20, +40 minutes after stressor).

	Model 1			Model 2		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	0.471	0.450 – 0.493	**<.001**	0.472	0.450 – 0.493	**<.001**
Time (linear)	0.063	0.044 – 0.081	**<.001**	0.063	0.045 – 0.081	**<.001**
$Time^2$ (quad.)	-0.096	-0.114 – -0.078	**<.001**	-0.096	-0.114 – -0.078	**<.001**
T/P	0.014	-0.008 – 0.035	.216	0.014	-0.008 – 0.035	.222
Blinding	-0.000	-0.016 – 0.016	.965	0.000	-0.016 – 0.017	.971
Time x T/P	0.020	0.001 – 0.038	**.038**	0.020	0.001 – 0.038	**.037**
$Time^2$ x T/P	-0.013	-0.031 – 0.005	.163	-0.013	-0.031 – 0.005	.164
Dominance				-0.004	-0.026 – 0.017	.697
Time x Dominance				-0.001	-0.020 – 0.017	.877
$Time^2$ x Dominance				-0.003	-0.021 – 0.014	.710
T/P x Dominance				0.016	-0.005 – 0.038	.141
Time x T/P x Dominance				0.020	0.002 – 0.038	**.035**
$Time^2$ x T/P x Dominance				-0.021	-0.039 – -0.003	**.023**
Random Parts						
N_{SUBID}		116			116	
ICC_{SUBID}		0.805			0.804	
Observations		462			462	
R^2 / Ω_0^2		.922 / .918			.922 / .918	

Table 4.2.

Effects of Time x T/P on the cortisol response (pre-stressor and +0, +20, +40 minutes after stressor) to social evaluative stress when analyzed in subsamples, split by median trait dominance.

	Low Dominance			High Dominance		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	0.466	0.435 – 0.498	**<.001**	0.476	0.445 – 0.507	**<.001**
Time (linear)	0.063	0.038 – 0.089	**<.001**	0.065	0.039 – 0.090	**<.001**
$Time^2$ (quad.)	-0.091	-0.116 – -0.067	**<.001**	-0.102	-0.129 – -0.076	**<.001**
T/P	-0.001	-0.032 – 0.030	.957	0.029	-0.002 – 0.060	.071
Blinding	-0.009	-0.033 – 0.014	.435	0.008	-0.015 – 0.030	.507
Time x T/P	-0.005	-0.031 – 0.020	.688	0.044	0.018 – 0.070	**.001**
$Time^2$ x T/P	0.008	-0.017 – 0.032	.534	-0.034	-0.060 – -0.007	**.015**
Random Parts						
N_{SUBID}		59			57	
ICC_{SUBID}		0.789			0.829	
Observations		235			227	
R^2 / Ω_0^2		.915 / .911			.930 / .927	

Table 4.3.
Full model results for interactive effects of time, T/P, and trait prestige on the cortisol response to social-evaluative stress.

	Prestige Model		
	B	*CI*	*p*
Fixed Parts			
(Intercept)	0.472	0.45 – 0.49	**<.001**
Time (linear)	0.063	0.04 – 0.08	**<.001**
$Time^2$ (quad.)	-0.096	-0.11 – -0.08	**<.001**
TP	0.013	-0.01 – 0.04	.231
Prestige	-0.003	-0.03 – 0.02	.783
Blinding	-0.001	-0.02 – 0.02	.938
Time x TP	0.020	0.00 – 0.04	**.038**
$Time^2$ x TP	-0.013	-0.03 – 0.01	.164
Time x Prestige	0.003	-0.02 – 0.02	.746
$Time^2$ x Prestige	-0.003	-0.02 – 0.02	.774
TP x Prestige	0.001	-0.02 – 0.02	.949
Time x TP x Prestige	0.007	-0.01 – 0.03	.503
$Time^2$ x TP x Prestige	-0.008	-0.03 – 0.01	.392
Random Parts			
N_{SUBID}		116	
ICC_{SUBID}		0.807	
Observations		462	
R^2 / Ω_0^2		.922 / .918	

Table 4.4.
Time x T/P (Model 1) and Time x T/P x Dominance (Model 2) effects on negative affect in response to social evaluative stressor.

	Model 1			Model 2		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	1.389	1.328 – 1.450	**<.001**	1.388	1.329 – 1.448	**<.001**
Time (linear)	0.187	0.126 – 0.247	**<.001**	0.187	0.127 – 0.246	**<.001**
$Time^2$ (quad.)	-0.150	-0.194 – -0.106	**<.001**	-0.150	-0.193 – -0.107	**<.001**
T/P	0.044	-0.017 – 0.105	.158	0.044	-0.015 – 0.103	.149
Blinding	-0.001	-0.048 – 0.046	.976	-0.007	-0.053 – 0.040	.775
Time x T/P	0.050	-0.010 – 0.111	.107	0.049	-0.010 – 0.109	.106
$Time^2$ x T/P	-0.044	-0.088 – -0.001	**.048**	-0.044	-0.087 – -0.001	**.047**
Dominance				0.068	0.009 – 0.127	**.027**
Time x Dominance				0.008	-0.051 – 0.067	.787
$Time^2$ x Dominance				0.002	-0.041 – 0.045	.937
T/P x Dominance				0.043	-0.016 – 0.102	.154
Time x T/P x Dominance				0.080	0.021 – 0.139	**.009**
$Time^2$ x T/P x Dominance				-0.036	-0.079 – 0.007	.099
Random Parts						
N_{SUBID}		116			116	
ICC_{SUBID}		0.578			0.581	
Observations		348			348	
R^2 / Ω_0^2		.764 / .748			.781 / .768	

Table 4.5.

Effects of Time x T/P on negative affect in response to social evaluative stress when analyzed in subsamples, split by median trait dominance.

	Low Dominance			High Dominance		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	1.346	1.260 – 1.432	**<.001**	1.430	1.343 – 1.517	**<.001**
Time (linear)	0.177	0.090 – 0.264	**<.001**	0.200	0.116 – 0.285	**<.001**
$Time^2$ (quad.)	-0.163	-0.222 – -0.103	**<.001**	-0.137	-0.201 – -0.072	**<.001**
T/P	0.036	-0.050 – 0.121	.419	0.057	-0.030 – 0.144	.205
Blinding	-0.010	-0.074 – 0.054	.760	-0.001	-0.072 – 0.069	.972
Time x T/P	0.002	-0.085 – 0.089	.967	0.098	0.013 – 0.182	**.026**
$Time^2$ x T/P	-0.055	-0.114 – 0.004	.070	-0.033	-0.097 – 0.032	.322
Random Parts						
N_{SUBID}		59			57	
ICC_{SUBID}		0.547			0.636	
Observations		177			171	
R^2 / Ω_0^2		.738 / .718			.810 / .800	

Table 4.6.
Full model results for interactive effects of time, T/P, and trait dominance on the specific negative affect subscales, fear and hostility.

