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Personality × Hormone Interactions in Adolescent Externalizing Psychopathology

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The "dual-hormone" hypothesis predicts that testosterone and cortisol will jointly regulate aggressive and socially dominant behavior in children and adults (e.g., Mehta & Josephs, 2010). The present study extends research on the dual-hormone hypothesis by testing the interaction between testosterone, cortisol, and personality disorder (PD) traits in predicting externalizing problems in a community sample of adolescent males and females. Participants were 106 youth from the community, ranging in age from 13–18 (M_{age} = 16.01 years, SD_{age} = 1.29), and their parents. Parents and youth provided ratings on an omnibus measure of personality pathology and externalizing problems. Youth provided saliva samples via passive drool from which testosterone and cortisol levels were obtained. Robust moderation of the joint effects of testosterone and cortisol on parent-reported externalizing problems was found for both higher-order PD traits associated with externalizing psychopathology (Disagreeableness and Emotional Instability). Higher testosterone was associated with externalizing outcomes, but only when cortisol was low, and only among youth with high levels of Disagreeableness and Emotional Instability. These findings provide the first evidence for the dual-hormone hypothesis in a mixed-sex sample of community adolescents, but importantly offer novel evidence for the importance of personality traits. Examination of the joint regulation of externalizing problems by testosterone and cortisol in the context of adolescent personality may help to clarify inconsistent main effects of testosterone and cortisol on clinical externalizing phenotypes.

Keywords: adolescence, cortisol, externalizing problems, personality disorder, testosterone

The *externalizing spectrum* is a broad, cross-cutting dimension that organizes multiple behaviors and accounts for much of the comorbidity among disinhibitory forms of psychopathology (e.g., Achenbach & Edelbrock, 1978; Krueger, 1999). The externalizing spectrum construct has been useful in reconciling high rates of diagnostic co-occurrence, identifying common personality correlates underlying distinct types of psychopathology, and elucidating etiologic pathways common to disorders falling on the same spectrum (e.g., Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007; Dick et al., 2008; Krueger & Markon, 2006; Krueger, McGue, & Iacono, 2001). The externalizing spectrum comprises psychopathology reflecting disinhibitory and antagonistic processes, including aggression, delinquency, and substance use problems (Krueger & South, 2009). Normal-range personality characteristics, such as impulsivity and social dominance, also show strong links to the externalizing spectrum (Johnson, Leedom, & Muhtadie, 2012; Olson, Schilling, & Bates, 1999).

In the present study, we focus on the most common manifestations of youth externalizing: aggression and rule-breaking behaviors (e.g., Burt, 2012). Our goal is to leverage what we know of the associations between hormones and normal-range manifestations of externalizing behavior, and integrate this with research on externalizing psychopathology. By doing so, we hope to illustrate the utility of a spectrum conceptualization in elucidating hormone-externalizing associations.

Hormone-Externalizing Behavior Associations

Although hormone–behavior relationships in humans have been most frequently investigated in adults, it is in fact during adolescence that the largest and most dramatic hormonal changes occur—most notably, changes in sex steroid concentrations (Granger, Schwartz, Booth, & Arentz, 1999). However, the liter-

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ature exploring hormone-externalizing behavior associations has revealed mixed findings in both adolescents and adults (Archer, Graham-Kevan, & Davies, 2005).

One possible reason for these mixed findings is attributable to an historical emphasis on single hormones in behavioral studies. Recently, researchers have begun examining *joint* hormonal regulation of behavior. For example, a growing literature supports the joint regulation of normal-range socially dominant behavior by two hormones—testosterone and cortisol—such that higher levels of testosterone (T) are associated with more socially dominant behavior, but only among individuals with low levels of cortisol (C – Mehta & Josephs, 2010; van Honk, Harmon-Jones, Morgan, & Schutter, 2010; but see Denson, Mehta, & Tan, 2013). T and C, which are the outputs of distinct endocrine systems (the hypothalamic-pituitary-gonadal [HPG] and hypothalamic-pituitary-adrenal [HPA] axes, respectively), are linked to many behavioral (and sometimes opposing) outcomes—such as dominance/approach (for T) versus submissiveness/withdrawal (for C). T's link to dominance is explained, in part, by its association with high reward sensitivity, low punishment sensitivity, low fear, and behavioral approach (Carré & McCormick, 2008; Mehta & Josephs, 2006; Oyegbile & Marler, 2005; van Honk et al., 2004), whereas C's link to submissiveness can be understood by its association with high fear, high punishment sensitivity, and behavioral withdrawal (Schulkin, 2007; Schulkin, Gold, & McEwen, 1998). Further, evidence shows that C is associated with suppression of HPG axis activity, inhibition of T on target tissues, counterregulation of the prefrontal-cortex-amygdala circuit that is modulated by T—all of which suggest the possibility that the HPA and HPG axes act on behavior interdependently (Burnstein, Maiorino, Dai, & Cameron, 1995; Chen, Wang, Yu, Liu, & Pearce, 1997; Denson, Ronay, von Hippel, & Schira, 2013; Johnson, Kamilaris, Chrousos, & Gold, 1992; Smith, Syms, Nag, Lerner, & Norris, 1985; Tilbrook, Turner, & Clarke, 2000; Urry et al., 2006).

Understanding the interplay between T and C is particularly important in predicting aggressive behavior, which emerges as a result of high reward sensitivity/behavioral approach in the context of low inhibitory/regulatory mechanisms. Indeed, support for this "dual hormone hypothesis" (Mehta & Josephs, 2010) has emerged in the prediction of aggressive behaviors in clinical samples of male adolescents, with some support for the prediction of rule-breaking behaviors as well (Dabbs, Jurcovic, & Frady, 1991; Popma et al., 2007), and in the prediction of psychopathic traits in nonclinical adult samples (examining C reactivity rather than basal C; Glenn, Raine, Schug, Gao, & Granger, 2011). In addition to aggressive behavior, which is often considered pathological, "normal" behaviors that reflect social dominance, such as competitiveness and leadership, are associated with interactive effects of T and C (Mehta & Josephs, 2010; Glenn et al., 2011; Denson, Mehta, et al., 2013). The current study aims to test, for the first time, whether the interactive effects of C and T are associated with adolescent externalizing problems (i.e., rule-breaking and aggressive behaviors) in a mixed-sex community sample, and also to test the novel hypothesis that this interactive effect is moderated by pathological personality traits. Specifically, we aim to extend this line of research by examining the potential moderating role of individual personality characteristics on these joint hormone effects.

Personality as a Psychological Context for Hormone-Externalizing Behavior Associations

Personality traits—broad individual patterns of thoughts, feelings, and behavior (Allport, 1937)—are highly relevant when exploring the emergence, development, and consequences of psychopathology (Tackett, 2006; Widiger & Smith, 2008). In support of this, recent studies have reported the value of personality traits as an elucidating context for investigating associations between biological indicators and psychopathology (e.g., DeYoung & Clark, 2012), including work that has specifically focused on single hormone-psychopathology associations (Davies, Sturge-Apple, & Cicchetti, 2011; Gunnar, Kryzer, van Ryzin, & Phillips, 2011; Tackett, Kushner, et al., 2014). The current study proposes to examine the moderating influence of personality traits (specifically, personality disorder [PD] traits) on hormone-externalizing behavior associations in adolescents.

Obviously, not all PD traits should be associated with hormonal activity, and certainly, even fewer are likely to be associated with both T and C. One major advantage of PD trait models is their dimensional nature, which captures clinical *and* subclinical variance in pathology domains across different populations (e.g., clinical vs. nonclinical, juveniles vs. adults), as opposed to measures that focus on discrete "extremes" of behavior (Tackett, Balsis, Oltmanns, & Krueger, 2009). Indeed, PD trait models recently have been empirically derived within child and adolescent samples (De Clercq, De Fruyt, Van Leeuwen, & Mervielde, 2006), offering an important opportunity to begin examining personality moderation of hormone–behavior relationships at earlier developmental stages.

