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Antecedents and outcomes associated with altered neuroendocrine coupling in a young adolescent sample

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Abstract of the Dissertation

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Previous research has identified puberty as a time of marked vulnerability to negative psychological outcomes, despite the fact that most individuals navigate this transition with little disturbance. The current study investigated neuroendocrine coupling, or the extent to which hormone reactivity is correlated, as a potential mechanism explaining the relationship between puberty and negative outcomes, as well as investigating early life stressors that may influence hormone coupling patterns. Cortisol, DHEA, and testosterone levels were collected from 394 9-year-old children as part of a larger longitudinal study investigating temperament and psychopathology; previously collected information about parenting styles, parental psychopathology, and parental marital discord were examined as predictors of variations in hormone coupling in children, while child psychopathology was examined as an outcome of altered hormone coupling patterns. Results from the current study provided evidence of hormone coupling in 9-year-old children, and identified BMI as a predictor of the strength of hormone coordination; additionally, this study found limited support for the impact of parenting styles on later hormone functioning. Finally, hormone coupling was found to be associated with lifetime anxiety disorder diagnoses. The current study added to the existing literature on mechanisms by which puberty and negative psychological outcomes are related, and generated important questions for future research which will further our understanding of the biological processes surrounding adolescent development and their relation to the emergence of psychiatric illness.

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List of Abbreviations

ADHD	Attention-deficit hyperactivity disorder
BMI	Body mass index
CBCL	Child Behavior Checklist
DAS	Dyadic Adjustment Scale
DD	Dysthymic disorder
DELFIA	Dissociation-enhanced lanthanide fluorescent immunoassay
DHEA	Dehydroepiandrosterone
FSH	Follicle stimulating hormone
GnRH	Gonadotropin releasing hormone
HPA	Hypothalamic-pituitary-adrenal
HPG	Hypothalamic-pituitary-gonadal
ICC	Intraclass correlation
K-SADS	Kiddie Schedule for Affective Disorders
LH	Leutinizing hormone
MDD	Major depressive disorder
MLM	Multi-level modeling
NOS	Not otherwise specified
ODD	Oppositional defiant disorder
PAPA	Preschool Age Psychiatric Assessment
PDS	Pubertal Development Scale
PSDQ	Parenting Styles and Dimensions Questionnaire
SCID	Structured Clinical Interview for DSM-IV
SD	Standard deviation

Introduction

Puberty, the transition from childhood to adulthood, has recently been conceptualized not as a singular event, but a series of hormonal, neural, psychological, and social changes taking place over many years. While all healthy adolescents experience these changes, there has been growing interest in individual differences that may alter or disrupt typical hormonal processes during puberty. This is especially true given research showing that the rates of many negative outcomes, including psychological disorders and serious risk-taking behaviors, rise during and closely after puberty (Downing & Bellis, 2009; Graber, 2003; Hyde, Mezulis, & Abramson, 2008).

The myriad developmental events of puberty are primarily initiated through hormonal changes, many of which occur well before puberty is outwardly apparent. During middle childhood (between ages 6 and 9; Bordini & Rosenfield, 2011), pubertal changes begin in the hypothalamic-pituitary-gonadal (HPG) axis with the release of gonadotropin releasing hormone (GnRH) from the hypothalamus, which then leads to increasing levels of leutinizing hormone (LH) and follicle stimulating hormone (FSH) from the pituitary gland. Sex hormones, including testosterone and estrogen, are then released from the gonads, and are responsible for both secondary sex characteristics and neural maturation during adolescence; the release of sex hormones and subsequent physical changes are known as gonadarche (Rosenfield et al., 2008; Saenger & Dimartino-Nardi, 2001; Styne, 2004).

As the HPG axis is maturing, the hypothalamic-pituitary-adrenal (HPA) axis is also maturing. While the HPA axis is primarily known for its role in regulating stress responding, it also plays an important role in puberty (Dorn, Dahl, Woodward, & Biro,

2006). The HPA axis regulates cortisol and dehydroepiandrosterone (DHEA), and both hormones are released in increasing amounts when an individual is exposed to environmental stress. Although these hormones are released throughout the lifespan, previous research has shown that HPA activity increases during puberty, either as one of many pubertal processes or as a consequence of other hormonal changes (Gunnar et al., 2009; Walker, Walder, & Reynolds, 2001). DHEA also plays a role in the physical maturation associated with puberty, as rising levels of the hormone are associated with the development of pubic hair, oily skin, and voice changes; this rise in DHEA and subsequent physical outcomes are referred to as adrenarche (Auchus & Rainey, 2004). While DHEA acts predominantly as a sex hormone during puberty, it returns to its role as a stress hormone following adolescence (Hucklebridge, Hussain, Evans, & Clow, 2005).

Despite the complexity of these processes, most adolescents progress through puberty without experiencing any marked negative outcomes; others, however, have a difficult transition to adulthood. As such, previous research has investigated how individual differences may interact with changes around puberty to impact adolescent behavior. Pubertal timing has been an important area of interest, with research demonstrating that early pubertal timing (relative to one's peers) is associated with negative behavioral outcomes including depression, increased antisocial and risk-taking behavior, earlier initiation of sexual activity, and substance abuse (Angold, Costello, & Worthman, 1998; Brooks-Gunn et al., 1985; Caspi & Moffitt, 1991; Conley & Rudolph, 2009; see Mendle, Turkheimer & Emery, 2007 for a review). These findings have led to a subsequent interest in understanding the mechanisms by which hormonal processes are disrupted or altered for some individuals during puberty, especially in light of research

suggesting that environmental stress can impact the pubertal transition (Belsky et al., 2007; Ellis & Garber, 2000; Graber, Brooks-Gunn, & Warren, 1995; Romans, Martin, Gendall, & Herbison, 2003; Saxbe & Repetti, 2009).

The impact of early life stress on later cortisol functioning has been studied extensively (see Gunnar & Aquendo, 2007, for a review). Previous research suggests that early exposure to negative life events, including maltreatment, abuse, or neglect, is related to HPA axis dysfunction; this can take the form of increased or decreased cortisol reactivity, neither of which is desirable for optimal physiological or psychological functioning in the face of stress (Cicchetti & Rogosch, 2001; Gunnar, 2000; Heim et al., 2000, 2002). The relationship between negative or unresponsive parenting and HPA axis dysfunction has also been widely investigated. Using a longitudinal design, Essex and colleagues (2011) explored how negative parenting before age 5 impacted HPA axis functioning between the ages of 9 and 15. Their findings suggested that children who were exposed to parental depression (which has been associated with negative parenting and other forms of stress; Hammen & Shih, 2014; Lovejoy, Graczyk, O'Hare, & Neuman, 2000) or familial expressed anger had lower than average morning cortisol levels at age 9; in contrast, children who were exposed to *both* stressors demonstrated higher than average cortisol levels at age 9, suggesting that HPA reactivity to stress differs based on the quality and magnitude of early life stress. Dougherty, Tolep, Smith, & Rose (2013) also found that maternal depression interacted with maternal hostility to predict greater cortisol reactivity to a lab stressor in preschool-age children. Additionally, low levels of structure (e.g., control and consistency) have been demonstrated to be associated with higher basal cortisol, as well as heightened cortisol reactivity following a lab stressor

(Ellenbogen & Hodgins, 2009). Conversely, Marsman et al. (2012) found that parental warmth was related to lower basal cortisol levels in adolescents.

Little research has investigated the effects of early life stress and negative family experiences on DHEA and testosterone, however. One notable exception is Ellis and Essex (2007), who assessed negative family environment at a mean age of 4 years, and subsequently measured DHEA at age 7. DHEA levels were dichotomized to create "adrenarcheal" and "preadrenarcheal" groups because many children did not have detectable levels of DHEA. Parents of children in the adrenarcheal group reported lower levels of authoritative parenting and higher levels of negativity and dissatisfaction with their children's behavior.