	Fear			Hostility		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	1.482	1.409 – 1.555	**<.001**	1.233	1.181 – 1.284	**<.001**
Time (linear)	0.344	0.261 – 0.426	**<.001**	0.112	0.052 – 0.171	**<.001**
$Time^2$ (quad.)	0.103	0.046 – 0.159	**<.001**	-0.204	-0.259 – -0.149	**<.001**
T/P	0.043	-0.030 – 0.117	.246	0.049	-0.003 – 0.100	.067
Blinding	0.081	0.008 – 0.154	**.031**	0.069	0.018 – 0.121	**.009**
Time x T/P	-0.033	-0.088 – 0.023	.248	-0.002	-0.051 – 0.048	.949
$Time^2$ x T/P	0.087	0.004 – 0.170	**.042**	0.047	-0.012 – 0.106	.122
Dominance	-0.017	-0.074 – 0.039	.553	-0.035	-0.090 – 0.020	.213
Time x Dominance	-0.012	-0.095 – 0.070	.768	0.030	-0.029 – 0.089	.318
$Time^2$ x Dominance	-0.027	-0.083 – 0.029	.348	-0.010	-0.064 – 0.045	.724
T/P x Dominance	0.025	-0.048 – 0.098	.504	0.062	0.011 – 0.114	**.019**
Time x T/P x Dominance	0.099	0.016 – 0.182	**.020**	0.063	0.004 – 0.122	**.038**
$Time^2$ x T/P x Dominance	-0.059	-0.116 – -0.003	**.040**	-0.064	-0.118 – -0.009	**.023**
Random Parts						
N_{SUBID}		116			116	
ICC_{SUBID}		0.454			0.502	
Observations		348			348	
R^2 / Ω_0^2		.696 / .672			.771 / .756	

Table 4.7. Full model results for interactive effects of time, T/P, and trait prestige on the negative affect response to social-evaluative stress.

	Prestige Model		
	B	*CI*	*p*
Fixed Parts			
(Intercept)	1.399	1.34 – 1.46	**<.001**
Time (linear)	0.181	0.12 – 0.24	**<.001**
$Time^2$ (quad.)	-0.150	-0.20 – -0.10	**<.001**
TP	0.036	-0.03 – 0.10	.267
Prestige	-0.072	-0.14 – -0.01	**.031**
Blinding	-0.001	-0.05 – 0.05	.977
Time x TP	0.057	-0.00 – 0.12	.073
$Time^2$ x TP	-0.046	-0.09 – 0.00	.063
Time x Prestige	-0.025	-0.09 – 0.04	.447
$Time^2$ x Prestige	0.070	0.02 – 0.12	**.006**
TP x Prestige	0.036	-0.03 – 0.10	.270
Time x TP x Prestige	0.011	-0.05 – 0.07	.727
$Time^2$ x TP x Prestige	0.007	-0.04 – 0.06	.783
Random Parts			
N_{SUBID}		116	
ICC_{SUBID}		0.572	
Observations		348	
R^2 / Ω_0^2		.772 / .757	

Table 4.8. Time x T/P (Model 1) and Time x T/P x Dominance (Model 2) effects on positive affect in response to social evaluative stressor.

	Model 1			Model 2		
	B	*CI*	*p*	*B*	*CI*	*p*
Fixed Parts						
(Intercept)	2.155	2.056 – 2.254	**<.001**	2.155	2.057 – 2.254	**<.001**
Time (linear)	-0.437	-0.503 – -0.371	**<.001**	-0.437	-0.503 – -0.371	**<.001**
$Time^2$ (quad.)	0.028	-0.027 – 0.083	.324	0.027	-0.028 – 0.082	.332
T/P	0.039	-0.060 – 0.138	.443	0.038	-0.060 – 0.137	.445
Blinding	0.042	-0.057 – 0.141	.405	0.037	-0.061 – 0.136	.459
Time x T/P	-0.012	-0.077 – 0.054	.730	-0.012	-0.078 – 0.055	.731
$Time^2$ x T/P	0.046	-0.010 – 0.101	.110	0.046	-0.009 – 0.101	.103
Dominance				0.052	-0.046 – 0.151	.302
Time x Dominance				-0.002	-0.068 – 0.064	.945
$Time^2$ x Dominance				0.045	-0.009 – 0.100	.107
T/P x Dominance				0.074	-0.024 – 0.172	.142
Time x T/P x Dominance				0.004	-0.062 – 0.070	.903
$Time^2$ x T/P x Dominance				-0.030	-0.084 – 0.025	.286
Random Parts						
N_{SUBID}		116			116	
ICC_{SUBID}		0.743			0.744	
Observations		347			347	
R^2 / Ω_0^2		.883 / .874			.889 / .879	

Table 4.9. Factor loadings for behavioral variables. Four additional variables were rated but did not satisfactorily load on any one factor: Fidgety (with hands, body); Fidgety (with equipment); Creative; and Fast talker. Three variables were cross-loaded and so were left out of analyses: Angry (Performance & Dominance); Warm (Performance & Warmth); and In control (Performance & Dominance).

	Overall Performance	Dominance	Warmth	Cronbach's α
Competent	**0.92**	0.15	-0.23	0.80
Intelligent	**0.91**	0.02	-0.11	0.67
How good?	**0.89**	0.21	-0.16	0.85
Would you hire?	**0.86**	0.23	-0.16	0.83
Engaged	**0.86**	0.02	0.13	0.75
Coherent	**0.79**	0.24	-0.14	0.71
Bored	**-0.73**	0.13	-0.37	0.63
Likeable	**0.68**	0.09	0.21	0.57
Persuasive	**0.62**	0.46	-0.05	0.77
Maintained Eye Contact	**0.56**	0.14	-0.18	0.56
High Status	-0.1	**0.89**	0.18	0.68
Follower-like	-0.02	**-0.83**	-0.11	0.63
Powerful	0.25	**0.77**	-0.07	0.76
Dominant (in appearance)	0.06	**0.76**	-0.06	0.72
Dominant (sounding)	0.15	**0.75**	0.13	0.81
Nervous	-0.06	**-0.73**	-0.16	0.57
Stressed	-0.08	**-0.7**	-0.08	0.71
Stumbling over words	0.11	**-0.69**	-0.04	0.66
Confident	0.31	**0.66**	0.2	0.79
Awkward	-0.39	**-0.64**	0.16	0.63
Strong Posture	0.21	**0.62**	-0.08	0.71
Leader-like	0.41	**0.61**	0.1	0.75
Quiet	-0.15	**-0.53**	-0.36	0.77
Smiley/Smiling	-0.37	0.13	**0.95**	0.88
Humorous	-0.46	0.31	**0.87**	0.69
Happy	0.01	0.18	**0.82**	0.72
Friendly	0.46	-0.04	**0.66**	0.67

CHAPTER V

TESTOSTERONE, STRESS, AND MOTIVATION

The previous chapter provides evidence for testosterone's causal effects on the cortisol and affect responses to stress. Testosterone treatment caused increased cortisol reactivity and increased negative affect in anticipation of and in response to the social-evaluative stressor, especially for men high in trait dominance.