Although the study is largely breaking new ground, we can nonetheless formulate some general, tentative hypotheses based on previous literature. Specifically, previous research on personality–psychopathology associations highlights those traits most relevant for the youth externalizing spectrum, whereas previous work on individual differences moderation of hormone-psychopathology associations in youth provides some indication about the expected directionality of these effects. Robust evidence supports the association of the higher-order personality traits of agreeableness, conscientiousness, and neuroticism with externalizing behaviors in adults (Jones, Miller, & Lynam, 2011; Kotov, Gamez, Schmidt, & Watson, 2010; Miller & Lynam, 2001; Trull & Sher, 1994) and youth (Lahey & Waldman, 2007; Martel, 2009; Nigg, 2006; Tackett, 2006). Further, a recent study using the same higher-order PD trait model as that used in the present study found the higher-order traits of Disagreeableness and Emotional Instability (which represent the pathological variants of agreeableness, impulsivity, and neuroticism in PD trait models) to show strong and moderate associations, respectively, with the externalizing spectrum in youth (Tackett, Herzhoff, Reardon, De Clercq, & Sharp, 2014). The few existing studies that find personality moderation of hormone–behavior associations in youth have reported that hormone–behavior associations are stronger at higher-risk levels of personality traits (Davies et al., 2011; Gunnar et al., 2011; Tackett, Kushner, et al., 2014), all of which have focused on a single hormone, C, and limited measurement of youth personality, with one exception (Tackett, Kushner, et al., 2014). This is consistent with the idea that broadly distributed personality traits moderate the threshold of clinically significant associations, allowing such

associations to be detected even in more normative, generalizable samples. Thus, we hypothesized that youth with particularly high levels of trait Disagreeableness and Emotional Instability should be most likely to demonstrate the dual hormone hypothesis effects on externalizing behavior outcomes.

The Present Study

In the present study, we hypothesized that PD traits would moderate the relationship between hormone concentrations (T × C) and externalizing problems in an adolescent community sample. At the higher-order trait level, we hypothesized that youth high on Disagreeableness and Emotional Instability traits would show a stronger association between T × C and youth externalizing. At the lower-order trait level, we conducted follow-up exploratory analyses to offer some insight into which specific trait domains may be particularly relevant for this association. Based on previous findings (Alink et al., 2008; Archer et al., 2005; Dabbs et al., 1991; Popma et al., 2007), we did not expect to observe hormone main effects on youth externalizing problems. We examined these research questions in a mixed-sex sample of community adolescents, using both self- and parent-report of youth externalizing problems.

Method

Participants

Participants were 47 boys and 59 girls between the ages of 13 and 18 years (M_{age} = 16.01 years, SD_{age} = 1.29) and their parents (96 mothers and 10 fathers). The youth participant's ethnicity was reported by parents and youth as follows: 74.5% White, 12.3% Other, 6.6% Asian, 4.7% African Canadian, 0.9% Latino/Latina, and 0.9% Pacific Islander. On average, parents reported a total average annual household income in the 70,001 to 80,000 Canadian dollars bracket and 87.7% of parents reported having a degree from a 4-year college or more, 6.6% reported some college education, and 5.7% reported high school graduate without college education. Exclusion criteria for youth and parent participants included lack of English fluency, or a history of mental retardation, autism, or schizophrenia in the target youth. Participants were recruited from a sample that had previously participated in a study on child personality and psychopathology (response rate for follow-up participation was 80%, with 72% of this sample participating in the lab portion of the study). For their participation, adolescent participants were reimbursed with a gift card and parents with 50 Canadian dollars. All study methods and materials were approved by the university office of research ethics. Parents and adolescents provided informed consent/assent before the study.

Materials

Table 1 presents sample items and internal consistencies for all scales and subscales for both parent- and youth-report.

Dimensional Personality Symptom Itempool (DIPSI). The DIPSI (De Clercq et al., 2006) was used to assess youth's personality pathology traits by parent- and self-report. The DIPSI is an empirically derived measure of personality pathology traits origi-

Table 1
Sample Items and Internal Consistencies for Personality Pathology Traits and Externalizing Problems

Variable	Sample items	Cronbach's α	
		Parent	Youth
Externalizing Problems		.90	.90
DIPSI higher-order			
Disagreeableness		.98	.97
Emotional Instability		.97	.97
DIPSI lower-order			
Hyperexpressive Traits	Exhibits his/her feelings at all occasions	.89	.83
Hyperactive Traits	Can never sit still	.79	.80
Dominance-Egocentrism	Manipulates other children repeatedly to have his/her way	.84	.81
Impulsivity	Acts constantly without considering the consequences	.88	.81
Irritable-Aggressive Traits	Gets easily irritated	.92	.91
Disorderliness	Never takes care of his/her belongings	.90	.85
Distraction	Never finishes his/her work	.89	.85
Risk Taking	Likes to take risks	.83	.89
Narcissistic Traits	Considers him/herself more worthy than others	.85	.85
Affective Lability	His/her feelings towards others are constantly changing	.90	.89
Resistance	Breaks rules all the time, both at school and at home	.85	.83
Lack of Empathy	Feels no emotions when other children get hurt	.87	.86
Dependency	Needs someone around all the time	.86	.84
Anxious Traits	Panics very easily	.91	.86
Lack of Self-Confidence	Always has doubts about him/herself	.73	.84
Insecure Attachment	Often clings to other people	.55	.58
Submissiveness	Obeys other children all the time	.81	.85
Ineffective Coping	Is very sensitive to stress	.91	.92
Separation Anxiety	Constantly fears being on his/her own one day	.87	.75
Depressive Traits	Often feels empty inside	.78	.83
Inflexibility	Cannot adjust to sudden changes in plans	.92	.82

Note. DIPSI = Dimensional Personality Symptom Itempool (De Clercq et al., 2006).

nally developed in Dutch and more recently validated in an English translation (Tackett & De Clercq, 2009). It assesses 27 lower-order facets that load onto four higher-order factors. Youth and parents were asked to indicate the degree to which a statement is characteristic of the youth on a scale from 1 (*not characteristic*) to 5 (*highly characteristic*). Analyses focused on two of the higher-order trait dimensions: Disagreeableness and Emotional Instability; the 12 Disagreeableness facets, and the 9 Emotional Instability facets (see Table 1).

Child Behavior Checklist (CBCL) and Youth Self Report (YSR). The CBCL and YSR (Achenbach & Rescorla, 2001) were used to assess youth's Externalizing Problems by parent- and self-report. The CBCL and YSR are widely used questionnaire measures of child problem behaviors. They assess seven narrowband problem behavior scales of which five load onto the two broadband Externalizing and Internalizing Problems scales. Youths and parents were asked to indicate how true a problem behavior was for the youth in the past 6 months on a scale from 0 (*not true* [*as far as you know*]) to 2 (*very true or often true*). Analyses focused on the broadband Externalizing Problems scale (see Table 1).

Cortisol and testosterone. C and T were measured one time, using passive-drool.[1] After collection, saliva samples were frozen at −20 °C until they were shipped on dry ice to Clemens Kirschbaum's laboratory at Technical University of Dresden for analyses of hormone concentrations. After thawing, saliva was centrifuged at 3,000 rpm for 5 min, which resulted in a clear supernatant of low viscosity. Salivary concentrations were measured using commercially available chemiluminescence-immunoassays with high sensitivity (IBL International, Hamburg, Germany). The intra- and interassay coefficients for C and T were each below 8%.

Procedure

To limit the influence of menstrual variation on hormone levels, saliva samples from women were collected during the follicular period of their menstrual cycle (i.e., first 10 days) starting from and including the first day of menstruation. To limit the influence of diurnal variation on hormonal levels, samples were collected between noon and sundown (Kirschbaum & Hellhammer, 1994). Participants were asked to not eat or drink for at least two hours and to not smoke for at least four hours before their session. Immediately after they gave consent upon arriving to the lab, participants were asked to rinse out their mouth with water and drink a small cup of water. After 30 minutes of sedentary activities such as answering demographic questions and completing questionnaires, participants were asked to drool through a sanitary straw into a 2-ml vial. Caregivers completed their questionnaires in a separate room at the laboratory. The complete study took approximately 2.5 hours and included additional behavioral tasks and questionnaires.

Results

Descriptive Statistics and Correlations

Examination of variable frequencies revealed one extreme value for C that was Winsorized before analyses. Means and standard deviations for all personality pathology variables, the Externalizing Problems variable, C, T, and age are presented in Table 2. For further analyses, C and T were log-transformed to more closely approximate normally distributed variables. Specifically, initial skew values for C (1.43) and T (2.25) improved following a log transformation (−0.29 and −0.33). Given that boys ($M = 35.83$, $SD = 30.37$) had significantly higher testosterone levels than did girls ($M = 7.81$, $SD = 7.72$), $t(50.75) = 6.17$, $p < .001$, T values were standardized within sex, consistent with previous research (Josephs et al., 2006; Mehta & Beer, 2010; Mehta & Josephs, 2010; Mehta, Wuehrmann, & Josephs, 2009; Newman et al., 2005; Zyphur, Narayanan, Koh, & Koh, 2009). In addition, we report Pearson correlations between higher-order DISPI factors, all 12 Disagreeableness facets, and all 9 Emotional Instability facets with Externalizing Problems and transformed values for C and T in Table 2. As expected, these correlations revealed little evidence for main effects between T and C and Externalizing Problems.