The relationship between hormone levels across time, or neuroendocrine coupling, may be an additional factor to consider when investigating how early life stress influences hormonal processes. Research in animal samples (including rats and guinea pigs) suggests that in healthy adults, the HPG and HPA axes regulate one another, such that HPA axis functioning is inhibited when higher concentrations of HPG hormones are present, and vice versa (Lürzel, Kaiser, Krüger, & Sachser, 2011; Vaiu, 2002). This arrangement is advantageous as steroid hormones, such as testosterone, can protect the organism against the deleterious effects of excessive cortisol (including memory deficits, cognitive dysfunction, and deficits in processing of emotional information; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; Prickaerts & Steckler, 2005), as well as regulate approach behavior in threatening social situations (Toufexis & Wilson, 2012; Wirth & Schultheiss, 2007). Inversely, during times of high stress, gonadal functioning is inhibited in favor of survival activities (Gomez, Manalo, & Dallman, 2004; Handa et al., 1994).

This conceptualization is supported by findings in both cross-sectional and longitudinal studies of adult human samples which show that testosterone and cortisol are typically negatively associated (Elias, 1981; Roy, Kirschbaum, & Steptoe, 2003; Zilioli & Watson, 2012).

As the HPA and HPG axes mature simultaneously during adolescence, however, "positive" (i.e., higher levels of one hormone predicting higher levels of another hormone) or even null coupling between testosterone and cortisol may be developmentally advantageous (Shirtcliff & Ruttle, 2010). Previous research in pre-pubertal male rats has found that testosterone levels are unaffected by HPA functioning and vice versa, which may be beneficial because biological resources are not being used to regulate reproductive functioning during times of stress (Gomez, Houshyar, & Dallman, 2002; Gomez, Manalo, & Dallman, 2004; Romeo, Lee, Chhua, McPherson, & McEwen, 2004). In humans, Matchock et al. (2007) assessed HPA-HPG functioning in a cross-sectional design. They found that HPA-HPG functioning was inconsistent across Tanner stages, with pre-pubescent individuals displaying a more positive or null coupling relationship, while more advanced adolescents (Tanner stage 5) had more adult-like negative patterns of cortisol-testosterone coupling.

In contrast, DHEA and cortisol have been shown to demonstrate positive correlations in adults (Hucklebridge, Hussain, Evans, & Clow, 2005). Oskis et al. (2012), however, found only trend level associations between cortisol and DHEA in their sample of 10 to 18 year old adolescents; this difference is noteworthy, as the results may have been influenced by developmental differences between their participants. Indeed, DHEA-cortisol coupling is hypothesized to become more positively associated ("tighter") as

adolescents transition into adulthood, reflecting the changing role of DHEA from more of a sex hormone to more of a stress hormone (Hucklebridge, Hussain, Evans, & Clow, 2005; Ruttle et al., in press; Tung, Lee, Tsai, & Hsiao, 2004). Similarly, while the relationship between DHEA and cortisol remains positive across the lifespan, looser coupling during adolescence may lead to greater flexibility in reaction to puberty and stressful events.

To our knowledge, only one previous study has explored the impact of early life stress on neuroendocrine coupling in an adolescent sample. Ruttle et al. (in press) assessed early life stress in children during their first year of life (ages 1, 4, and 12 months) as well as at 3.5 and 4.5 years old; early life stress was defined as exposure to parental depression, family expressed anger, parenting stress, maternal role overload, and financial stress. Saliva samples for hormone assays were collected when the children were approximately 11, 13, and 15 years old, and entered into a multi-level model to both assess hormone coupling patterns in adolescence as well as to determine if early life stress predicted alterations in the typical developmental pattern of neuroendocrine coupling. Overall, Ruttle et al. reported positive cortisol-DHEA coupling beginning at the earliest time point (age 11), and strengthening over time; cortisol and testosterone were similarly positively coupled at age 11, but transitioned to a negative coupling pattern by age 15. Additionally, children who were exposed to early life stress displayed tight, positive cortisol-DHEA coupling at age 11 that then became stronger at age 13, but did not differ significantly from coupling at age 15. Non-exposed children, in contrast, displayed a linear positive coupling between DHEA and cortisol that became tighter across time. A significant gender effect emerged when assessing the impact of early life

stress on cortisol-testosterone coupling; specifically, cortisol-testosterone coupling was not impacted by early life stress in boys, but girls who were exposed to this stress displayed a similar pattern of cortisol-testosterone coupling as cortisol-DHEA coupling, with more adult–like, negative patterns of coupling occurring earlier in development. Notably, at age 11 children who were exposed to high levels of life stress did not demonstrate significant differences in hormone coupling patterns compared to children exposed to low levels of life stress, suggesting that positive coupling may be the normative pattern of endocrine interaction during early adolescence regardless of life stress.

Additionally, the outcomes associated with dysregulated neuroendocrine coupling have not been addressed in either adult or adolescent samples. If early adolescent cortisol-DHEA coupling, for example, is more adult-like (tightly positively coupled) at an earlier age, but then fails to develop to its full capacity, DHEA may not be recruited to counter-balance the effect of cortisol as intended in adulthood (Goodyer, Park, Netherton, & Herbert, 2001). This early but incomplete development of coupling patterns may have behavioral consequences both in adolescence and adulthood. Similarly, earlier emergence of an adult-like negative cortisol-testosterone coupling pattern may explain the incidence of earlier puberty in children and adolescents exposed to life stress, including negative parenting (Ellis, 2004). Disruption of the typical developmental trajectory of neuroendocrine functioning may result in negative behavioral outcomes for young adolescents, including externalizing and internalizing symptoms.

The current study examined neuroendocrine coupling in a community sample of 9-year-old children, with three primary hypotheses. The patterns of cortisol-DHEA and

cortisol-testosterone coupling have not been investigated in a sample at the earliest stages of pubertal development; this extended prior research by determining whether the same pattern of findings demonstrated in Ruttle et al. (in press) is present earlier in the pubertal transition; Hypothesis 1 stated that both cortisol-DHEA and cortisol-testosterone hormone coupling would be positive, and associated with pubertal status. This hypothesis stemmed from Ruttle et al. (in press)'s findings in their youngest adolescents, as well as Matchock et al. (2007). We also examined the impact of stressful life events, as well as parenting behaviors, marital discord, and parental depression (which can have both genetic effects and be a proxy for a variety of environmental stressors; see Downey & Coyne, 1990, and Hammen and Shih, 2014), to determine if patterns of coupling were impacted by psychosocial factors. While Ruttle et al. (in press) did find an effect of early life stress on hormone coupling trajectories over time, such that children exposed to early life stress demonstrated more adult-like coupling patterns earlier in development, these effects did not significantly impact cortisol-DHEA coupling patterns prior to age 13, and had a paradoxical effect on cortisol-testosterone coupling in girls at age 11. Hypothesis 2, therefore, stated that children who experience higher levels of life stress, harsher or more permissive parenting, parental depression, or come from families with higher levels of marital discord, would demonstrate a similar magnitude of positive cortisol-DHEA coupling as those children who do not experience these stressors; cortisol and testosterone, by contrast, would be more positively coupled in those children exposed to high levels of life stress than in non-exposed children, although this effect could vary by sex. Finally, we assessed the impact of coupling patterns on concurrent emotional and behavioral problems to explore the impact of disrupted coupling on early adolescent

functioning. Previous research has shown that early pubertal development is associated with an increase in both internalizing and externalizing psychopathology (Graber, 2013; Negriff & Susman, 2011); Hypothesis 3, therefore, stated that children who demonstrate more psychopathology would have more developmentally mature patterns of hormone coupling (namely, tighter positive cortisol-DHEA and cortisol-testosterone coupling) than children with low levels of psychopathology.

Additionally, a secondary aim of the current study was to elucidate relationships between single hormone values (cortisol, DHEA, and testosterone) and the study variables of interest, including parenting styles, stress at different points in childhood, and child and parental psychopathology. As noted above, while there are many investigations of the relationship between cortisol and early life factors in the existing literature, there are only a few investigating these relationships for DHEA and testosterone. In sum, this study extended previous work by examining neuroendocrine coupling in a younger sample, investigating specific psychosocial factors that may impact this coupling (and subsequent pubertal development), and exploring problematic outcomes that may be associated with atypical patterns of coupling in order to shed light on potential mechanisms by which early puberty is related to psychopathology and high-risk behavior.

Method

Participants

Participants were 394 9-year-old children from a larger longitudinal study of temperament and risk for later psychopathology. The initial sample was recruited through commercial mailing lists of families with 3-year-old children living with at least one

biological parent within a 20-mile radius of the university. 559 children participated in the age 3 assessment (M = 3.5 years, SD = .26), and 84.6% (N=473) returned to complete assessments when they were approximately 6 years old (M = 6.08 years, SD = .41). 490 families completed the age 9 assessment; the current sample was drawn from the families who also completed saliva collection at age 9 (N=419; 85.5% of age 9 sample).