The next chapter examines the same interaction as the previous chapter (testosterone treatment and trait dominance), but extends this work to motivated persistence in a physical task. Motivation – a state of goal-directed cognition and behavior that underlies pursuit of reward or avoidance of punishment (1) – underlies the ability to succeed and persevere through stressful encounters (2). The next chapter also advances the previous chapter by examining if social-evaluative stress impacts the effects of testosterone and trait dominance on motivation. Stress has been linked to disrupted goal pursuit and demotivation (3). Given the previous chapter's findings showing that testosterone and high trait dominance related to increased stress reactivity, I examine if a social-evaluative stressor detrimentally impacts motivation more so for high trait dominance individuals with high testosterone levels. I also examine the extent to which stress reactivity (i.e., cortisol and negative affect) mediates stress-linked changes in motivation.

CHAPTER VI

GET A GRIP: TESTOSTERONE AND TRAIT DOMINANCE INTERACTIVELY ALTER MOTIVATED PERSISTENCE ON A HANDGRIP ENDURANCE TASK

Introduction

In many aspects of life, motivated persistence is required for sustained and proactive approach towards goals, which can lead to success even amidst stressful, difficult circumstances (1). Motivation is defined by the prospect of reward or the fear of punishment, either of which provides orientation towards a goal and can evoke behavioral responses that allow an individual to persist towards those goals (2).

Research in animals and humans indicates testosterone might be a biological substrate of this goal-directed motivation. Administering exogenous testosterone in chicks and rodents increases persistent behavior directed towards finding food (3-5) and social interactions (6). In research on humans, testosterone is broadly linked to increased motivations to gain or maintain social status (7), for example through increased persistence in pursuing victories after losing in competitions (8). More specifically, one study has shown that basal testosterone levels positively correlate with perseverance on an unsolvable puzzle task; testosterone reactivity to a competition did not relate to perseverance in the same study (9). Testosterone therefore might influence lower level processes like motivation that underlie pursuit of social status (7), though the causal effects of testosterone are unknown. Examining testosterone's causal effects on motivation may provide new insights into the biological underpinnings that direct motivated behaviors, like exercising (10) or abstaining from addictive substances (11), which may have real impact on health.

In the present study, we test the causal effects of testosterone on motivated persistence in a handgrip task that measures psychological motivation to persevere in pursuit of a physical goal (12). Exogenous testosterone previously has been found to increase handgrip strength after several months of administration in a sample of older men (13)[5]. But handgrip strength alone is not sufficient to successfully persevere on the handgrip task: Psychological motivation to overcome fatigue or soreness and resist quitting likely underlies persistence on the handgrip task (12). Thus the present study examines the extent to which testosterone's effects on motivation generalizes to a handgrip task where the goal is to persist for as long as possible. The physical nature of the task also aligns closely with common goals for better health like exercising and therefore provides a face-valid measure of the motivations that may underlie persevering in pursuit of physical goals.

Prior work indicates that status-relevant individual difference factors moderate testosterone's effects on behavior. In particular, trait dominance – the degree to which an individual prefers to gain social rank through fear, intimidation, or force (17) – may be an important individual difference factor that moderates the effects of testosterone on motivation. Self-report trait dominance is an explicit marker of concern for status that heightens testosterone's effects on implicit motivations to pursue status, producing increased behavioral responses to status-relevant situations (18). For example, men with high testosterone who were high in trait dominance behaved more dominantly in an

[5] *cf.* 14 for evidence of a negative relationship between endogenous testosterone and handgrip strength that is moderated by endogenous cortisol levels, and *cf.* 15 for evidence that prenatal androgen exposure positively correlates with handgrip strength but not persistence on the handgrip task in men. Further, the measure of prenatal exposure to testosterone (the 2D:4D digit ratio) has not been found to correlate with testosterone levels (16).

interaction with a potential sexual competitor (18). Similarly, individuals given exogenous testosterone acted more aggressively (19) and behaved more competitively after winning (and less competitively after losing; 20), but only at higher levels of trait dominance. Trait dominance has also been linked to increased handgrip strength (21,22) and has been hypothesized to moderate the otherwise null main effects of endogenous testosterone reactivity on motivated persistence (9). But the effects of trait dominance on motivated persistence and its interaction with testosterone have not been investigated.

Finally, this study will examine the interactive effects of exogenous testosterone and dominance on changes in motivation in response to a social-evaluative stressor. Acute stress has been found to disrupt goal pursuit, leading to reduced motivation on a decision making task (23). In other work (from which the present report's data was collected), high trait dominance individuals given exogenous testosterone were more reactive to a social-evaluative stressor, leading to increased cortisol levels and increased negative affect (24). The effects of the stressor might extend to the handgrip task as well, potentially disrupting subsequent motivation and performance on the physical task. This study explores cortisol and negative affective responses to the social-evaluative stressor as putative mediators of the interactive effects of testosterone and trait dominance on stress-linked changes in motivation. In sum, this study examines changes in motivation on the handgrip task across three time points – from baseline to several hours after gel application, and shortly after a social-evaluative stressor to examine changes linked to stress exposure – and explores the extent to which stress responses mediate these changes in motivation.

Methods and Materials

Protocol

The handgrip persistence task was executed within a larger testosterone administration project reported elsewhere (24; Figure 6.1). Briefly, men (n = 120) aged 18 – 40 (Mean: 22.50 years; SE: 0.33) were recruited for a 6-hour laboratory session. All participants were screened for medical conditions prior to scheduling a laboratory session that started between 9:00 and 11:00 AM. Upon entering the laboratory, experimenters situated the participant in an individual testing room and obtained informed consent from the participants. A blunted-tip syringe containing either a 150-mg dose of testosterone (Androgel, AbbeVie, North Chicago, IL) or placebo gel was given to the participants to self-administer under the supervision of the experimenter. The placebo gel was made from the same inactive ingredients as the testosterone gel; lacking testosterone was the only difference between the two gels. The gel was allowed to absorb into the body for 3 hours while the participant filled in baseline questionnaires. The University of Oregon's Institutional Review Board approved all methods.

Blinding. Participants were randomly assigned to either a single-blind condition, in which the participant was told what gel he received, or a double-blind condition, in which the participant was merely told he had an equal chance of receiving testosterone or placebo. The experimenter never knew which gel was administered. Blinding was manipulated in order to measure and control for the expectation biases of assuming one had received testosterone or placebo (25). All analyses control for blinding.