All independent variables were standardized to test the hypothesis of a T × C × PD trait interaction predicting Externalizing Problems. To test this hypothesis, age, sex, and time of waking were entered first into hierarchical regression analyses, followed by T values, C values, and the PD trait at Step 2, the 2-way interaction terms between T, C, and the PD trait at Step 3, and the 3-way interaction term between T, C, and the PD trait at Step 4, in predicting Externalizing Problems. Analyses were conducted separately by parent and youth report (see Table 3). Analyses revealed multiple significant interactions but only for parent-reported Externalizing Problems (unless stated otherwise, the significance level was corrected to $\alpha = .01$, due to the number of tests performed). These significant interactions were probed using simple slope analyses at C levels 1 *SD* below and above the mean using Hayes' (2013) PROCESS modeling and are described separately by DIPSI higher-order factors, Disagreeableness facets, and Emotional Instability facets.

To examine the robustness of these findings, we also ran analyses (a) separately for males and females, and accounting for the following covariates: (b) time of testing and pubertal status measured using the Physical Development Scale (Petersen, Crockett, Richards, & Boxer, 1988), (c) Internalizing Problems measured using the CBCL (Achenbach & Rescorla, 2001), and (d) either Disagreeableness when examining the 3-way interaction with Emotional Instability or Emotional Instability when examining the 3-way interaction with Disagreeableness. Findings from all of these analyses confirmed the overall pattern of results, supporting the robustness of our main findings reported below.

T × C Interaction Effects Moderated by DIPSI Higher-Order Factors

Disagreeableness ($\beta = -0.25$, $t(95) = -4.52$, $p < .001$) and Emotional Instability ($\beta = -0.29$, $t(95) = -3.29$, $p = .001$)

[1] There is some evidence pointing to the temporal stability of T (Kreuz & Rose, 1972; Liening et al., 2010; Sellers, Mehl, & Josephs, 2007) and more recently, C (Liening et al., 2010), as well as evidence that one-time hormone measures can reliably predict outcomes such as marital status (Booth & Dabbs, 1993; Gettler, McDade, & Kuzawa, 2011), physiological reactivity to environmental threat (Josephs, Sellers, Newman, & Mehta, 2006; Mehta, Jones, & Josephs, 2008; Newman, Sellers, & Josephs, 2005), aggression (Ehrenkranz, Bliss, & Sheard, 1974, but see, Tremblay et al., 1998), and social dominance (Ehrenkranz et al., 1974; Mehta & Josephs, 2010; Tremblay et al., 1998).

Table 2
Means, Standard Deviations, and Correlations Between Personality Pathology Traits, Externalizing Problems, and Testosterone and Cortisol

Variable	*M*	*SD*	Correlations	
			Testosterone[a]	Cortisol[a]
Age	16.01	1.29		
Testosterone (pg/ml)	20.24	25.15		
Cortisol (nmol/l)	5.63	3.59		
Parent-reports on				
Externalizing Problems	6.57	7.24	.03	−.02
DIPSI higher-order factors				
Disagreeableness	1.68	0.59	.00	−.08
Emotional Instability	1.52	0.56	−.03	.01
DIPSI lower-order factors				
Hyperexpressive Traits	1.63	0.73	.00	−.07
Hyperactive Traits	1.83	0.72	−.02	−.02
Dominance-Egocentrism	1.74	0.71	.05	−.14
Impulsivity	1.63	0.86	.08	.04
Irritable-Aggressive Traits	1.61	0.75	−.02	.01
Disorderliness	2.22	0.95	.00	−.10
Distraction	1.54	0.70	.12	−.06
Risk Taking	1.55	0.62	−.09	−.13
Narcissistic Traits	1.92	0.66	−.10	−.07
Affective Lability	1.76	0.86	.01	.01
Resistance	1.38	0.63	−.03	−.19*
Lack of Empathy	1.37	0.53	−.02	−.08
Dependency	1.36	0.63	−.03	−.02
Anxious Traits	1.52	0.72	.03	.04
Lack of Self-Confidence	1.51	0.63	−.10	.00
Insecure Attachment	1.59	0.64	−.11	.03
Submissiveness	1.51	0.57	.06	−.01
Ineffective Coping	1.90	0.86	−.02	.05
Separation Anxiety	1.27	0.63	−.02	.04
Depressive Traits	1.45	0.66	.00	−.01
Inflexibility	1.53	0.73	−.02	−.08
Youth-reports on				
Externalizing problems	11.68	8.95	−.07	.04
DIPSI higher-order factors				
Disagreeableness	2.13	0.61	−.05	.15
Emotional Instability	2.05	0.64	−.03	.16
DIPSI lower-order factors				
Hyperexpressive Traits	2.24	0.81	.01	.03
Hyperactive Traits	2.48	0.80	−.07	.17
Dominance-Egocentrism	2.25	0.74	−.12	−.01
Impulsivity	1.93	0.83	−.04	.11
Irritable-Aggressive	1.88	0.78	−.09	.24*
Disorderliness	2.44	0.85	−.02	.21*
Distraction	2.03	0.74	.05	.18
Risk Taking	2.41	0.90	−.04	−.01
Narcissistic Traits	2.50	0.82	−.02	−.04
Affective Lability	2.20	1.04	−.06	.25**
Resistance	1.65	0.71	−.04	.07
Lack of Empathy	1.54	0.53	−.04	.14
Dependency	1.90	0.79	.00	.14
Anxious Traits	2.22	0.90	−.17	.11
Lack of Self-Confidence	2.09	0.92	−.07	.06
Insecure Attachment	2.06	0.71	.08	.16
Submissiveness	1.92	0.73	.08	.23*
Ineffective Coping	2.41	0.97	−.08	.22*
Separation Anxiety	1.64	0.79	.00	−.03
Depressive Traits	2.13	0.93	.01	.13
Inflexibility	2.11	0.73	−.01	.08

Note. DIPSI = Dimensional Personality Symptom Itempool (De Clercq et al., 2006).
[a] Correlations for testosterone and cortisol are based on the log-transformed variables. In addition, correlations for testosterone are further based on variables standardized within sex.
* $p < .05$. ** $p < .01$.

Table 3
Hierarchical Regressions Predicting Parent- and Youth-Reported Externalizing Problems From the Testosterone × Cortisol or Testosterone × Cortisol × Personality Pathology Trait Interactions