Procedure

Information about stressful life events was collected at all three waves (when children were approximately 3, 6, and 9 years old), while assessments of maternal and paternal parenting styles were collected at ages 3 and 6. The children's saliva was collected at home for hormonal analyses at age 9 only. Information about the child's internalizing and externalizing symptoms from the age 9 visit was also utilized. Parents were instructed how to conduct the at-home saliva collection during their in-person study visit, and were subsequently contacted by study staff to answer any questions and coordinate sample pick-up.

Measures

Hormone collection and assay. Children's saliva was collected via passive drool immediately upon waking, 30 minutes after waking, and 30 minutes before bed on three consecutive weekdays, resulting in nine saliva samples per participant. Cortisol was assayed from all nine samples, while DHEA and testosterone were assayed only from the samples taken 30 minutes after waking. Samples were collected in this way because peak cortisol concentration in saliva is typically around 30 minutes post-waking (Edwards, Clow, Evans, & Hucklebridge, 2001). While DHEA and testosterone do not show marked diurnal slopes as cortisol does, we were interested in comparing samples collected at the

exact same time to reduce the possibility that relationships between hormones could be influenced by variations in timing (e.g., a stressful event which happened upon waking could impact cortisol levels upon waking but not 30 minutes later, making a comparison with the DHEA/testosterone levels collected at 30 minutes post-waking inaccurate); therefore, only the samples collected 30 minutes after waking were utilized in the current study. Participants were instructed to freeze saliva samples immediately after collection until a member of the study staff retrieved the samples from the participants' homes. The samples were then stored at -20°C until they were transported on dry ice to the Biochemistry Laboratory at the University of Trier in Trier, Germany for analysis. All samples were assayed for DHEA and testosterone using commercially available enzyme immunoassays specifically designed for use with saliva using the manufacturer's recommended protocol (Salimetrics, State College, PA). Cortisol samples were assayed using a time-resolved fluorescence immunoassay with flourometric end-point detection (DELFIA). All samples were assayed in duplicate. The intra-assay coefficients of variation for DHEA and testosterone are between 2.5 and 6.7, while the inter-assay coefficients of variation are between 5.6 and 14.05. The intra-assay coefficients of variation for cortisol are between 4.0 and 6.7, and the inter-assay coefficients of variation for cortisol are between 7.1 and 9.0. We applied a natural log transformation to the data to reduce skew, and all analyses used transformed cortisol, DHEA and testosterone values. Other than for multi-level modeling analyses, hormone samples were averaged across three days, resulting in a single average cortisol, DHEA, and testosterone level for each participant; for multi-level modeling analyses, coordination of hormone values

across the three days was examined, so each day's hormone samples were investigated separately and a single average value was not utilized.

Sample exclusion. Several criteria were used to determine sample exclusion. First, samples that were not frozen in a home freezer or that melted in transit to the university were excluded, as sample accuracy may be compromised following a freeze-thaw cycle. Along with saliva samples, participants returned a diary in which they recorded information about the time samples were taken, whether or not they had eaten or drunk anything prior to taking the sample, and activities on sample days. Any DHEA or testosterone sample that was collected more than 30 minutes after the intended time was excluded; similarly, any cortisol sample that was collected more than 15 minutes before or after the intended time was excluded. As all three hormones have a diurnal pattern, including samples that were taken more than 30 (DHEA and testosterone) or 15 (cortisol) minutes after their intended time may obscure the results, as we want to compare hormone levels around the same peak time; additionally, this ensures that differences between children's hormone levels are due to developmental differences rather than timing of sample. The timing exclusion criterion for cortisol samples was more stringent due to cortisol's sharper diurnal peak compared to DHEA or testosterone (Matchock, Dorn, & Susman, 2007). Additionally, samples were excluded if the child was using an oral or inhaled corticosteroid medication, an antipsychotic, or methyphenidate – extended release (Concerta) during sample collection, as these medications have been shown to affect hormone levels in children and adolescents (Granger et al., 2012).

Following sample exclusion, children were included in the regression and correlational analyses if they had at least one valid sample of cortisol, DHEA, or

testosterone; in order to be included in the multi-level modeling analyses, children had to have at least one valid sample each of cortisol, DHEA, and testosterone, to allow for observation of correlations between hormones. 419 children completed home saliva collection at age 9; of these children, 394 had at least one useable cortisol sample, 372 had at least one useable DHEA sample, and 380 had at least one useable testosterone sample. Out of these children, 366 had sufficient hormone data to be included in the MLM analyses.

Parenting measure. The Parenting Styles and Dimensions Questionnaire (PSDQ; Robinson, Mandleco, Frost Olsen, & Hart, 2001) is a 37-item questionnaire designed to assess parenting styles. Factor analysis of these items yields three factors: authoritative (warm/supportive, but establishes structure/limits), authoritarian (unsupportive, controlling, and punitive), and permissive (warm/supportive, but lacking structure/limits) (Robinson et al., 2001). Items, answered on a Likert scale from 1 (Never) to 5 (Always), include, "I encourage my child to talk about his/her troubles" (authoritative), "I yell or shout when my child misbehaves" (authoritarian), and "I find it difficult to discipline my child" (permissive). The PSDQ factors have been demonstrated to have good internal consistency (Cronbach's αs between = .73 and .88; Winsler, Madigan, & Aquilino, 2005). In this sample, Chronbach's αs for both maternal and paternal reports were between .69 and .87 at age 3, and between .67 and .88 at age 6.The PSDQ was administered at the age 3 and age 6 visits to both mothers and fathers. At age 3, 359 mothers and 303 fathers completed the PSDQ; 383 mothers and 312 fathers completed the measure at age 6. The test-retest stability for the PSDQ parenting factors from age 3 to 6 were between $r = .47$ and $r = .67$ (all ps $<.001$).

Stressful life events. At ages 3 and 6, information about stressful life events was collected from parents during the Preschool Age Psychiatric Assessment (PAPA; Egger & Angold, 2004) interview; information about the same stressful life events was collected from parents and children at the age 9 assessment during the Kiddie Schedule for Affective Disorders (K-SADS-PL; Kaufman et al., 1997) interview. Stressful life events include, but are not limited to, major transitions (moving to a new house, birth of a new sibling or introduction of a new child into the home, or being removed from the family home), chronic stressors (financial distress or neglect), and traumas (natural disasters, exposure to domestic violence, or physical/sexual abuse). Assessment of stressful life events was completed for 387 children at age 3, 406 children at age 6, and 419 children at age 9. Interrater reliability for lifetime stressful life events is .99.

Parental depression. The Structured Clinical Interview for DSM-IV (SCID; First, Gibbon, Spitzer, & Williams, 1996) was conducted with both biological mothers and fathers at the age 3 assessment, and a follow-up interview was conducted at the age 9 assessment. Lifetime diagnoses of Major Depressive Disorder (MDD) or Dysthymic Disorder (DD) were utilized in the current study. SCID data was available for 409 mothers and 401 fathers; interrater reliability for the depressive disorder item (including both MDD and DD) was kappa = .93 at age 3, and kappa = .91 for the age 9 follow-up interview.

Parental marital discord. The Dyadic Adjustment Scale (DAS; Spanier, 1976) is a 32-item scale designed to assess relationship adjustment and satisfaction. Questions are designed to ascertain the levels of agreement about issues including finances, recreation, and child-rearing; as well as levels of conflict and cooperation within the marriage. The

full version of the DAS was administered to both mothers and fathers at the age 3 assessment, and an abbreviated form of the DAS (Sabourin, Valois, & Lussier, 2005) was administered at the age 6 assessment. The DAS has been demonstrated to have strong internal consistency (Cronbach's α = .96; Spanier, 1976). In this sample, Chronbach's αs ranged from .83 to .86; the test-retest stability between assessments was r = .72 for mothers and r = .66 for fathers. 302 mothers and 262 fathers completed the DAS at age 3; at age 6, 347 mothers and 296 fathers completed the measure.