Pharmacological Manipulation. As described elsewhere (24), the testosterone dose and the timing of the research protocol was based on prior topical testosterone administration research that showed that peak serum concentrations occurred approximately 3 hours after a 150-mg testosterone gel dose (26), with physiological effects and neural reactivity 3-6 hours after testosterone administration (27, 28). In order to complete this protocol during peak concentrations and also within a 6-hour time period, this project utilized a 150-mg dose of testosterone approximately three hours prior to data collection portions of the study, and approximately four hours (Mean = 3.98 hours, SE = 0.015 hours) prior to the social-evaluative stressor.

Handgrip Protocol. Immediately after gel administration, participants were asked to perform the handgrip task as a baseline measure. This baseline assessment was performed immediately after gel application due to time constraints on the laboratory day. Given previous work on the pharmacokinetics of topical testosterone application (e.g., 26), testosterone vs. placebo administration is unlikely to cause any differences so soon after administration. The handgrip task was repeated approximately three hours after gel administration (Mean = 175.2 minutes; SE = 5.5 minutes) and approximately thirty

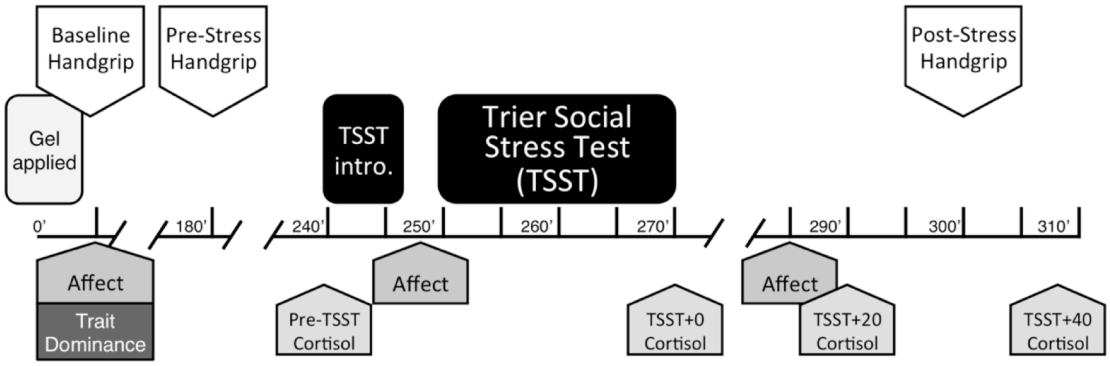

Figure 6.1. Study protocol timeline.

minutes after the end of the social-evaluative stressor (Mean = 29.7 minutes; SE = 1.1 minutes), which is equivalent to approximately five hours after gel administration (Mean = 294.0 minutes, SE = 1.5 minutes).

In the handgrip task, participants were asked to squeeze a small marble between a foam-padded handgrip with their dominant hand, keeping the ball aloft for as long as they were able. Participants were filmed via a small digital camera on a tripod. Prior to the baseline trial of the handgrip task, participants were read the following statement (see Supplementary Materials for full instructions):

> Handgrips are usually used to strengthen one's forearms and hands. In research, handgrips are used to determine how much willpower and persistence an individual is able to exert to achieve a desired goal. In today's study, we are going to video record you as you squeeze the handgrip…The videos will later be viewed by trained researchers in order to determine how long you were able to keep the marble aloft – essentially a measure of how good you were at this task.

These instructions were written so that participants were motivated to exert themselves to persevere to achieve a goal. The social-evaluative components – specifically directing the participant's attention to the camera and mentioning that others would watch and review the tapes and compare the participant to other participants – were included to provide greater incentive to perform well on the task.

To begin the task, the experimenter or participant held the ball in place while the participant squeezed the handgrip in his dominant hand. The task was considered completed when the ball fell from the handgrip due to the participant declining to continue the task. To measure performance on the task, a trained research assistant measured the duration for which the participant was able to keep the marble aloft between the handles of the handgrip. To check reliability of the trained research assistant,

the lead author watched 10% of the videos (n = 36) selected at random and measured duration independently (Cronbach's $\alpha = .999$).

Social-evaluative stressor. Social-evaluative stress was induced via the Trier Social Stress Test, a well-validated social stressor paradigm (29). Participants were given ten-minutes to prepare a five-minute speech as part of a mock job interview for a managerial position on campus. After the speech, participants were asked to count backwards, out loud by 13 from a large four-digit number; if a mistake occurred, participants were asked to begin again from the initial value. Unbeknownst to the participants, the two-panelists, acting as the selection committee, were trained to maintain neutral facial affect and to refrain from any positive verbal or nonverbal feedback for the participant.

Cortisol response to stress. Saliva was collected at six time points throughout the laboratory protocol. Four of these saliva samples (once immediately before and +0, +20, and +40 minutes after the TSST) were assayed for cortisol concentration to provide an endocrine biomarker of the response to social-evaluative stress. The two other saliva samples – at the start of the laboratory session and approximately three hours after gel application – are not analyzed for cortisol here (see 24 for more details). Approximately 2 mL of saliva was collected per sample and was assayed in our laboratory using commercially available enzyme immunoassay kits (DRG, Germany). The average intra-assay coefficients of variation (CVs) were 4.68% and the inter-assay CV was 14.8% averaged across low and high control samples. To index cortisol responses to the stressor, the area under the curve with respect to increase (AUC_I) was calculated as a measure of exposure to cortisol that emphasizes changes in concentration overtime (30).

Testosterone Concentrations. To ensure the testosterone treatment increased testosterone levels compared to placebo treatment, we examined testosterone levels in the five saliva samples that followed gel application. Assays were run in our laboratory with commercially available salivary testosterone kits (intra-assay CV, for samples within range = 6.55%; inter-assay CV = 16.1%). In instances where exogenous testosterone treatment resulted in testosterone levels too high for the EIA kits (34.4% of samples within the exogenous testosterone group, 17% of all samples), the kit's maximum value (5250 pg/mL) was substituted as a conservative estimate of the actual concentration. If one of the two duplicate samples was within the kit's range, we calculated the average of that known value and the 5250 pg/mL maximum.

Questionnaires. As described elsewhere (24), trait dominance was determined from the Dominance and Prestige Scale (17). Across 17 items, this scale measures the extent to which status is obtained via force, fear, and intimidation (e.g., "I try to control others rather than permit them to control me.") vs. appearing competent, using adept social skills, or via respect (e.g., "Members of my peer group respect and admire me.") on a scale from 1 (not at all) to 7 (very much).

The affect responses to the social-evaluative stressor were determined from the Positive and Negative Affect Schedule – Extended Form (PANAS-X; 31), which consists of two, broad scales of general positive (average Cronbach's $\alpha = 0.84$) and general negative affect (average Cronbach's $\alpha = 0.89$) answered on a scale from 1 (not at all or very little) to 4 (quite a bit). In the present report, we focus on the stress-linked responses found in prior work (24) that showed that the interactive effects of exogenous

testosterone and trait dominance altered general negative affect and two of its subscales, fear and hostility.