Interaction terms	Parent report				Youth report			
	β	*p*	*B*	95% CI	β	*p*	*B*	95% CI
Testosterone × Cortisol	−0.11	.246	−0.98	[−2.65, 0.69]	−0.21	.032	−2.26	[−4.33, −0.19]
DIPSI higher-order factors								
T × C × Disagreeableness	**−0.25**	**.000**	**−2.05**	**[−2.95, −1.15]**	−0.11	.130	−1.26	[−2.90, 0.38]
T × C × Emotional Instability	**−0.29**	**.001**	**−2.22**	**[−3.55, −0.88]**	−0.02	.820	−0.20	[−1.89, 1.50]
DIPSI Disagreeableness facets								
T × C × Hyperexpressive Traits	−0.23	.005	−1.90	[−3.22, −0.58]	−0.15	.156	−1.26	[−3.00, 0.49]
T × C × Hyperactive Traits	−0.23	.004	−2.09	[−3.50, −0.68]	−0.10	.292	−0.93	[−2.67, 0.81]
T × C × Dominance-Egocentrism	**−0.24**	**.002**	**−2.09**	**[−3.38, −0.79]**	0.00	.972	0.04	[−2.18, 2.26]
T × C × Impulsivity	**−0.26**	**.000**	**−2.06**	**[−3.12, −1.01]**	−0.02	.836	−0.20	[−2.12, 1.72]
T × C × Irritable-Aggressive Traits	**−0.25**	**.000**	**−1.82**	**[−2.80, −0.84]**	−0.09	.265	−1.03	[−2.84, 0.79]
T × C × Disorderliness	−0.12	.149	−1.10	[−2.59, 0.40]	−0.11	.216	−1.19	[−3.08, 0.71]
T × C × Distraction	−0.15	.066	−1.42	[−2.95, 0.10]	−0.06	.561	−0.70	[−3.08, 1.68]
T × C × Risk Taking	−0.11	.221	−0.89	[−2.33, 0.55]	−0.02	.773	−0.33	[−2.55, 1.90]
T × C × Narcissistic Traits	−0.10	.343	−0.83	[−2.55, 0.90]	−0.04	.746	−0.32	[−2.29, 1.64]
T × C × Affective Lability	**−0.32**	**.000**	**−2.14**	**[−3.16, −1.12]**	−0.13	.153	−1.27	[−3.02, 0.48]
T × C × Resistance	−0.08	.405	−0.69	[−2.32, 0.94]	−0.01	.947	−0.07	[−2.05, 1.92]
T × C × Lack of Empathy	−0.24	.006	−1.78	[−3.04, −0.53]	0.05	.520	0.74	[−1.54, 3.02]
DIPSI Emotional Instability facets								
T × C × Dependency	**−0.32**	**.003**	**−2.37**	**[−3.89, −0.85]**	−0.03	.730	−0.37	[−2.48, 1.74]
T × C × Anxious Traits	−0.13	.189	−1.28	[−3.20, 0.64]	−0.12	.343	−0.95	[−2.92, 1.03]
T × C × Lack of Self-Confidence	**−0.27**	**.002**	**−1.90**	**[−3.09, −0.71]**	0.05	.599	0.54	[−1.48, 2.55]
T × C × Insecure Attachment	−0.13	.233	−1.05	[−2.78, 0.69]	−0.07	.470	−0.61	[−2.26, 1.05]
T × C × Submissiveness	−0.23	.050	−1.80	[−3.59, 0.00]	−0.05	.649	−0.54	[−2.87, 1.80]
T × C × Ineffective Coping	−0.22	.006	−1.86	[−3.19, −0.54]	−0.11	.198	−1.41	[−3.57, 0.75]
T × C × Separation Anxiety	−0.23	.042	−1.46	[−2.86, −0.06]	0.04	.750	0.28	[−1.48, 2.05]
T × C × Depressive Traits	−0.24	.021	−1.61	[−2.97, −0.25]	0.05	.597	0.44	[−1.21, 2.09]
T × C × Inflexibility	−0.20	.050	−1.96	[−3.92, 0.00]	−0.20	.076	−1.66	[−3.50, 0.18]

Note. CI = confidence interval (values in square brackets denote 95% confidence intervals of unstandardized regression coefficients); DIPSI = Dimensional Personality Symptom Itempool (De Clercq et al., 2006); T = testosterone; C = cortisol. The full regressions with 2-way interactions included age, sex, and time of waking at Step 1, as well as main effects for testosterone and cortisol at Step 2. The full regressions with 3-way interactions included age, sex, and time of waking at Step 1, main effects for testosterone, cortisol, and the personality pathology trait at Step 2, the 2-way interaction terms between testosterone, cortisol, and the personality pathology trait at Step 3, as well as the 3-way interaction term between testosterone, cortisol, and the personality pathology trait at Step 4. Bold = significant after Bonferroni correction.

moderated the T × C interaction effects predicting parent-reported broadband Externalizing Problems (see Table 3). As hypothesized, the T × C interaction did not predict parent-reported Externalizing Problems at low levels of PD traits [Disagreeableness: $b = 0.84$, $t(95) = 1.24$, $p = .219$; Emotional Instability: $b = 0.32$, $t(95) = 0.33$, $p = .742$]. At high levels of PD traits, however, the T slopes for parent-reported Externalizing Problems were significant at low C for both Disagreeableness ($b = 4.47$, $t(95) = 5.04$, $p < .001$) and Emotional Instability ($b = 6.19$, $t(95) = 4.32$, $p < .001$). These interactions were nonsignificant at high C levels for Emotional Instability: $b = -1.68$, $t(95) = -1.08$, $p = .283$, and Disagreeableness: $b = -2.05$, $t(95) = -2.12$, $p = .037$. To illustrate the overall pattern of results, findings were graphed at high and low values for T, C, and the PD trait (see Figure 1).

T × C Interaction Effects Moderated by DIPSI Facets

After Bonferroni correction, 4 of 12 Disagreeableness facets moderated the T × C interaction in predicting parent-reported Externalizing Problems ($p < .004$ for the 12 regressions for parent- and youth-report each). Dominance-Egocentrism ($\beta = -0.24$, $t(95) = -3.21$, $p = .002$), Impulsivity ($\beta = -0.26$, $t(95) = -3.88$, $p < .001$), Irritable-Aggressive Traits ($\beta = -0.25$, $t(95) = -3.69$, $p < .001$), and Affective Lability ($\beta = -0.32$, $t(95) = -4.18$, $p < .001$) all moderated the T × C interaction effects predicting parent-reported broadband Externalizing Problems (see Table 3). As hypothesized, the T × C interaction did not predict parent-reported Externalizing Problems at low levels of PD traits: (a) Dominance-Egocentrism: $b = 1.11$, $t(95) = 1.22$, $p = .224$; (b) Impulsivity: $b = 0.59$, $t(95) = 0.82$, $p = .413$; (c) Irritable-Aggressive Traits: $b = 0.07$, $t(95) = 0.10$, $p = .918$; and (d) Affective Lability: $b = 0.33$, $t(95) = 0.44$, $p = .663$. At high levels of PD traits, however, the T slopes for parent-reported Externalizing Problems were significant at low C for the following: (a) Dominance-Egocentrism: $b = 3.80$, $t(95) = 3.14$, $p = .002$; (b) Impulsivity: $b = 3.68$, $t(95) = 3.67$, $p < .001$; (c) Irritable-Aggressive Traits: $b = 5.16$, $t(95) = 4.61$, $p < .001$; and (d) Affective Lability: $b = 5.13$, $t(95) = 4.19$, $p < .001$. Consistent with the dual hormone hypothesis, none of these interactions were significant at high C levels: (a) Dominance-Egocentrism: $b = -2.32$, $t(95) = -1.66$, $p = .100$; (b) Impulsivity: $b = -2.29$, $t(95) = -2.10$, $p = .038$; (c) Irritable-Aggressive Traits: $b = -1.32$, $t(95) = -1.51$, $p = .135$; and (d) Affective Lability: $b = -2.26$, $t(95) = -2.25$, $p = .027$. To illustrate the overall pattern of results, findings for all four significant Disagreeableness

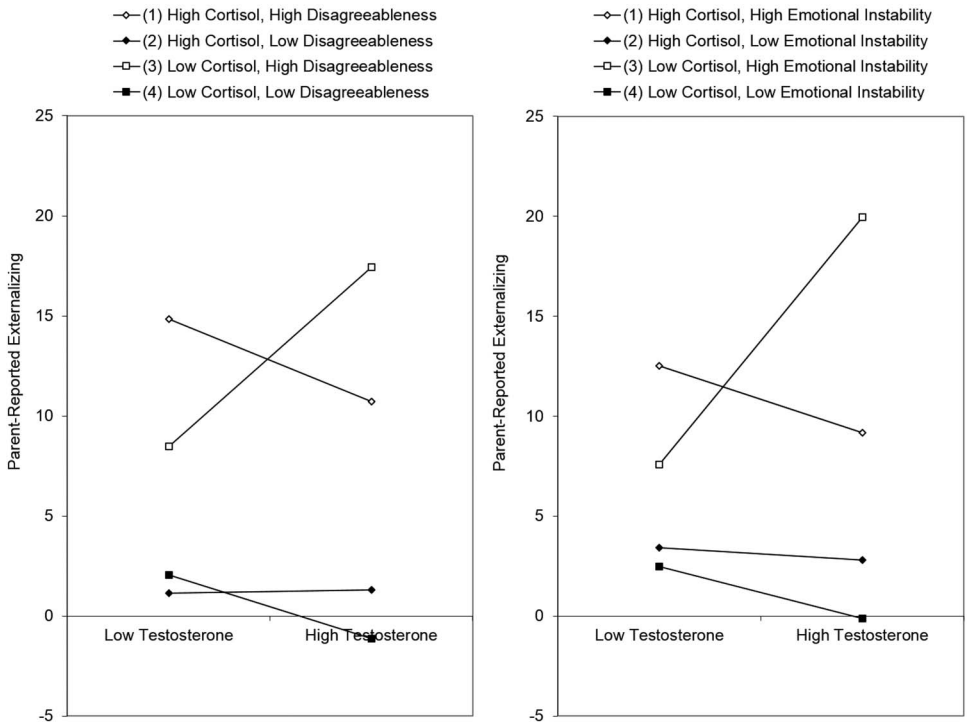

Figure 1. Interaction between cortisol and testosterone in predicting Externalizing Problems at high and low personality pathology.

facets were graphed at high and low values for T, C, and the Disagreeableness facet (see Figure 2).