Children's internalizing and externalizing symptoms. The Child Behavior Checklist (CBCL/4-18; Achenbach, 1991) is a 113-item parent-report measure that assesses children's behavioral and emotional problems. It was administered to both parents at the age 9 assessment. The CBCL includes higher order internalizing and externalizing factors, and both factors have been demonstrated to have good internal consistency (Cronbach's αs = .90 and .94, respectively; Achenbach & Rescorla, 2001). In this sample, Cronbach's αs were between .86 and .88 for the CBCL internalizing and externalizing factors.

Children's lifetime psychopathology. As noted above, parents and children were interviewed about the children's lifetime psychopathology at age 9 during the Kiddie Schedule for Affective Disorders (K-SADS-PL; Kaufman et al., 1997) interview. Lifetime diagnoses of depressive disorders (including depression NOS), anxiety disorders, attention-deficit hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD), as well as dimensional symptom scores, were used in these analyses. K-SADS were completed for 419 children in the current sample. Interrater reliability for lifetime diagnoses was kappas between .67 and .85 (with the exception of ODD diagnoses, which

had a kappa of .32), while interrater reliability for lifetime dimensional symptom scores was Cronbach's αs between .93 and .99.

Pubertal development. The Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) was administered to children and their parents at the age 9 assessment. The PDS assesses pubertal development on five indices via self- and parent-report; both boys and girls are asked about growth of body hair, skin changes (especially pimples), and growth in height, while boys are also asked about voice deepening and growth of facial hair, and girls are asked about breast development and menstruation. Respondents rate each item on a scale from 1 (not yet started) to 4 (seems complete). For the purposes of this study, only maternal reports of pubertal development were used, in light of research suggesting that mothers are accurate reporters of their children's pubertal development, while such data do not exist regarding father's reports; Coleman & Coleman, 2002). A single sum score of the PDS items was used in the current analyses; the average sum score for girls was 7.64 (out of a possible 20), while for boys the average sum score was 6.54, suggesting that the majority of the current sample was in the earliest stages of visible pubertal development.

Data analysis strategy

Data were analyzed using multi-level modeling (MLM) procedures. MLM is useful for investigating multiple levels of data, or when data are "nested" within individuals. For the current study, Level 1 variables included cortisol, DHEA, and testosterone values from three different days, while Level 2 variables were those that are constant within the individual (e.g., sex, parenting behavior, etc.). This strategy enabled us to test for evidence of neuroendocrine coupling in a sample of 9-year-old children, as

well as to examine predictors and outcomes associated with different patterns of hormone coupling.

To address factors that may influence neuroendocrine coupling in early adolescence, we examined models with cortisol as the Level 1 outcome variable, and DHEA or testosterone as Level 1 predictors; cortisol-DHEA and cortisol-testosterone coupling served as the primary outcomes of interest. The Level 2 variables of interest were included in separate models. The first set of models included stressful life events from different age ranges on Level 2; a "Total life events" score comprised of the sum of all stressful life events in a given time period was entered as Level 2 variables in the stress model. Summed stress scores were computed separately for different age ranges in order to determine whether stress at various developmental points may differentially impact neuroendocrine coupling, and the three summed scores were entered into the same model.

Next, we tested a second set of models including parenting measures as Level 2 variables predicting neuroendocrine coupling. Separate models were run for each parent at each age range (e.g., age 3 maternal parenting, age 6 paternal parenting, etc.).

Third, we examined parental depression and marital dysfunction as Level 2 variables predicting neuroendocrine functioning on Level 1. Parental depression was measured on a dichotomous scale (present vs. absent), while marital dysfunction was measured on a continuous scale; both variables were entered into the model separately for each parent and each assessment period (e.g., father's DAS score at age 3, mother's lifetime depression diagnosis, etc.).

In order to ascertain the relationship between pubertal status and coupling patterns, we tested a fourth set of models including PDS score as a Level 2 variable predicting cortisol-DHEA and cortisol-testosterone coupling.

Finally, we examined possible outcomes associated with certain patterns of neuroendocrine coupling by entering CBCL Internalizing and Externalizing scores (mothers' and fathers' reports entered separately) and K-SADS lifetime diagnoses and dimensional scores into the model at Level 2. Significant findings in this analysis would suggest that variations in hormone coupling are associated with internalizing and externalizing behaviors and/or specific disorders in our sample of young adolescents.

Results

Tables 1 through 5 show bivariate correlations between cortisol, DHEA and testosterone levels and the variables of interest described above, as well as means and standard deviations for each variable. Briefly, all three hormones were positively correlated (*r*s from .27-.63, all *p*s <.001), and body mass index (BMI) and PDS were significantly positively associated with DHEA and testosterone, but not cortisol. Sex was negatively correlated with testosterone, such that girls demonstrated higher testosterone levels than boys in this sample. Regarding major variables of interest, significant negative correlations emerged between testosterone and age 6 maternal authoritarian parenting and CBCL-Externalizing score, while a positive correlation emerged between testosterone and stressful life events at age 9. Multiple linear regression analysis was then used to investigate associations of parenting styles and child psychopathology with levels of each of the three age 9 hormones (cortisol, DHEA, and testosterone) while controlling for sex, race, and BMI, variables which have been shown to influence pubertal development

(Butts and Seifer, 2010; Freedman et al. 2002; Kaplowitz, 2008; Kaplowitz et al. 2001). Of note, PDS was not utilized as a covariate in these analyses due to the fact that outward physical development (which is measured by the PDS) is a result of rather than a predictor of hormone levels. Correlations between PDS and hormone levels were analyzed, however, to ascertain the level of agreement between the measures. Parenting styles for each parent at each assessment period were entered into regression analyses simultaneously (e.g., the first regression tested the three maternal parenting style factors at age 3 predicting hormone levels, the second tested paternal parenting style factors at age 3, the third tested maternal parenting style factors at age 6, etc.). A similar approach was used when assessing the impact of marital distress (using the DAS), in that regression models tested each parent at each assessment period separately (e.g., maternal DAS at age 3, paternal DAS at age 6). Regression models examining the impact of stress on single hormones included summed stress scores from each assessment period in the same model, in order to determine at which age stress had the most impact on hormone functioning (as there was likely considerable overlap of some stressors at each age range). Lifetime parent depressive disorders were entered into regressions separately by parent. Finally, in analyses of age 9 child psychopathology, CBCL-I and CBCL-E were entered into the models simultaneously, as were dimensional child psychopathology scores and lifetime diagnoses from the K-SADS. Each set of analyses was run separately for each hormone. We tested two-way interactions with sex, and included significant interactions in the final models.

Next, we utilized multi-level modeling procedures to determine whether significant hormone coupling (cortisol-DHEA and cortisol-testosterone) was present in

this sample; following this, we investigated associations between parenting styles, marital discord, life stress, child sex, child psychopathology, and coupling patterns. BMI is a potential confounding variable in these analyses due to its strong association with both cortisol-DHEA and cortisol-testosterone coupling and its known effect on pubertal development, (He & Karlberg, 2001; Kaplowitz, 2008); it was therefore included as a covariate in these analyses to control for the possibility that the effects of parenting, child sex, or child psychopathology on coupling were actually due to BMI. Again, PDS was not utilized as a covariate in these analyses (see discussion of potential covariates below), but was included as a predictor variable in analyses of hormone coupling.

Multiple regression analyses testing predictors of single hormone values and interactions with sex.

Cortisol. No main effects emerged in multiple linear regression models using parenting behavior, stress, marital discord, parental psychopathology, or child psychopathology to predict age 9 cortisol level (Tables 6 through 9); two significant interactions with sex emerged, however. First, a significant interaction between sex and age 3 maternal authoritative parenting was present, B = -.03 (SE = .01), t = -2.60, p <.05. When this interaction was decomposed, however, we found that neither simple slope was significant: for girls, B = .04 (SE = .03), t = 1.33, *ns*; for boys, B = .01 (SE = .02), t = .41, *ns*; Figure 1. Additionally, sex interacted with child depressive disorder diagnosis to predict cortisol levels, B = -2.58 (SE = .68), t = -3.77, p <.001. When this interaction was decomposed, we found that boys with a depressive disorder diagnosis had significantly lower cortisol levels than those boys without a diagnosis, B = -2.26 (SE = .60), t = -3.64,

p <.001, while there was no significant difference in cortisol levels between girls with or without a depressive disorder, B = .31 (SE = .28), t = 1.11, *ns* (Figure 2).