Analytical Plan

Multilevel model construction. In order to examine the interactive effects of testosterone and dominance on motivation, multilevel models were constructed that test for Time (three levels: Baseline, Pre-Stress, & Post-Stress) x Testosterone/Placebo (T/P) x Dominance cross-level interactions on handgrip task duration. This model used two Helmert contrast codes for the effect of time: The first contrast compares handgrip duration at baseline to the pooled effect of the two handgrip durations after gel application; the second contrast compares the pre-stress handgrip duration to the post-stress handgrip duration. This analytical strategy allows for a conservative test of the effects of testosterone from baseline to the two samples that occurred several hours after gel (Contrast 1) and allows examination of changes in motivation from pre- to post-stress, controlling for baseline motivation (Contrast 2; see Supplementary Materials for analyses with an alternative contrast scheme). This resulted in a two-level model for individual *i* at time *j*:

Level I (Equation 1):

$$Duration_{ij} = \beta_{0j} + \beta_{1j} Time_{Base.v.PostGel_{ij}} + \beta_{2j} Time_{PreTSST.v.PostTSST_{ij}} + r_{ij}$$

Level II (Equations 2-4):

$$\beta_{0j} = \gamma_{00} + \gamma_{01} TP_j + \gamma_{02} Dominance_{ij} + \gamma_{03} TP\ x\ Dominance_j + \gamma_{04} Blinding_j + \mu_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11} TP_j + \gamma_{12} Dominance_j + \gamma_{13} TP\ x\ Dominance_j + \mu_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21} TP_j + \gamma_{22} Dominance_j + \gamma_{23} TP\ x\ Dominance_j$$

Since the primary goal of the present study was to examine the effects of testosterone on motivation, the baseline to post-gel contrast was modeled as a random effect by participant. To decompose significant interaction terms, follow-up analyses were conducted to examine simple slopes within testosterone and placebo treatment groups at ±1 standard deviation of trait dominance (32).

Indices of Moderated Mediation. This study also examines the extent to which changes in motivation on the endurance task are mechanistically explained by cortisol reactivity and negative affect responses to the social evaluative stressor. Indices of moderated mediation were tested to examine the extent to which cortisol and negative affect responses to the social-evaluative stressor mediated the interactive effects of testosterone and trait dominance (33). In separate models, cortisol AUC_I and stress-linked negative affect – measured as the mean of the anticipatory and recovery values of negative affect – were explored as mediators of the pre- to post-stress change in hand grip duration. Indices of moderated mediation were bootstrap bias corrected (N = 10,000 samples) and the 95% confidence intervals (95%CI) were examined to see if they included zero. All indices are reported in unstandardized units, per published recommendations (33). Baseline negative affect and baseline handgrip duration were co-varied on the stress-linked negative affect and change in pre- to post-stress handgrip duration, respectively.

Results

Preliminary Results

Five participants (n = 2 from testosterone group) did not successfully complete all portions of the laboratory day relevant to this report and one participant's performances

(from the placebo group) were not recorded due to technical errors, leaving n = 114 participants in total. Four additional participants (n = 3 from testosterone group) were missing videos from one of the three assessments due to experimenter error. These participants were left in the analyses as the multilevel models can generally account for this sporadic missing data. Durations on the handgrip task were slightly positively skewed and thus were square-root transformed to normalize the distribution.

Testosterone Manipulation Check. As a manipulation check, we examined mean testosterone concentration in the five saliva samples that occurred after gel administration. Application of the testosterone gel significantly increased testosterone concentration (*M* = 2959.9 pg/mL, *95%CI*[2472.5, 3447.3]) compared to the placebo group (*M*= 163.6, *95%CI*[121.8, 205.3]).

Test for Baseline Differences. As the baseline handgrip occurred immediately after gel application – but prior to any expected effects of testosterone application – we examined if motivation differed across treatment groups. As expected, assignment to testosterone vs. placebo treatment group did not alter baseline handgrip duration (T/P: B = -0.210, 95%CI[-0.611, 0.190]) and it did not interact with trait dominance (T/P x Dominance: B = 0.011, 95%CI[-0.391, 0.414]).

Effects of Testosterone and Dominance on Motivation

Main Effects of Testosterone Treatment. Within the multilevel model, testosterone treatment was not found to increase motivation on the handgrip task compared to placebo from baseline to the two post-gel-application samples ($Time_{Base\ v.\ Post\text{-}gel}$ x T/P: B = 0.063; 95%CI[-0.059, 0.185], p = 0.311). These results indicate testosterone does not cause significantly different motivated persistence compared to

placebo, though the weak but positive effect is in the expected the direction based on prior work (9). The main effect of testosterone treatment did not alter changes in motivation from pre- to post-stress ($Time_{Pre\text{- v. }Post\text{-}Stress}$ x T/P: B = -0.007, 95%CI[-0.191, 0.177], p = 0.940).

Main Effects of Trait Dominance. Next, based on previous work linking trait dominance to increased handgrip strength (21, 22), we used the multilevel model analyses to examine relationships between trait dominance and motivated persistence on the handgrip task. Trait dominance predicted significantly increased motivation on the handgrip task (Dominance: B = 0.328, 95%CI[0.012, 0.644]; Table S6.1). But the Time x Trait Dominance interactions indicated that trait dominance was not associated with changes in motivation across the multiple time points ($Time_{Base\text{ v. }Post\text{-}gel}$ x Dominance: B =0.052, 95%CI[-0.067, 0.172]; $Time_{Pre\text{- v. }Post\text{-}Stress}$ x Dominance: B = -0.129, 95%CI[-0.308, 0.049]).

Interactive Effects of Testosterone and Dominance. Examining the Time x T/P x Trait dominance interaction revealed significant differences in handgrip duration from baseline to the two handgrip assessments after gel application ($Time_{Base\text{ v. }Post\text{-}gel}$ x T/P x Dominance: B = 0.135; 95%CI[0.015, 0.254]) as well as from pre- to post-stress ($Time_{Pre\text{- v. }Post\text{-}Stress}$ x T/P x Dominance: B = -0.202, 95%CI[-0.380, -0.023]; Figure 6.2 and Table S6.1). Decomposing these interaction terms revealed that from baseline to the two post-gel-application assessments, testosterone increased motivated persistence but only for men high in trait dominance ($Time_{Base\text{ v. }Post\text{-}gel}$: B = 0.449, Z = 3.72, p < .001). For men low in trait dominance, testosterone had no effect from baseline to the combined post-gel assessments ($Time_{Base\text{ v. }Post\text{-}gel}$: B = 0.075, Z = 0.611, p = 0.54). In the placebo condition,