After Bonferroni correction, 2 of 9 Emotional Instability facets moderated the T × C interaction in predicting parent-reported Externalizing Problems ($p < .006$ for the nine regressions for parent- and youth-report each). Dependency ($\beta = -0.32$, $t(95) = -3.10$, $p = .003$) and Lack of Self-Confidence ($\beta = -0.27$, $t(95) = -3.18$, $p = .002$) moderated the T × C interaction effects predicting parent-reported broadband Externalizing Problems. As hypothesized, the T × C interactions in predicting parent-reported Externalizing Problems were *not* significant at low levels of PD traits: (a) Dependency: $b = 0.15$, $t(95) = 0.17$, $p = .867$; and (b) Lack of Self-Confidence: $b = 0.23$, $t(95) = 0.26$, $p = .799$. At high levels of PD traits, however, the T slopes for parent-reported Externalizing Problems were significant at low C for the following: (a) Dependency: $b = 5.32$, $t(95) = 4.27$, $p < .001$; and (b) Lack of Self-Confidence: $b = 6.05$, $t(95) = 4.59$, $p < .001$. Consistent with the dual hormone hypothesis, *neither* of these interactions were significant at high C levels: (a) Dependency: $b = -1.81$, $t(95) = -0.98$, $p = .327$; and (b) Lack of Self-Confidence: $b = -0.36$, $t(95) = -0.28$, $p = .780$. To illustrate the overall pattern of results, findings for the 2 significant Emotional Instability facets were graphed at high and low values for T, C, and the Emotional Instability facet (see Figure 3).

Discussion

The current study provides the first evidence for the dual-hormone hypothesis of C and T in the prediction of externalizing problems in a mixed-sex, community, adolescent sample, but only in the context of personality pathology traits. Specifically, this approach suggests that the joint influence of T and C on adolescent externalizing is especially pronounced at high levels of Disagreeableness and Emotional Instability personality traits, for parent-reported traits and behaviors. Previous investigations in adolescents have been limited to clinical and incarcerated samples of male participants (Dabbs et al., 1991; Popma et al., 2007) in predicting externalizing problems. The present findings suggest that hormonal influences on core aspects of externalizing extend to adolescents from the community and to adolescents of both sexes, when considering variations in more broadly distributed personality traits.

Upon analyzing moderation by PD traits, evidence for the dual-hormone hypothesis (operationalized as a significant T × C interaction) emerged. Specifically, hypotheses at the higher-order level were supported, such that moderation occurred for Disagreeableness and Emotional Instability traits in the prediction of broadband Externalizing Problems. Simple slopes analyses revealed virtually identical effects across all these interactions: high T predicted greater externalizing when C was low, but only in the context of high PD traits. Thus, failure to observe robust T × C interactions when PD traits are not considered may be a result of the heterogeneity of normal-range externalizing characteristics in a mixed-sex, community sample of youth. Future research will determine whether PD traits continue to serve as powerful tools to elucidate dual-hormone hypothesis effects in normative, mixed-sex populations.

Analyses at the lower-order trait level were exploratory, because of the lack of previous research on this topic. Thus, a stringent

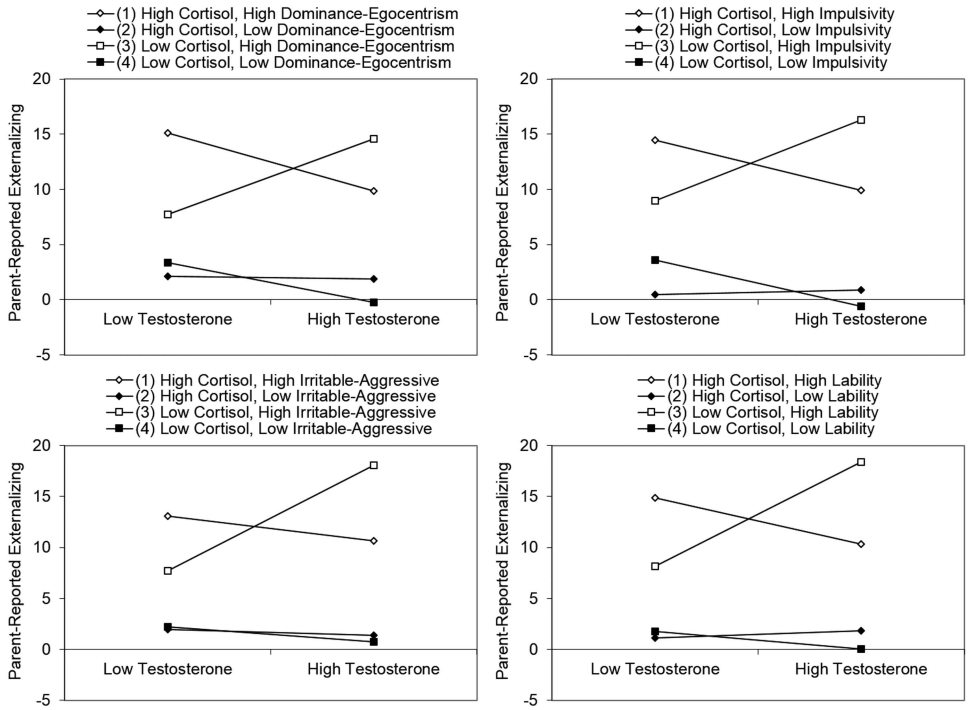

Figure 2. Interaction between cortisol and testosterone in predicting Externalizing Problems at high and low Disagreeableness facets.

significance level was applied to these results. These analyses suggested that several facets from the Disagreeableness trait domain were driving these findings: Dominance-Egocentrism, Impulsivity, Irritable-Aggressive Traits, and Affective Lability. These traits are largely consistent with the broader dual-hormone hypothesis literature on normal-range social dominance characteristics that appear to be relevant for T-C functioning. Indeed, on probing these interaction effects, all facets followed the same pattern, demonstrating T effects on externalizing when C levels were low. Two facets from the Emotional Instability domain also emerged as significant: Dependency and Lack of Self-Confidence. The direction of these effects followed a similar pattern: the T × C interaction was associated with Externalizing Problems, but only for youth showing high-risk levels of these PD facets. Given the exploratory nature of these analyses, however, they should be replicated in future studies.

The results from this study provide a number of additional contributions to the growing literature examining the dual-hormone hypothesis. For example, the unique characteristics of this sample (inclusion of males and females, broad adolescent age range, community participants) serve to bridge findings from clinical and incarcerated samples of adolescents (Dabbs et al., 1991; Popma et al., 2007) and nonclinical (primarily college student) samples of adults (Denson, Mehta, et al., 2013; Geniole, Carré, & McCormick, 2011; Mehta & Josephs, 2010; Zilioli & Watson, 2012). Hormonal links to youth antisocial behavior are often stronger in clinical samples than in community samples (Alink et al., 2008; Popma et al., 2007), which may reflect a higher threshold for hormonal influences on externalizing problems at earlier ages. The findings from the present study suggest that this threshold may be moderated by normatively distributed social dominance constructs (such as PD traits), which are easily detectable across adolescence. It may also be the case that aspects of pubertal development, such as pubertal timing, are important moderators of hormonal influences on behavior (Susman et al., 2010), which are largely masked in adult samples (when pubertal variables such as timing are infrequently measured and pubertal status has reached asymptote). Indeed, the current sample was innovative in including adolescents across a broad age range, but this age range also limited the ability to detect age-specific findings across adolescent development. We suggest that future studies examine such effects much more closely across different ages and critical developmental periods, such as the pubertal transition.

An additional implication from this study highlights the importance and potential utility in gathering other-informant reports on personality pathology. The significant findings presented here were significant for parent-reported PD traits, with virtually no predictive validity found for youth self-reported PD traits, despite parent–child agreement of similar magnitude ($rM_{avg} = .29$) to that found for other psychopathology constructs (Achenbach, McConaughy, & Howell, 1987). Although research with adults is often restricted to self-report data, these results suggest that other informants (such as parents, in the present study) may have better insight into PD traits, providing more valid measures of these constructs than would be obtained from self-reports alone. These findings correspond with evidence from adult populations that informant reports may provide incremental variance beyond self-reports when measuring PD constructs (Oltmanns & Turkheimer, 2009).