DHEA. In multiple linear regression models using parenting behavior, stress, marital discord, parental psychopathology, or child psychopathology to predict age 9 DHEA level, three significant main effects emerged (Tables 10 through 13). Specifically, higher BMI (B =.06 [SE = .02], t = 3.60, p <.001) was associated with higher DHEA levels and an anxiety diagnosis was associated with higher DHEA levels, B = .44 (SE = .22), t = 2.01, p < .05; this effect was qualified by a significant interaction with sex, however. Specifically, anxiety disorder diagnoses interacted with sex to predict DHEA levels, B = -.70 (SE = .32), t = -2.23, p < .05; when this interaction was decomposed, girls with an anxiety disorder diagnosis had significantly higher levels of DHEA than girls without this diagnosis (B =.44 [SE = .22], t = 2.02, p <.05), but an anxiety disorder diagnosis did not significantly impact DHEA levels in boys (B = -.27 [SE = .23], t = -1.17, *ns*; Figure 3). Sex also interacted with an ADHD diagnosis to predict DHEA levels, B = 1.13 (SE = .49), t = 2.30, p < .05. Decomposing this interaction revealed that neither simple slope was significant, but there was a trend for ADHD diagnoses in girls to predict lower DHEA levels (B = -.74 [SE = .43], t = -1.74, p = .08), while this association was not present for boys (B = .39 [SE = .24], t = 1.61, *ns*; Figure 4).

Testosterone. Multiple linear regression models using parenting behavior, stress, marital discord, parental psychopathology, or child psychopathology to predict age 9 testosterone level yielded the following results (Tables 14 through 17). BMI (B = .03 [SE = .01], t = 4.20, p <.001) was significantly positively associated with testosterone levels, while sex was significantly negatively associated with testosterone (B = -.15 [SE = .05], t

= -3.21, p <.01) . After controlling for these covariates, maternal CBCL-E was significantly negatively associated with testosterone levels, B = -.01 (SE = .01), t = -2.32, p <.05. Regarding parenting, maternal permissive parenting at age 6 was positively associated with testosterone levels, B = .06 [SE = .03], t = 2.00, p <.05. Age 6 maternal authoritarian parenting (B = -.02 [SE = .01], t = -2.90, p <.01) was also significantly negatively associated with testosterone levels, while there was a trend for age 6 paternal authoritarian parenting to be positively associated with testosterone, (B = .01 [SE = .01], t = 1.89, p = .06); the former effect, however, was qualified by a significant interaction with sex, B = .02 (SE = .01), t = 1.97, p <.05. Probing this interaction revealed that higher levels of maternal authoritarian parenting at age 6 were associated with significantly lower testosterone levels in girls (B = -.02 [SE = .01], t = -2.90, p <.01), but not boys (B = -.001 [SE = .01], t = -.05, *ns*); Figure 5. Another significant interaction emerged between anxiety disorder diagnoses and sex predicting testosterone levels, B = -.27 (SE = .12), t = -2.33, p <.05. When this interaction was decomposed, neither simple slope was significant, but a trend-level association emerged such that girls with anxiety disorder diagnoses had higher levels of testosterone than those without the disorder (B = .15 [SE = .08], t = 1.79, p = .07), while there was no difference for boys (B = -.12 [SE = .08], t = -1.55, *ns*; Figure 6). Finally, a history of maternal depression was associated with lower testosterone (B = -.20 [SE = .07], t = -2.92, p < .01), but this effect was also qualified by a significant interaction with sex (B = .24 [SE = .10], t = 2.45, p < 05). Probing this interaction revealed that girls who have mothers with history of depression have lower testosterone levels than girls whose mothers do not have this history, B = -.20 (SE = .07),

t = -2.82, p <.01, while there is no difference for boys, B = .04 (SE = .07), t = .57, *ns*; Table 7.

Multilevel models testing within-individual coupling between hormones

To determine if significant hormone coupling was present, cortisol was entered as the outcome, while either DHEA or testosterone were added into the model as a Level 1 predictor. Prior to this, however, we had to test the unconditional means models to determine whether sufficient Level 2 variability existed, thus justifying the use of Level 2 (between persons) predictors.

The unconditional means model for cortisol was as follows:

$$CORTISOL_{ti} = \beta_{00} + r_{0i} + e_{ti}$$

where β_{00} is a given individual's mean cortisol level, r_{0i} is the random effect at Level 2 (indicating sufficient individual differences in cortisol levels to justify testing for Level 2 factors that could contribute to hormone levels), and e_{ti} represents individual error. The ICC value for cortisol was .48; using a ICC threshold of .25 (Guo, 2005; Heinrich & Lynn, 2001), this indicated sufficient Level 2 variability to warrant the use of multi-level modeling techniques. We also tested the unconditional means models for DHEA and testosterone, as those hormones would be used as Level 1 independent variables:

$$DHEA\_LN_{ti} = \beta_{00} + r_{0i} + e_{ti}$$

$$TESTO\_LN_{ti} = \beta_{00} + r_{0i} + e_{ti}$$

The ICCs for DHEA and testosterone were .77 and .68, respectively, also meeting the minimum threshold criteria.

We then added group mean centered DHEA and testosterone levels to the models in order to determine if significant hormone coupling was present:

$$CORTISOL_{ti} = \beta_{0i} + \beta_{1i}*(DHEA\_LN_{ti}) + e_{ti}$$

$$CORTISOL_{ti} = \beta_{0i} + \beta_{1i}*(TESTO\_LN_{ti}) + e_{ti}$$

In these models, $CORTISOL_{ti}$ is the given individual's cortisol level at time 1, β_{0i} is the predicted level of cortisol at the mean level of DHEA/testosterone, β_{1i} is the coefficient describing the concurrent changes in cortisol and DHEA/testosterone across days, and e_{ti} represents individual error. These tests determined that both DHEA and testosterone were significantly positively coupled with cortisol, indicating that levels of these hormones rise and fall concurrently (DHEA: $\beta_{10} = .23$, $SE = .06$, $p < .001$; testosterone: $\beta_{10} = .79$, $SE = .11$, $p < .001$), although calculation of effect sizes suggests that the magnitude of the coupling is greater for cortisol-testosterone coupling ($r^2 = .54$) than for cortisol-DHEA coupling ($r^2 = .37$).

Multilevel models testing predictors of coupling between hormones

Next, we examined Level 2 variables that could be associated with altered hormone coupling in young adolescents. We entered grand mean centered Level 2 variables on both the cortisol intercept and DHEA/testosterone slope to assess whether these factors significantly influence hormone coupling; an example of these Level 2 models is shown below:

Level 1 model: $CORTISOL_{ij} = \beta_{0j} + \beta_{1j}*(DHEA\_LN_{ij}) + r_{ij}$

Level 2 model:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}*(CHILD\_BMI_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}*(CHILD\_BMI_j) + u_{1j}$$

where γ_{00} is the intercept term predicting level of cortisol from the individual's DHEA level when the between-person predictor (BMI) is at the sample mean level, γ_{00} is the

coupling parameter at the sample mean BMI, and γ_{11} is the coefficient describing the extent to which BMI influences cortisol-DHEA coupling.

Potential covariates. Unlike in our assessment of single hormone outcomes, there is not strong empirical evidence that certain demographic variables may predict hormone coupling alterations. We therefore decided upon covariates by first testing to see if the variables were significantly associated with either cortisol-DHEA or cortisol-testosterone coupling. The covariates of interest were sex, race, and body mass index; using the model described above, these variables were individually modeled on both the cortisol intercept and slope as Level 2 variables into models testing hormone coupling. BMI was the only potential covariate that was significantly related to hormone coupling – BMI was significantly associated with decreased cortisol levels, γ_{01} = -.02 (SE = .01), p <.05, as well as tighter cortisol-DHEA (γ_{11} = .04 [*SE* = .02], p <.001) and cortisol-testosterone (γ_{11} = .05 [*SE* = .02], p <.05) coupling patterns; it was therefore included as a covariate in all subsequent analyses; see Table 20. BMI was selected as the most appropriate covariate as other possible confounding variables such as sex and race were not significantly associated with hormone coupling patterns. As BMI is significantly correlated with PDS levels (see Table 1), however, PDS was included as a secondary covariate in models testing the impact of BMI on coupling to determine if the significant effect of BMI on coupling was driven by pubertal development; including PDS scores on both the cortisol intercept and the slope did not significantly alter the previously-reported results; sex and race, therefore, are not included in the multi-level models reported here (Tables 18 through 20).