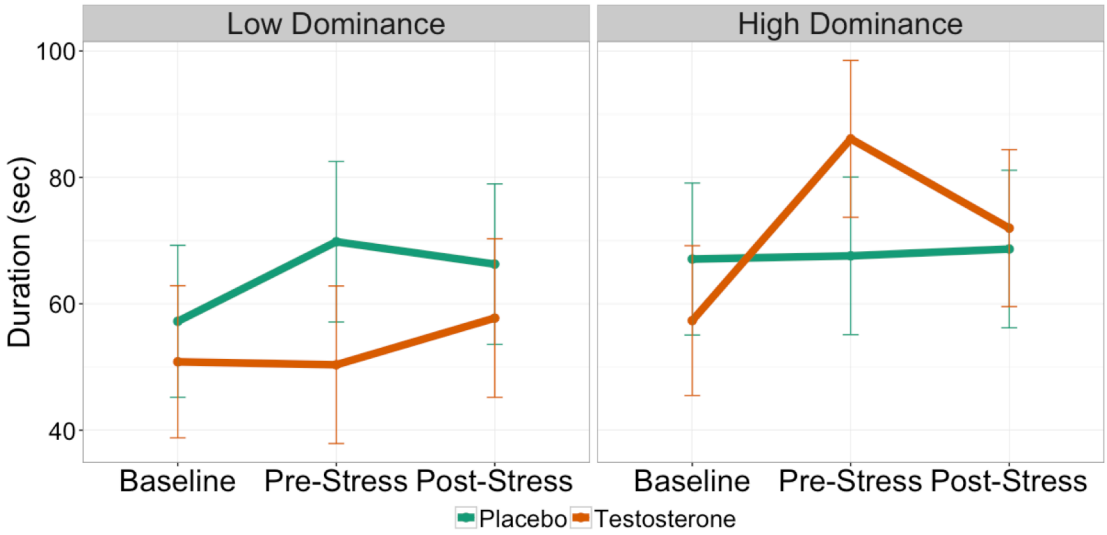

Figure 6.2. Effects of testosterone vs. placebo on motivated persistence are moderated by trait dominance. For purposes of clarity, this model shows estimated marginal means of duration (in raw, untransformed seconds) from the multilevel model. Analyses in text use duration measured in square-root transformed seconds in order to correct a positive skew in the distribution. Error bars represent 95% confidence intervals.

motivation was unchanged from baseline to post-gel for men high in trait dominance ($Time_{Base\ v.\ Post\text{-}gel}$: B = 0.054, Z = 0.440, p = 0.66) but marginally increased for men low in trait dominance ($Time_{Base\ v.\ Post\text{-}gel}$: B = 0.218, Z = 1.79, p = 0.074).

A similar pattern was found when decomposing the interaction for the second time contrast, although in these comparisons testosterone treatment was associated with decreased motivation from pre- to post-stress for men with high trait dominance ($Time_{Pre\text{-}v.\ Post\text{-}Stress}$: B = -0.389, Z = 2.15, p = 0.032). But testosterone did not alter motivation from pre- to post-stress in men who were low in trait dominance ($Time_{Pre\text{-}v.\ Post\text{-}Stress}$: B = 0.272, Z = 1.49, p = 0.14). In the placebo group, changes in pre- to post-stress motivation were not observed for men at high ($Time_{Pre\text{-}v.\ Post\text{-}Stress}$: B = 0.025, Z = 0.136, p = 0.89) or low dominance ($Time_{Pre\text{-}v.\ Post\text{-}Stress}$: B = -0.120, Z = 0.649, p = 0.516). These effects must be interpreted with caution as the study lacked a strong control for the social-evaluative

stressor and thus these demotivating effects of stress cannot be separated from possible confounds of time or mere statistical phenomena like regression to the mean.

In summary, testosterone and high trait dominance robustly increased motivation after gel application, but following a stressful encounter, this same combination of testosterone and high trait dominance was linked to a reduction motivation. The social-evaluative stressor was not found to perturb motivation in low trait dominance men given testosterone or placebo. Supplementary analyses with alternative contrasts confirmed that testosterone treatment coupled with high trait dominance produced an increase in motivated persistence that was reduced following social-evaluative stress (see Supplementary Materials, Table 6.2).

Mediation of testosterone and dominance interaction by stress reactivity variables

We investigated moderated mediation models to explore the extent to which responses to the social-evaluative stressor – specifically, cortisol AUC_I and negative affect – predicted changes in motivated persistence. Although cortisol reactivity was not found to mediate the interactive effects of testosterone and trait dominance on handgrip performance ($\omega_{Cortisol}$ = -0.067, 95%CI[-0.298, 0.108]), negative affect in response to the social-evaluative stressor was found to marginally mediate the interactive effects of testosterone and trait dominance on handgrip duration ($\omega_{NegativeAffect}$ = -0.181, 95%CI[-0.416, 0.024]). As reported elsewhere (24), men who were given testosterone and were high in trait dominance reported higher levels of negative affect linked to the stressor (i.e., mean negative affect in anticipation of and while recovering from the social-evaluative stressor, controlling for baseline negative affect). These higher levels of negative affect were in turn associated with reduced motivation from pre- to post-stressor

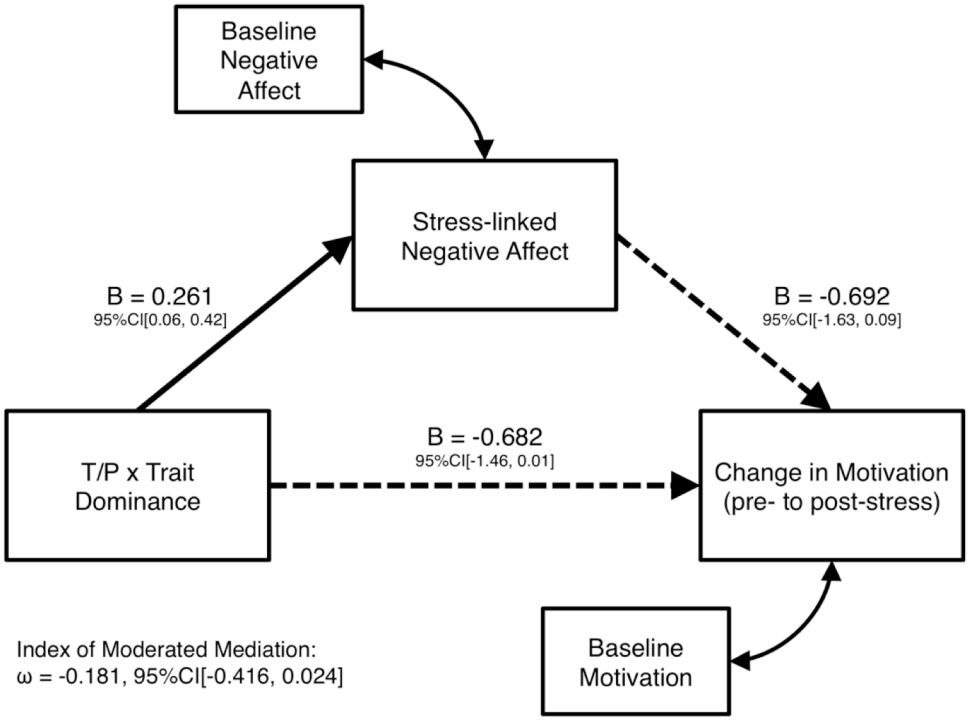

Figure 6.3. Model of moderated mediation analyses. Not shown are main effects of testosterone and trait dominance on stress-linked negative affect and motivation. All estimates are unstandardized per convention (Hayes, 2013). All 95% confidence intervals are bootstrap bias-corrected (n = 10,000 samples).