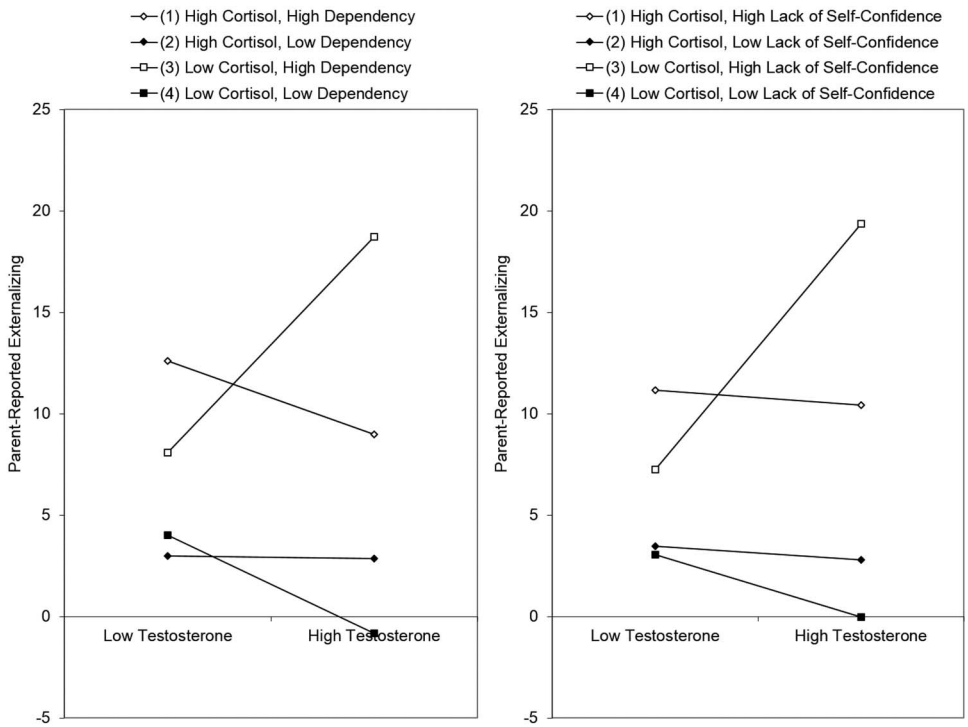

Figure 3. Interaction between cortisol and testosterone in predicting Externalizing Problems at high and low Emotional Instability facets.

Studies have also demonstrated that the effects of T on negative adolescent outcomes such as risky or delinquent behaviors depend on exposure to high-risk environmental contexts (Booth, Johnson, Granger, Crouter, & McHale, 2003; Rowe, Maughan, Worthman, Costello, & Angold, 2004). That is, hormones show a complex interplay with environmental context, and covary with adaptive or maladaptive pathways, depending on environmental features. Recent research with adults has also paid increasing attention to contextual features, such as social threat or challenge, and has suggested that attention to environmental context may ultimately provide a more nuanced view of the mechanisms underlying the joint T-C relationship with behavior (Mehta & Josephs, 2010; Denson, Mehta, et al., 2013; Zilioli & Watson, 2012). The examination of additional moderators such as PD traits represents an excellent mechanism for further tests of Person × Environment Fit, as the same personality traits can also be both adaptive and maladaptive in different circumstances. Furthermore, given the relative ease with which PD traits can be measured (compared to biological indicators), they offer a clinically tractable mechanism for examining Person × Environment Fit that may help explain why some adolescents with high-risk hormone profiles are able to channel their behavior in adaptive ways. Attention to environmental context may also prove useful in incorporating estimates of hormonal reactivity to different types of environmental stimuli. At least one study to date has found T to interact with C reactivity, rather than baseline C, in predicting antisocial features of psychopathy in a primarily male sample of community adults (Glenn et al., 2011). Examination of hormonal reactivity will allow for controlled manipulation of environmental features (e.g., social vs. nonsocial stressors)—and differential hormonal reactivity to such features—to further our understanding of contextual moderation of the dual-hormone hypothesis.

Although main effects of C and T were not predicted (and largely not found), analyses revealed multiple findings for the relevance of the T × C interaction on parent-reported externalizing problems for youth with high levels of externalizing-relevant PD traits. Various hypotheses have been put forth to explain the interactive effect of T and C on socially dominant behaviors, including the possibility that inhibitory influences of high C counteract high T levels either at the behavioral or neuroendocrinological levels, or both (Dabbs et al., 1991; Popma et al., 2007; Mehta & Josephs, 2010; Zilioli & Watson, 2012). Probing the significant interactions in the present study largely supported the existing evidence for the inhibitory effect of high C on T, such that T main effects were found only when C levels were low. It is also important for future studies to expand the examination of personality traits considered as potential moderators of hormone–behavior associations, with recognition that such personality traits may not directly map on to target hormones or behaviors in clear and straightforward ways.

The sample size, although consistent with other studies on this topic (e.g., Dabbs et al., 1991; Popma et al., 2007), may have limited the ability of the present study to detect significant interaction effects. Future studies with larger samples will be important to determine whether such effects are meaningful at the broader population level, and will also prove useful in focused analyses examining potential age-specific effects. Additional limitations of the study were the storage of saliva at −20 °C instead of −80 °C,

which some research recommends because higher temperatures can be problematic for sex hormones (e.g., Dabbs, 1991; Granger et al., 2004), and the single measurement of T and C taken at various points in the afternoon. Although all samples were collected between noon and sundown and we were able to control for time of waking, future investigations could improve on measurement by standardizing time of day across all participants and collecting multiple measures of T and C, preferably across multiple days. It is important to emphasize, however, that recent research has found that measurements of C and T 14 days apart are highly correlated (rs = .65–.78; Liening et al., 2010), supporting the temporal stability of such measurements. A related point is the need for future studies to consider the potential differences between acute and chronic effects of T and C on externalizing behaviors. Although the present study cannot disentangle such effects, it is possible that acutely versus chronically high levels of T and C relate differentially with risk-taking behavior (Coates & Herbert, 2008). Thus, this remains an important direction for future research.

Taken together, these results provide the first evidence for the dual-hormone hypothesis in a mixed-sex sample of community adolescents. Importantly, these findings suggest that externalizing-relevant personality pathology traits are important moderators of the T × C relationship, which may be masked in more normative samples if personality moderators are not considered. These effects were only significant in parent-report of personality and behavior, consistent with work in adult populations demonstrating limited validity of self-report when assessing PD traits. The effects were quite robust, however, across broadband Externalizing Problems and underscore the impact of the joint relationship of the HPG and HPA axes in disinhibitory psychopathology in community youth.

References

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin, 85,* 1275–1301. doi:10.1037/0033-2909.85.6.1275

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin, 101,* 213–232. doi:10.1037/0033-2909.101.2.213

Achenbach, T. M., & Rescorla, L. A. (2001). *Manual for the ASEBA school-age forms & profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth & Families.

Alink, L. R. A., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Mesman, J., Juffer, F., & Koot, H. M. (2008). Cortisol and externalizing problems in children and adolescents: Mixed meta-analytic evidence for the inverse relation of basal cortisol and cortisol reactivity with externalizing problems. *Developmental Psychobiology, 50,* 427–450. doi:10.1002/dev.20300

Allport, G. W. (1937). *Personality: A psychological interpretation*. New York, NY: Holt, Rinehart & Winston.

Archer, J., Graham-Kevan, N., & Davies, M. (2005). Testosterone and aggression: A reanalysis of Book, Starzyk, and Quinsey's (2001) study. *Aggression and Violent Behavior, 10,* 241–261. doi:10.1016/j.avb.2004.01.001

Baker, L. A., Jacobson, K. C., Raine, A., Lozano, D. I., & Bezdjian, S. (2007). Genetic and environmental bases of childhood antisocial behavior: A multi-informant twin study. *Journal of Abnormal Psychology, 116,* 219–235. doi:10.1037/0021-843X.116.2.219

Booth, A., & Dabbs, J. M. (1993). Testosterone and men's marriages. *Social Forces, 72,* 463–477.