Pubertal Development Scale (PDS). Following determination of covariates, to ascertain the impact of pubertal development on hormone coupling patterns we next entered maternal report of child's PDS score as a level 2 predictor variable. In all analyses, cortisol was used as the outcome variable, DHEA or testosterone was entered as a Level 1 predictor, and individual predictor variables (e.g., PDS, parenting, etc.) were entered as Level 2 variables, modeled on both the cortisol intercept and the DHEA/testosterone slope. By adding these variables on Level 2, we were able to measure between-person differences in coupling that may be associated with the variables of interest. In models using PDS as a Level 2 predictor, no significant effects emerged for either cortisol-DHEA or cortisol-testosterone coupling (Table 21).

Parenting. We next examined parenting as a predictor of coupling patterns (Tables 22 through 25). In a model controlling for BMI, paternal parenting at age 3 was associated with hormone coupling; specifically, both authoritative (γ_{11} = -.03 [*SE* = .01], p <.05) and authoritarian (γ_{12} = -.05 [*SE* = .01], p <.05) parenting significantly predicted looser cortisol-testosterone coupling. These paternal parenting variables were not significantly associated with the cortisol intercept. Age 6 paternal permissive parenting also predicted looser cortisol-DHEA coupling, γ_{13} = -.04 [*SE* = .02], p <.05. No significant associations were found between age 3 or 6 maternal parenting and hormone coupling.

Child stress, parental depression, and marital discord. Mixed models were run modeling child life stress, parental depression, and marital discord on both the cortisol intercept and DHEA/testosterone slopes. No significant associations emerged in these analyses (see Tables 26 through 32).

Child psychopathology. Finally, we ran mixed models with lifetime dimensional symptom scores and diagnoses modeled on both the cortisol intercept and DHEA/testosterone slopes. While there were no significant associations between dimensional symptom scores and hormone coupling (Table 33), there was a significant association between lifetime diagnosis of an anxiety disorder and cortisol-DHEA coupling; specifically, the two were negatively associated such that presence of a lifetime anxiety disorder was associated with looser cortisol-DHEA coupling, γ_{12} = -.40 [*SE* = .15], p <.01 (Table 34). Mixed models were also run modeling CBCL-Internalizing and Externalizing scores as Level 2 variables on the cortisol intercept and DHEA/testosterone slopes, but no significant effects emerged in those analyses (Tables 35 and 36).

Discussion

The current study utilized a prospective, longitudinal design to investigate the presence of hormone coupling in a young adolescent sample, while also investigating the impact of early- and middle-childhood factors in influencing hormone coupling patterns and the associations between hormone coupling and child psychopathology. The study had three primary hypotheses: 1) significant hormone coupling would be present in the sample as a whole and would also be positively associated with pubertal status; 2) children who experience higher levels of life stress, harsher or more permissive parenting, parental depression, or come from families with higher levels of marital discord, will demonstrate a similar magnitude of positive cortisol-DHEA coupling as those children who do not experience these stressors; cortisol and testosterone, by contrast, will be more positively coupled in those children exposed to high levels of life stress than in non-exposed children; and 3) children who demonstrate more psychopathology will have

more developmentally mature patterns of hormone coupling (at this age, tighter positive cortisol-DHEA and cortisol-testosterone coupling) than children with low levels of psychopathology. While we found some support for Hypothesis 1, we found minimal to no support for Hypotheses 2 and 3.

Hypothesis 1.

Children in our sample demonstrated significant positive coupling between both cortisol-DHEA and cortisol-testosterone, an effect which extends previous work in this area by demonstrating HPA and HPG axis coordination earlier in development than previously documented. Our coupling patterns are consistent with Ruttle et al. (in press), in that their participants demonstrated consistently positive cortisol-DHEA coupling which became tighter across time, and their participants' cortisol-testosterone coupling was positively coupled during the earliest phase of development (age 11) and then became increasingly negatively coupled across time. Marceau et al. (in press) similarly found that positive cortisol-DHEA and cortisol-testosterone coupling in their sample of 11-16 year olds. Interestingly, these coupling patterns were not moderated by pubertal status as hypothesized, but were moderated by BMI, in that children with higher BMI demonstrate tighter hormone coupling than those children with lower BMI; this pattern was strongest for cortisol-DHEA coupling. While the PDS directly assesses pubertal maturation, a minimum BMI is an important physical requirement for the initiation of puberty (Kaplowitz, 2008), and may reflect maturation at this stage of development with more variability than the PDS, which showed limited variability in this sample (mean = 7.06; SD = 1.73). As has been previously demonstrated (Raman et al., 2009), utilizing measures that index physical maturation in late childhood/early adolescence may not

adequately capture the extent to which pubertal maturation has begun, while investigating more sensitive measures, such as hormones, can provide more information about developmental differences among same-aged children.

Hypothesis 2.

Following previous literature in this area, we hypothesized that children exposed to stress in the form of stressful life events, parental depression and marital discord, or maladaptive parenting styles would demonstrate more adult-like hormone coupling patterns than those children not exposed to these factors. Previous work has demonstrated that stressful life events are related to both earlier adult-like hormone coupling, but also incomplete development of the coupling process; in other words, the HPA and HPG axes of children exposed to stress coordinate with one another earlier in development, but do not seem to fully coordinate as intended later in development. Exposure to stressful life events, maternal depression, or parental marital discord did not significantly impact hormone coupling in this sample, however. Although contrary to research on the impact of early-life stress on later pubertal development, our findings are consistent with Ruttle et al. (2013), who found that exposure to stressful life events predicted more adult-like coupling patterns only in their older adolescents (ages 13 and 15), and not in their 11-year-old participants. This finding, combined with the current study's findings, suggest that early life stress may have an impact on hormone coordination later in development, once the relevant systems have come online, but not earlier.

Our study did find, however, some evidence that early parenting styles may influence hormone coupling patterns, although the results were somewhat inconsistent. Specifically, paternal authoritative and authoritarian parenting at age 3 significantly

predicted looser cortisol-testosterone coupling, while age 6 paternal permissive parenting predicted looser cortisol-DHEA coupling. Both authoritative and authoritarian parenting styles are characterized by high control, suggesting that a more demanding environment early in development, with or without high levels of warmth (as featured in authoritative but not authoritarian parenting), may contribute to later maturation; this may be especially true if fathers are the more controlling parent. Permissive parenting at age 6, however, may result in a relaxed environment with limited demands; these low expectations and investment may lead children to mature more slowly than their same-aged peers. Interestingly, authoritative and authoritarian parenting styles impacted the cortisol-testosterone relationship, while permissive parenting impacted the cortisol-DHEA relationship, suggesting that parenting styles may differentially impact the HPA and HPG axes.

Hypothesis 3. To our knowledge, the current study was the first to investigate associations between childhood psychopathology and hormone coupling patterns in an early adolescent sample. In the most closely related study, Han et al. (in press) investigated the role of late childhood behavior problems on hormone reactivity during adolescent stress- and anger-inducing paradigms. They found that adolescents with higher levels of externalizing problems and lower levels of internalizing problems demonstrated positive cortisol-testosterone coupling in response to an anger induction, but did not examine baseline levels of coupling as we did in the current study. We found no evidence of coupling and child psychopathology associations, with one exception: presence of a lifetime anxiety disorder diagnosis significantly predicted looser cortisol-DHEA coupling. In our sample, the most common lifetime anxiety diagnoses are specific

phobia and separation anxiety (13.3% of the sample had one of these two diagnoses, while 22.9% of the sample had any anxiety disorder diagnosis); although there are mixed findings about the relationship between anxiety and development, the only study to examine specific phobias (e.g., fear of suffocation, fear of dogs, etc.) suggested that pre-menarcheal girls were more likely to endorse such fears than post-menarcheal girls (Stone & Barker, 1939). Additionally, Canals et al. (1992) found that both boys and girls in Tanner stage I exhibited more state anxiety than those in stages II-IV. It is possible, therefore, that the children in this study who have an anxiety diagnosis may be less developmentally advanced than the sample as a whole, and would therefore show a less mature pattern of cortisol-DHEA coupling. It is also interesting to note that the same effect did not emerge when investigating dimensional anxiety symptom scores. This suggests that presence vs. absence of an anxiety disorder is more impactful than the magnitude of the anxiety.