(Figure 6.3). Further, indices of moderated mediation for the negative subscales, fear and hostility, which were implicated in prior work as underlying the negative response to stress (24), were found to have similar directions and magnitudes as the general negative affect scale (ω_{Fear} = -0.221, 95%CI[-0.460, 0.002]; $\omega_{Hostility}$ = -0.101, 95%CI[-0.315, 0.092]). Thus testosterone increases negative affect in response to social-evaluative stress and this negative affect may mechanistically explain decreases in motivation following the stressor, especially for individuals who are high in trait dominance.

Discussion

This study provides evidence that exogenous testosterone given to men who are high in trait dominance was associated with increased motivation as demonstrated by

persistence on a handgrip task. This work extends prior work on testosterone and motivation (9) by demonstrating that testosterone causally increases motivated performance on a physically demanding task, but only in dominant men. The present findings also add to the small but growing research on the interactive effects of testosterone and trait dominance (18-20) by showing that trait dominance accentuated testosterone's effects on motivated persistence. Future work should continue to investigate the interactive effects of testosterone and trait dominance on directing motivated persistence in order to determine the extent to which motivation underlies the status-relevant, dominant behavior found in prior work.

This study also reveals that a social stressor may reduce the interactive effects of testosterone and dominance on motivation. Performance on the handgrip task significantly declined for high dominant men who were given testosterone compared to placebo. Mediation analyses revealed that this decline in performance was mechanistically linked to negative affect, specifically fear and hostility, but not cortisol responses to the stressor. The stress-linked effects on the testosterone and dominance interaction corresponds to earlier work in which dominant women given testosterone avoided pursuing competitions after a status-threatening loss (20), and suggests negative affect in response to social threat may direct motivation associated with these status-relevant behavioral outcomes. While testosterone has been linked to increased neural responses to socially threatening stimuli (34), future work should focus on the interactive effects of testosterone and trait dominance on the neurobiology that supports negative affect and threat and may therefore influence motivated goal pursuit.

In a broader context, this negative affect mechanism is consistent with prior work suggesting that negative affect can disrupt motivation to abstain from addictive substances (35). Further, separate lines of research have linked high levels of testosterone (36) and trait dominance (37) to increased risk for substance abuse. The present work suggests subjective responses to stressors may be a putative target for intervention for these men who may be at increased risk for stress-linked substance abuse. We do caution over-interpretation of the present study, as it did not contain a strong control for the social-evaluative stressor, and so confounding factors like time or the repeated nature of the assessments may explain the stress-linked changes in motivation. Also, alternative conceptual explanations – perhaps the lack of motivation is actually a sign of redirecting energy towards dealing with the status-relevant aspects of the stressor (24) rather than with the unrelated, physical task – may better explain the results. But our findings do align with extant research on stress and motivation (23) and the marginal relationship with stress-linked affect supports the argument that the social-evaluative stressor is altering motivational persistence.

Although the main effects of testosterone treatment did not significantly alter motivation, the direction of the effects of testosterone on changes in motivation were consistent with the positive correlation seen in previous work (9). This discrepancy between the present findings and prior research could depend on several factors. For example, it is possible that the effects of testosterone on motivation found in Welker & Carré (9) are specific to non-physical persistence and that for physical tasks, high levels of trait dominance may be necessary for testosterone to motivate performance. Perhaps more importantly, motivated persistence was linked to endogenous basal testosterone

levels in the earlier work, but not to testosterone reactivity (9). Since exogenous administration results in an acute increase in testosterone concentrations, testosterone administration may be a better model of testosterone reactivity than of basal testosterone measures. Indeed, in attempting to explain their null effects of testosterone reactivity, Welker & Carré (9), hypothesized that "individual differences factors [relating to dominance] may play a key role in moderating the effects of testosterone reactivity" on motivated persistence behavior (p. 87). The present work provides initial confirmatory evidence of this hypothesis, insofar as exogenous testosterone models testosterone reactivity.

The present study reports evidence of testosterone's effects on motivation via a simple physical task, but motivation is a multiply determined cognitive construct that is reciprocally linked to aspects of executive function and cognitive control (2), and so may benefit from more neurally-informed investigations. For example, motivated persistence towards a goal requires continued, heightened valuations of both the goal and the means to pursuing the goal; inhibition of competing goals or other distracting information; and regulation of affect that occurs contemporaneously with goal pursuit (38-40). More precise experimental tasks that measure specific neurocognitive processes would allow delineation of which cognitive aspects of motivation are altered by testosterone and trait dominance (41). This neurocognitive point of view could then highlight putative neural systems that mechanistically explain the effects of testosterone and trait dominance on motivation and inform interventions seeking to support goal-directed motivation and behavior outside of the laboratory.

Finally, future work should focus on the implications of these changes in motivation for goal-directed behavior, especially those relevant to health behavior like diet or exercise. These results show that testosterone increases motivation in dominant men, which may predict increased ability to pursue goals and persevere towards success in challenging endeavors (1). But testosterone also makes these dominant men more susceptible to the demotivating effects of stress. Thus the benefits of testosterone on motivation and perseverance may be lost for high dominant men in times of stress, leading to dominant men with high levels of testosterone possibly shrinking from pursuing goals. Testosterone and trait dominance could therefore be a liability for goal pursuit during stressful encounters, which may reduce the likelihood of successfully achieving personal health goals.

Supplementary Materials

Supplementary Methods

Participants were read the following instructions prior to the first (baseline) handgrip assessment:

> Handgrips are usually used to strengthen one's forearms and hands. In research, handgrips are used to determine how much willpower and persistence an individual is able to exert to achieve a desired goal. In today's study, we are going to video record you as you squeeze the handgrip. Your goal is to squeeze the handgrip for as long as you can, keeping the marble from falling from between the handles of the handgrip. The videos will later be viewed by trained researchers in order to determine how long you were able to keep the marble aloft – essentially a measure of how good you were at this task.
>
> While you grip the handgrip, please hold your arm parallel to the desktop with the handgrip upright. I will hold the marble in place. You can start squeezing when I say so. Please use your dominant hand for this task.

For later trials, the experimenter read the following instructions:

We are going to video record you again as you squeeze the handgrip. Keep in mind that you goal is still to squeeze the handgrip for as long as you can and that the videos will be viewed by trained researchers to determine how good you were at the task.