Booth, A., Johnson, D. V., Granger, D. A., Crouter, A. C., & McHale, S. (2003). Testosterone and child and adolescent adjustment: The moderating role of parent-child relationships. *Developmental Psychology, 39,* 85–98. doi:10.1037/0012-1649.39.1.85

Burnstein, K. L., Maiorino, C. A., Dai, J. L., & Cameron, D. J. (1995). Androgen and glucocorticoid regulation of androgen receptor cDNA expression. *Molecular and Cellular Endocrinology, 115,* 177–186. doi:10.1016/0303-7207(95)03688-1

Burt, S. A. (2012). How do we optimally conceptualize the heterogeneity within antisocial behavior? An argument for aggressive versus non-aggressive behavioral dimensions. *Clinical Psychology Review, 32,* 263–279. doi:10.1016/j.cpr.2012.02.006

Carré, J. M., & McCormick, C. M. (2008). Aggressive behavior and change in salivary testosterone concentrations predict willingness to engage in a competitive task. *Hormones and Behavior, 54,* 403–409. doi:10.1016/j.yhbeh.2008.04.008

Chen, S., Wang, J., Yu, G., Liu, W., & Pearce, D. (1997). Androgen and glucocorticoid receptor heterodimer formation. A possible mechanism for mutual inhibition of transcriptional activity. *The Journal of Biological Chemistry, 272,* 14087–14092. doi:10.1074/jbc.272.22.14087

Coates, J. M., & Herbert, J. (2008). Endogenous steroids and financial risk taking on a London trading floor. *PNAS Proceedings of the National Academy of Sciences of the United States of America, 105,* 6167–6172. doi:10.1073/pnas.0704025105

Dabbs, J. M. (1991). Salivary testosterone measurements: Collecting, storing, and mailing saliva samples. *Physiology & Behavior, 49,* 815–817. doi:10.1016/0031-9384(91)90323-G

Dabbs, J. M., Jurkovic, G. J., & Frady, R. L. (1991). Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology, 19,* 469–478. doi:10.1007/BF00919089

Davies, P. T., Sturge-Apple, M. L., & Cicchetti, D. (2011). Interparental aggression and children's adrenocortical reactivity: Testing an evolutionary model of allostatic load. *Development and Psychopathology, 23,* 801–814. doi:10.1017/S0954579411000319

De Clercq, B., De Fruyt, F., Van Leeuwen, K., & Mervielde, I. (2006). The structure of maladaptive personality traits in childhood: A step toward an integrative developmental perspective for *DSM-V*. *Journal of Abnormal Psychology, 115,* 639–657. doi:10.1037/0021-843X.115.4.639

Denson, T. F., Mehta, P. H., & Tan, D. H. (2013). Endogenous testosterone and cortisol jointly influence reactive aggression in women. *Psychoneuroendocrinology, 38,* 416–424. doi:10.1016/j.psyneuen.2012.07.003

Denson, T. F., Ronay, R., von Hippel, W., & Schira, M. M. (2013). Risk for aggression: Endogenous testosterone and cortisol modulate neural responses to induced anger control. *Social Neuroscience, 8,* 165–177. doi:10.1080/17470919.2012.655425

DeYoung, C. G., & Clark, R. (2012). The gene in its natural habitat: The importance of gene-trait interactions. *Development and Psychopathology, 24,* 1307–1318. doi:10.1017/S0954579412000727

Dick, D. M., Aliev, F., Wang, J. C., Grucza, R. A., Schuckit, M., Kuperman, S., . . . Goate, A. (2008). Using dimensional models of externalizing psychopathology to aid in gene identification. *Archives of General Psychiatry, 65,* 310–318. doi:10.1001/archpsyc.65.3.310

Ehrenkranz, J., Bliss, E., & Sheard, M. H. (1974). Plasma testosterone: Correlation with aggressive behavior and social dominance in man. *Psychosomatic Medicine, 36,* 469–475.

Geniole, S. N., Carré, J. M., & McCormick, C. M. (2011). State, not trait, neuroendocrine function predicts costly reactive aggression in men after social exclusion and inclusion. *Biological Psychology, 87,* 137–145. doi:10.1016/j.biopsycho.2011.02.020

Gettler, L. T., McDade, T. W., & Kuzawa, C. W. (2011). Cortisol and testosterone in Filipino young adult men: Evidence for co-regulation of both hormones by fatherhood and relationship status. *American Journal of Human Biology, 23,* 609–620. doi:10.1002/ajhb.21187

Glenn, A. L., Raine, A., Schug, R. A., Gao, Y., & Granger, D. A. (2011). Increased testosterone to cortisol ratio in psychopathy. *Journal of Abnormal Psychology, 120,* 389–399. doi:10.1037/a0021407

Granger, D. A., Schwartz, E. B., Booth, A., & Arentz, M. (1999). Salivary testosterone determination in studies of child health and development. *Hormones and Behavior, 35,* 18–27. doi:10.1006/hbeh.1998.1492

Granger, D. A., Shirtcliff, E. A., Booth, A., Kivlighan, K. T., & Schwartz, E. B. (2004). The "trouble" with salivary testosterone. *Psychoneuroendocrinology, 29,* 1229–1240. doi:10.1016/j.psyneuen.2004.02.005

Gunnar, M. R., Kryzer, E., Van Ryzin, M. J., & Phillips, D. A. (2011). The import of the cortisol rise in child care differs as a function of behavioral inhibition. *Developmental Psychology, 47,* 792–803. doi:10.1037/a0021902

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis*. New York, NY: The Guilford Press.

Johnson, E. O., Kamilaris, T. C., Chrousos, G. P., & Gold, P. W. (1992). Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neuroscience and Biobehavioral Reviews, 16,* 115–130. doi:10.1016/S0149-7634(05)80175-7

Johnson, S. L., Leedom, L. J., & Muhtadie, L. (2012). The dominance behavioral system and psychopathology: Evidence from self-report, observational, and biological studies. *Psychological Bulletin, 138,* 692–743. doi:10.1037/a0027503

Jones, S. E., Miller, J. D., & Lynam, D. R. (2011). Personality, antisocial behavior, and aggression: A meta-analytic review. *Journal of Criminal Justice, 39,* 329–337. doi:10.1016/j.jcrimjus.2011.03.004

Josephs, R. A., Sellers, J. G., Newman, M. L., & Mehta, P. H. (2006). The mismatch effect: When testosterone and status are at odds. *Journal of Personality and Social Psychology, 90,* 999–1013. doi:10.1037/0022-3514.90.6.999

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19,* 313–333. doi:10.1016/0306-4530(94)90013-2

Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin, 136,* 768–821. doi:10.1037/a0020327

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

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56,* 921–926. doi:10.1001/archpsyc.56.10.921

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2,* 111–133. doi:10.1146/annurev.clinpsy.2.022305.095213

Krueger, R. F., McGue, M., & Iacono, W. G. (2001). The higher-order structure of common DSM mental disorders: Internalization, externalization, and their connections to personality. *Personality and Individual Differences, 30,* 1245–1259. doi:10.1016/S0191-8869(00)00106-9

Krueger, R. F., & South, S. C. (2009). Externalizing disorders: Cluster 5 of the proposed meta-structure for DSM-V and ICD-11. *Psychological Medicine, 39,* 2061–2070. doi:10.1017/S0033291709990328

Lahey, B. B., & Waldman, I. D. (2007). Personality dispositions and the development of violence and conduct problems. In D. J. Flannery, A. T. Vazsonyi, & I. D. Waldman (Eds.), *The Cambridge handbook of violent behavior and aggression* (pp. 260–287). New York, NY: Cambridge University Press. doi:10.1017/CBO9780511816840.013

Liening, S. H., Stanton, S. J., Saini, E. K., & Schultheiss, O. C. (2010). Salivary testosterone, cortisol, and progesterone: Two-week stability, interhormone correlations, and effects of time of day, menstrual cycle, and oral contraceptive use on steroid hormone levels. *Physiology & Behavior, 99,* 8–16. doi:10.1016/j.physbeh.2009.10.001

Martel, M. M. (2009). A new perspective on Attention-Deficit/Hyperactivity Disorder: Emotion dysregulation and trait models. *Journal of Child Psychology and Psychiatry, 50,* 1042–1051. doi:10.1111/j.1469-7610.2009.02105.x

Mehta, P. H., & Beer, J. S. (2010). Neural mechanisms of the testosterone-aggression relation: The role of orbitofrontal cortex. *Journal of Cognitive Neuroscience, 22,* 2357–2368. doi:10.1162/jocn.2009.21389