Single-hormone associations. In addition to investigating hormone coupling, we also assessed significant associations between each hormone (cortisol, DHEA, or testosterone) independently and the variables of interest. First, higher levels of maternal authoritative parenting were associated with lower cortisol levels, but only in girls, suggesting that a highly warm and consistent environment may have a more pronounced effect on girls' stress levels than boys'. This is additionally supported by research showing that girls are more sensitive to the effects of stress and interpersonal dysfunction than boys are, especially approaching and into adolescence (Cyranowski, Frank, Young, & Shear, 2000; Davies & Windle, 1997; Hankin, Mermelstein, & Roesch, 2007).

The finding that higher levels of age 6 maternal authoritarian parenting were significantly associated with lower testosterone levels, but again only for girls, suggests that authoritarian parenting in this sample may have a protective effect on girls, in that it may be associated with a slight delay in pubertal development. This is especially likely given the low incidence of highly negative authoritarian parenting practices, including physical abuse and excessively harsh punishments, in our largely middle class community sample. Interestingly, as noted above, paternal authoritarian parenting also predicts less mature cortisol-testosterone coupling patterns, suggesting that a moderate level of control in the family environment may be advantageous during early and middle childhood. It is somewhat puzzling that maternal permissive parenting predicted higher testosterone levels, suggesting more advanced maturation, but it is important to note that the previous effects involved paternal parenting rather than maternal parenting; it is possible that while paternal permissiveness may indicate a relaxed environment with limited demands, maternal permissiveness may signal lack of oversight and investment in children, which has been previously shown to influence pubertal development (Ellis, 2004).

Child psychopathology was also significantly associated with hormone levels in the current sample. Specifically, boys with history of a depressive disorder had significantly lower cortisol levels than boys without the disorder, girls with anxiety disorders had higher DHEA and testosterone levels than other girls, and girls with ADHD had significantly lower DHEA levels than girls without ADHD. Because the number of boys diagnosed with depressive disorders in this sample was quite low (N=2) and because findings regarding psychopathology in girls were trend-level associations, these

results should be interpreted with caution. However, the findings that girls with anxiety have higher DHEA and testosterone levels are consistent with previous research suggesting that earlier pubertal development is associated with increased anxiety and depression in adolescent females (Graber, 2003; Reardon, Leen-Feldner, & Hayward, 2009).

We also found that higher CBCL-E scores were significantly associated with lower testosterone levels. At age 9, the majority of externalizing symptoms reported by both parents and children in this sample were ADHD symptoms, and fewer ODD or conduct disorder (CD)-related symptoms emerged in this age range (as indicated by ADHD and ODD symptoms scores at age 9; the mean number of current ADHD symptoms for children in this sample was 4.40, versus 1.00 ODD symptoms); this finding is further supported by the above results regarding girls with ADHD. While previous research has shown that earlier pubertal development is associated with higher levels of externalizing behaviors (Lynne et al., 2007; see Negriff & Susman, 2011, for a review), this research has focused almost exclusively on older adolescents, rather than those in the earlier stages of development. Our finding suggests that less developmentally mature children in this sample demonstrate higher levels of externalizing psychopathology, likely in the form of ADHD symptomatology, compared to their same age peers.

The current study had a number of important strengths, especially its examination of coupling and influences on and correlates of coupling in an early adolescent sample. Other strengths include the use of a large, community sample of children who have been followed prospectively for 6 years and assessment of multiple indices of environmental stressors via longitudinal assessments of child psychopathology, stress, parent

psychopathology, marital discord, and parenting styles, in many cases provided by multiple informants. There were also, however, notable weaknesses in the current study. First, due to the scope and breadth of questions being examined, multiple analyses were conducted, which may have resulted in type 1 error. Therefore, the current analyses should be interpreted with caution and confirmed via replication. Second, stressful life events were a primary variable of interest in this study, but there was limited variability in the number and kind of stressors that the children experienced, largely due to the use of a middle class, community sample that was generally not exposed to extreme stressors or traumas (see Table 5). This may have limited our ability to detect any effects that stress may have had on hormone coupling in this sample. Third, because parenting styles and marital discord were assessed with self-report, rather than observational measures, it is possible that parents minimized their negative behaviors and exaggerated positive behaviors, resulting in skewed assessments of their parenting and marital discord. Finally, the current sample was primarily Caucasian and from a relatively affluent part of the country, which may limit the generalizability of the findings.

In sum, the current study contributes to the expanding literature on the coordination of the HPA and HPG axes during pubertal development, and raises important questions to be addressed in further research. Future work should follow adolescents longitudinally to understand how coupling patterns change throughout development, and which individual factors may influence these patterns. Additionally, investigating the estradiol, a primarily female sex hormone, would add another dimension to our understanding of the functioning of hormones during adolescence. Investigating hormone patterns and symptoms of psychopathology over time may further our

understanding of the biological processes surrounding adolescent development and their relation to the emergence of psychiatric illness.

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Table 1
Bivariate correlations among hormone levels and covariates

	Cortisol	DHEA	Testosterone	Sex	PDS	Race	BMI
Cortisol	--	.27***	.41***	.04	-.05	.05	-.06
DHEA		--	.63***	-.07	.19***	.06	.18***
Testosterone			--	-.16**	.23***	.06	.21***
Sex				--	-.32***	.06	-.03
PDS					--	.07	.28***
Race						--	-.05
BMI							--
Mean (SD)/ N (%)	2.11 (.61)	4.09 (1.19)	3.26 (.46)	195 (female) (46.5)	7.06 (1.73)	377 (white) (90)	18.26 (3.53)

*p<.05, ** p<.01, *** p<.001

Table 2
Bivariate correlations among hormone levels and parenting style scores

	Cortisol	DHEA	Testosterone	Mean (SD)
Age 3 Maternal Parenting				
Authoritative	-.04	.08	.09	60.92 (6.73)
Authoritarian	.01	.03	-.03	20.08 (4.42)
Permissive	-.06	-.01	-.01	10.59 (3.09)
Age 3 Paternal Parenting				
Authoritative	-.02	-.06	-.03	56.20 (8.20)
Authoritarian	.06	-.05	.03	20.86 (4.75)
Permissive	.04	.08	.06	11.28 (3.25)
Age 6 Maternal Parenting				
Authoritative	.004	.02	.10	61.03 (6.71)
Authoritarian	.02	-.05	-.12*	19.83 (4.10)
Permissive	-.06	.04	.04	10.12 (3.00)
Age 6 Paternal Parenting				
Authoritative	.03	.01	.01	57.10 (8.46)
Authoritarian	-.01	.07	.10	20.52 (4.65)
Permissive	.01	-.01	-.01	10.63 (3.01)

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 3

Bivariate correlations among hormone levels and parental psychopathology and marital distress

	Cortisol	DHEA	Testosterone	Mean (SD)/ N (%)
Parental lifetime depression				
Mother	-.08	.04	-.07	141 (33.7)
Father	.04	-.02	-.01	76 (18.1)
Parental DAS score				
Age 3 Mother	-.06	.02	.06	15.88 (3.89)
Age 3 Father	-.01	-.03	.07	16.32 (3.46)
Age 6 Mother	-.02	-.01	.02	16.14 (3.88)
Age 6 Father	-.03	-.07	-.02	16.10 (3.67)

*p<.05, ** p<.01, *** p<.001

Table 4
Bivariate correlations among hormone levels and child stressful life events

	Cortisol	DHEA	Testosterone	Mean (SD)
Age 3 stress	.01	-.01	-.01	3.40 (2.39)
Age 6 stress	.03	.11*	.01	3.77 (2.38)
Age 9 stress	.01	.04	.11*	2.12 (1.66)

*p<.05, ** p<.01, *** p<.001

Table 5
Bivariate correlations among hormone levels and age 9 child psychopathology scores