Supplementary Analyses

In the main document, Helmert contrasts were used to analyze change in motivation across the three handgrip assessments as a conservative test of the null hypotheses. Here, alternative contrasts – including a dummy code that uses the second assessment as the reference level to examine change from baseline to pre-stress (Contrast 1: Baseline = 1; Pre-Stress = 0; Post-Stress = 0) and from pre- to post-stress (Contrast 2: Baseline = 0; Pre-Stress = 0; Post-

	Dependent Variable: Duration (square-root transformed sec.)		
	B	*CI*	*p*
Fixed Parts			
(Intercept)	7.721	7.404 – 8.038	**<.001**
$Time_{Base\ v.\ Post\text{-}gel}$	0.199	0.079 – 0.319	**.002**
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$	-0.053	-0.233 – 0.127	.565
Trait Dominance (Dom.)	0.328	0.012 – 0.644	**.044**
T/P	-0.105	-0.422 – 0.212	.518
Blinding	0.061	-0.257 – 0.379	.708
$Time_{Base\ v.\ Post\text{-}gel}$ x Dom.	0.052	-0.067 – 0.172	.393
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x Dom.	-0.129	-0.308 – 0.049	.159
$Time_{Base\ v.\ Post\text{-}gel}$ x T/P	0.063	-0.057 – 0.183	.305
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x T/P	-0.005	-0.186 – 0.175	.954
T/P x Dom.	0.258	-0.057 – 0.573	.112
$Time_{Base\ v.\ Post\text{-}gel}$ x T/P x Dom.	0.135	0.015 – 0.254	**.030**
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x T/P x Dom.	-0.202	-0.380 – -0.023	**.029**
Random Parts			
N_{SUBID}		114	
ICC_{SUBID}		0.549	
Observations		338	
R^2 / Ω_0^2		.776 / .737	

Table 6.1. Full output from multilevel model. Time consists of two Helmert-coded contrasts that compare baseline duration to the two post-gel assessments in the first contrast, and pre- vs. post-stress duration in the second contrast.

Stress = 1) – are used to demonstrate the robustness of these effects. Note that the use of positive ones in the standard dummy coding arrangement will result in estimates that are flipped compared to the original models. These contrasts are otherwise entered into the multilevel model as described in the main document.

Supplementary Results

In the main document, we report the piecemeal terms from the multilevel model. Here we provide the full output of the multilevel model (Table S6.1). The alternative analyses, using the dummy contrast codes that test changes from baseline to pre-stress, and

	Dependent Variable: Duration (square-root transformed sec.)		
	B	*CI*	*p*
Fixed Parts			
(Intercept)	7.973	7.589 – 8.358	**<.001**
$Time_{Base\ v.\ Pre\text{-}Stress}$	-0.651	-1.053 – -0.249	**.002**
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$	-0.106	-0.467 – 0.254	.565
Trait Dominance (Dom.)	0.509	0.127 – 0.892	**.010**
T/P	-0.036	-0.421 – 0.348	.853
Blinding	0.061	-0.257 – 0.379	.708
$Time_{Base\ v.\ Pre\text{-}Stress}$ x Dom.	-0.286	-0.687 – 0.115	.164
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x Dom.	-0.258	-0.615 – 0.099	.159
$Time_{Base\ v.\ Pre\text{-}Stress}$ x T/P	-0.194	-0.596 – 0.208	.345
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x T/P	-0.011	-0.371 – 0.350	.954
T/P x Dom.	0.594	0.212 – 0.976	**.003**
$Time_{Base\ v.\ Pre\text{-}Stress}$ x T/P x Dom.	-0.605	-1.007 – -0.204	**.004**
$Time_{Pre\text{-}\ v.\ Post\text{-}Stress}$ x T/P x Dom.	-0.403	-0.760 – -0.046	**.029**
Random Parts			
N_{SUBID}		114	
ICC_{SUBID}		0.549	
Observations		338	
R^2 / Ω_0^2		.776 / .737	

Table 6.2. Full output from multilevel model with alternative contrast coding. Time consists of two dummy-coded contrasts that compare baseline duration to the pre-stress assessment, and pre- vs. post-stress duration. The negative values of the interaction estimates result from the use of positive ones in the dummy codes but are actually representative of the same pattern found in the original analyses.

pre- to post-stress, reveal much the same pattern: T/P x Dominance is associated with increased motivation from baseline to the second assessment (three hours post-gel, pre-stress) but is associated with decreased motivation from pre- to post-stress (Table 6.2).

CHAPTER VII

GENERAL DISCUSSION

In sum, this work examined the causal manipulations of psychosocial and endocrine antecedents to social-evaluative stress responses. Cortisol, affective, and behavioral responses to social stressors were found to depend on the interactive effects of social status and hierarchy stability (Chapter 2) and the interactive effects of testosterone and trait dominance (Chapters 4 & 6). Specifically, a high status position buffered the effects of stress on cortisol reactivity and improved performance during the stressor, but only in a stable hierarchy. In an unstable hierarchy, a high status position increased cortisol and testosterone reactivity to the stressor and did not provide a boost to behavioral performance. In the latter two chapters, testosterone treatment increased cortisol and negative affect responses to social-evaluative stress, but only for dominant men. The interactive effects of testosterone and trait dominance also increased motivation on a physical persistence task, but exposure to stress resulted in reduced motivation for these same dominant men given testosterone.

Future work should examine the extent to which these constructs – social status, hierarchy stability, testosterone levels, and trait dominance – determine stress responses in ecologically valid contexts. For example, while social status has been extensively investigated in societal hierarchies (1), little work has investigated if hierarchy instability – for example a global recession, individual employment uncertainty, or change in interpersonal relationship status – upsets the known relationship between high status and lower stress responses (2). Similarly, testosterone is prescribed for its beneficial effects on health (3), but this biomedical research generally ignores the impact of individual

differences in status-relevant factors like trait dominance. It is therefore unknown what role status-relevant individual differences like trait dominance might play in modulating testosterone's effects on stress responses over longer time ranges, across the life span. This dissertation shows the criticality of these interactive effects on altering stress responses, but more work is necessary to link these well-controlled, laboratory studies to broader, longer-term effects on stress and health.

Further, while this work examined acute stress responses that have been previously linked to downstream consequences on health [i.e., cortisol reactivity (4) and negative affect (5)], future work must carefully examine the extent to which the psychosocial and biological pathways studied here alter immune functioning and subsequent health outcomes. Prior work has correlated lower societal ranking with increased inflammation (6) and increased inflammatory immune reactivity to stress (7). Yet none of this work has examined the effects of experimentally manipulated social status on immune functioning to be able to determine its causal effects or explored putative moderators of social status' effects on immune functioning, such as hierarchy stability. Testosterone is often linked to immune suppression (8) though the relationships between testosterone and immune functioning in humans are equivocal, especially when studied in a social context (9). Continued examination of the causal effects of these status-relevant factors could provide new insights on and targeted interventions for the psychosocial and endocrine antecedents to stress, health, and well-being.

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