Mehta, P. H., Jones, A. C., & Josephs, R. A. (2008). The social endocrinology of dominance: Basal testosterone predicts cortisol changes and behavior following victory and defeat. *Journal of Personality and Social Psychology, 94,* 1078–1093. doi:10.1037/0022-3514.94.6.1078

Mehta, P. H., & Josephs, R. A. (2006). Testosterone change after losing predicts the decision to compete again. *Hormones and Behavior, 50,* 684–692. doi:10.1016/j.yhbeh.2006.07.001

Mehta, P. H., & Josephs, R. A. (2010). Testosterone and cortisol jointly regulate dominance: Evidence for a dual-hormone hypothesis. *Hormones and Behavior, 58,* 898–906. doi:10.1016/j.yhbeh.2010.08.020

Mehta, P. H., Wuehrmann, E., & Josephs, R. A. (2009). When are low testosterone levels advantageous?: The moderating role of individual versus intergroup competition. *Hormones and Behavior, 56,* 158–162. doi:10.1016/j.yhbeh.2009.04.001

Miller, J. D., & Lynam, D. (2001). Structural models of personality and their relation to antisocial behavior: A meta-analytic review. *Criminology, 39,* 765–798. doi:10.1111/j.1745-9125.2001.tb00940.x

Newman, M. L., Sellers, J. G., & Josephs, R. A. (2005). Testosterone, cognition, and social status. *Hormones and Behavior, 47,* 205–211. doi:10.1016/j.yhbeh.2004.09.008

Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 47,* 395–422. doi:10.1111/j.1469-7610.2006.01612.x

Olson, S. L., Schilling, E. M., & Bates, J. E. (1999). Measurement of impulsivity: Construct coherence, longitudinal stability, and relationship with externalizing problems in middle childhood and adolescence. *Journal of Abnormal Child Psychology, 27,* 151–165. doi:10.1023/A:1021915615677

Oltmanns, T. F., & Turkheimer, E. (2009). Person perception and personality pathology. *Current Directions in Psychological Science, 18,* 32–36. doi:10.1111/j.1467-8721.2009.01601.x

Oyegbile, T. O., & Marler, C. A. (2005). Winning fights elevates testosterone levels in California mice and enhances future ability to win fights. *Hormones and Behavior, 48,* 259–267. doi:10.1016/j.yhbeh.2005.04.007

Petersen, A., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence, 17,* 117–133. doi:10.1007/BF01537962

Popma, A., Vermeiren, R., Geluk, C. A. M. L., Rinne, T., van den Brink, W., Knol D. L., . . . Doreleijers, T. A. (2007). Cortisol moderates the relationship between testosterone and aggression in delinquent male adolescents. *Biological Psychiatry, 61,* 405–411. doi:10.1016/j.biopsych.2006.06.006

Rowe, R., Maughan, B., Worthman, C. M., Costello, E. J., & Angold, A. (2004). Testosterone, antisocial behavior, and social dominance in boys: Pubertal development and biosocial interaction. *Biological Psychiatry, 55,* 546–552. doi:10.1016/j.biopsych.2003.10.010

Schulkin, J. (2007). Autism and the amygdala: An endocrine hypothesis. *Brain and Cognition, 65,* 87–99. doi:10.1016/j.bandc.2006.02.009

Schulkin, J., Gold, P. W., & McEwen, B. (1998). Induction of corticotropin-releasing hormone gene expression by glucocorti-coids: Implications for understanding the states of fear and anxiety and allostatic load. *Psychoneuroendocrinology, 23,* 219–243. doi:10.1016/S0306-4530(97)00099-1

Sellers, J. G., Mehl, M. R., & Josephs, R. A. (2007). Hormones and personality: Testosterone as a marker of individual differences. *Journal of Research in Personality, 41,* 126–138. doi:10.1016/j.jrp.2006.02.004

Smith, R. G., Syms, A. J., Nag, A., Lerner, S., & Norris, J. S. (1985). Mechanism of the glucocorticoid regulation of growth of the androgen-sensitive prostate-derived R3327H-G8–A1 tumor cell line. *The Journal of Biological Chemistry, 260,* 12454–12463.

Susman, E. J., Dockray, S., Granger, D. A., Blades, K. T., Randazzo, W., Heaton, J. A., & Dorn, L. D. (2010). Cortisol and alpha amylase reactivity and timing of puberty: Vulnerabilities for antisocial behaviour in young adolescents. *Psychoneuroendocrinology, 35,* 557–569. doi:10.1016/j.psyneuen.2009.09.004

Tackett, J. L. (2006). Evaluating models of the personality–psychopathology relationship in children and adolescents. *Clinical Psychology Review, 26,* 584–599. doi:10.1016/j.cpr.2006.04.003

Tackett, J. L., Balsis, S., Oltmanns, T. F., & Krueger, R. F. (2009). A unifying perspective on personality pathology across the lifespan: Developmental considerations for DSM-V. *Development and Psychopathology, 21,* 687–713. doi:10.1017/S095457940900039X

Tackett, J. L., & De Clercq, B. (April 2009). *Assessing childhood precursors to personality pathology: Validating the English version of the DIPSI.* Paper presented at 10th annual meeting of the European Conference on Psychological Assessment, Ghent, Belgium.

Tackett, J. L., Herzhoff, K., Reardon, K., De Clercq, B., & Sharp, C. (2014). The externalizing spectrum in youth: Incorporating personality pathology. *Journal of Adolescence, 37,* 659–668. doi:10.1016/j.adolescence.2013.10.009

Tackett, J. L., Kushner, S. K., Josephs, R. A., Harden, K. P., Page-Gould, E., & Tucker-Drob, E. M. (2014). Hormones: Empirical contribution: Cortisol reactivity and recovery in the context of adolescent personality disorder. *Journal of Personality Disorders, 28,* 25–39. doi:10.1521/pedi.2014.28.1.25

Tilbrook, A. J., Turner, A. I., & Clarke, I. J. (2000). Effects of stress on reproduction in nonrodent mammals: The role of glucocorticoids and sex differences. *Reviews of Reproduction, 5,* 105–113. doi:10.1530/ror.0.0050105

Tremblay, R. E., Schaal, B., Boulerice, B., Arseneault, L., Soussignan, R. G., Paquette, D., & Laurent, D. (1998). Testosterone, physical aggression, dominance, and physical development in early adolescence. *International Journal of Behavioral Development, 22,* 753–777. doi:10.1080/016502598384153

Trull, T. J., & Sher, K. J. (1994). Relationship between the five-factor model of personality and Axis I disorders in a nonclinical sample. *Journal of Abnormal Psychology, 103,* 350–360. doi:10.1037/0021-843X.103.2.350

Urry, H. L., van Reekum, C. M., Johnstone, T., Kalin, N. H., Thurow, M. E., Schaefer, H. S., Jackson, C. A., . . . Davidson, R. J. (2006). Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. *The Journal of Neuroscience, 26,* 4415–4425. doi:10.1523/JNEUROSCI.3215-05.2006

van Honk, J., Harmon-Jones, E., Morgan, B. E., & Schutter, D. J. L. G. (2010). Socially explosive minds: The triple imbalance hypothesis of reactive aggression. *Journal of Personality, 78,* 67–94. doi:10.1111/j.1467-6494.2009.00609.x

van Honk, J., Schutter, D. J. L. G., Hermans, E. J., Piutman, P., Tuiten, A., & Koppeschaar, H. (2004). Testosterone shifts the balance between sensitivity for punishment and reward in healthy young women. *Psychoneuroendocrinology, 29,* 937–943. doi:10.1016/j.psyneuen.2003.08.007

Widiger, T. A., & Smith, G. T. (2008). Personality and psychopathology. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (3rd ed.) (pp. 743–769). New York, NY: Guilford Press.

Zilioli, S., & Watson, N. V. (2012). The hidden dimensions of the competition effect: Basal cortisol and basal testosterone jointly predict changes in salivary testosterone after social victory in men. *Psychoneuroendocrinology, 37,* 1855–1865. doi:10.1016/j.psyneuen.2012.03.022

Zyphur, M. J., Narayanan, J., Koh, G., & Koh, D. (2009). Testosterone–status mismatch lowers collective efficacy in groups: Evidence from a slope-as-predictor multilevel structural equation model. *Organizational Behavior and Human Decision Processes, 110,* 70–79. doi:10.1016/j.obhdp.2009.05.004