	Cortisol	DHEA	Testosterone	Mean (SD) / N (%)
Maternal CBCL-Internalizing	-.01	-.02	-.06	4.02 (4.69)
Maternal CBCL-Externalizing	-.08	-.03	-.14**	4.45 (5.13)
Paternal CBCL-Internalizing	-.05	-.02	.04	3.70 (4.84)
Paternal CBCL-Externalizing	-.05	-.03	-.05	4.31 (5.36)
K-SADS lifetime psychopathology diagnoses				
Depression	-.03	-.03	-.03	8 (1.90)
Any Anxiety	.004	.02	.03	95 (22.70)
ADHD	.02	.03	-.05	54 (12.90)
ODD	.06	.03	-.01	13 (3.10)
K-SADS lifetime dimensional symptom scores				
Depression	.04	-.03	-.05	1.04 (3.51)
Any Anxiety	-.02	-.02	-.01	5.26 (6.85)
ADHD	.05	.05	-.04	4.86 (8.70)
ODD	-.03	-.02	-.08	1.18 (2.74)

*p<.05, ** p<.01, *** p<.001

Table 6
Hierarchical linear regression models using life stress to predict age 9 cortisol levels

	β	B	SE
Life Stress			
Sex	.05	.07	.07
Race (white/non-white)	-.01	-.03	.15
BMI	-.09	-.02	.01
Age 3 stress	.003	.001	.02
Age 6 stress	.02	.01	.02
Age 9 stress	.04	.02	.02
Age 3 stress x Sex	-.01	-.002	.03
Age 6 stress x Sex	-.02	-.004	.04
Age 9 stress x Sex	-.03	-.008	.05

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 7
Hierarchical linear regression models using parenting styles to predict age 9 cortisol levels

	β	B	SE
Age 3 Maternal Parenting			
Sex	.03	.04	.07
Race (white/non-white)	.03	.09	.17
BMI	-.01	-.002	.01
Authoritative parenting	.45*	.04	.02
Authoritarian parenting	-.11	-.02	.03
Permissive parenting	.14	.03	.04
Authoritative parenting x Sex	-.52*	-.03	.01
Authoritarian parenting x Sex	.17	.02	.02
Permissive parenting x Sex	-.29	-.03	.03
Age 3 Paternal Parenting			
Sex	.04	.05	.08
Race (white/non-white)	-.04	-.12	.18
BMI	-.09	-.02	.01
Authoritative parenting	-.01	-.001	.01
Authoritarian parenting	.03	.004	.01
Permissive parenting	.07	.01	.01
Authoritative parenting x Sex	-.16	-.01	.01
Authoritarian parenting x Sex	.06	.01	.02
Permissive parenting x Sex	-.25	-.03	.02
Age 6 Maternal Parenting			
Sex	.01	.02	.07
Race (white/non-white)	.03	.05	.11
BMI	-.01	-.001	.01
Authoritative parenting	.01	.000	.01
Authoritarian parenting	.06	.01	.01
Permissive parenting	-.08	-.02	.01
Authoritative parenting x Sex	-.12	-.01	.01
Authoritarian parenting x Sex	.19	.02	.02
Permissive parenting x Sex	.02	.003	.02
Age 6 Paternal Parenting			
Sex	.05	.06	.08
Race (white/non-white)	-.02	-.04	.13
BMI	-.06	-.01	.01
Authoritative parenting	.03	.002	.01
Authoritarian parenting	-.01	-.002	.01
Permissive parenting	.03	.01	.01
Authoritative parenting x Sex	-.08	-.004	.01
Authoritarian parenting x Sex	.22	.02	.02
Permissive parenting x Sex	-.21	-.03	.03

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 8

Hierarchical linear regression models using parental marital satisfaction and depressive disorders to predict age 9 cortisol levels

	β	B	SE
Age 3 Maternal DAS			
Sex	-.01	-.01	.08
Race (white/non-white)	.01	.03	.21
BMI	.04	.01	.01
DAS	-.07	-.01	.01
DAS x Sex	.05	.01	.02
Age 3 Paternal DAS			
Sex	.07	.09	.08
Race (white/non-white)	-.08	-.24	.21
BMI	-.05	-.01	.01
DAS	-.03	-.01	.01
DAS x Sex	-.03	-.01	.02
Age 6 Maternal DAS			
Sex	.05	.06	.07
Race (white/non-white)	.01	.02	.13
BMI	-.02	-.003	.01
DAS	-.02	-.004	.01
DAS x Sex	-.10	-.02	.02
Age 6 Paternal DAS			
Sex	.05	.06	.08
Race (white/non-white)	-.01	-.01	.14
BMI	.01	.002	.01
DAS	-.03	-.01	.01
DAS x Sex	-.01	-.002	.02
Maternal Lifetime Depressive Disorder			
Sex	.02	.03	.06
Race (white/non-white)	.04	.07	.11
BMI	-.05	-.01	.01
Depressive disorder	-.07	-.09	.07
Depressive disorder x Sex	.23	.19	.13
Paternal Lifetime Depressive Disorder			
Sex	.03	.04	.07
Race (white/non-white)	.02	.05	.11
BMI	-.05	-.01	.01
Depressive disorder	.04	.07	.09
Depressive disorder x Sex	-.07	-.07	.17

*p<.05, ** p<.01, *** p<.001

Table 9
Hierarchical linear regression models using child psychopathology to predict age 9 cortisol levels

	β	B	SE
K-SADS lifetime dimensional symptom scores			
Sex	.03	.04	.07
Race (white/non-white)	.04	.08	.10
BMI	-.05	-.01	.01
Depression	.06	.01	.01
Anxiety	-.04	-.003	.01
ADHD	.06	.004	.004
ODD	-.05	-.01	.01
Depression x Sex	-.28	-.04	.03
Anxiety x Sex	.11	.01	.01
ADHD x Sex	-.06	-.003	.03
ODD x Sex	.28	.04	.03
K-SADS lifetime diagnoses			
Sex	.03	.04	.08
Race (white/non-white)	.03	.06	.10
BMI	-.06	-.01	.01
Depression	.06	.31	.28
Anxiety	.04	.06	.11
ADHD	-.25	-.29	.23
ODD	.09	.32	.49
Depression x Sex	-.22***	-2.58	.68
Anxiety x Sex	-.02	-.03	.16
ADHD x Sex	.16	.33	.26
ODD x Sex	-.04	-.15	.53
Maternal report of CBCL-Internalizing/Externalizing score			
Sex	.04	.05	.06
Race (white/non-white)	.05	.10	.10
BMI	-.05	-.01	.01
CBCL-I	.05	.01	.01
CBCL-E	-.10	-.01	.01
CBCL-I x Sex	-.05	-.004	.02
CBCL-E x Sex	.34	.03	.02
Paternal report of CBCL-Internalizing/Externalizing score			
Sex	.04	.05	.07
Race (white/non-white)	.03	.08	.13
BMI	.01	.002	.01
CBCL-I	-.04	-.01	.01
CBCL-E	-.02	-.002	.01
CBCL-I x Sex	-.13	-.02	.02
CBCL-E x Sex	.17	.02	.02

*p<.05, ** p<.01, *** p<.001

Table 10
Hierarchical linear regression models using life stress to predict age 9 DHEA levels

	β	B	SE
Life Stress			
Sex	-.09	-.21	.13
Race (white/non-white)	.08	.41	.27
BMI	.18**	.06	.02
Age 3 stress	-.04	-.02	.03
Age 6 stress	.11	.06	.03
Age 9 stress	-.04	-.03	.04
Age 3 stress x Sex	-.17	-.05	.05
Age 6 stress x Sex	.27	.10	.06
Age 9 stress x Sex	.13	.06	.08

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 11
Hierarchical linear regression models using parenting styles to predict age 9 DHEA levels

	β	B	SE
Age 3 Maternal Parenting			
Sex	-.10	-.23	.13
Race (white/non-white)	.13*	.68	.30
BMI	.20***	.07	.02
Authoritative parenting	.08	.01	.01
Authoritarian parenting	.04	.01	.02
Permissive parenting	-.05	-.02	.02
Authoritative parenting x Sex	-.22	-.02	.02
Authoritarian parenting x Sex	.18	.03	.03
Permissive parenting x Sex	-.17	-.04	.05
Age 3 Paternal Parenting			
Sex	-.11	-.25	.13
Race (white/non-white)	.07	.38	.32
BMI	.16*	.05	.02
Authoritative parenting	-.07	-.01	.01
Authoritarian parenting	-.07	-.02	.02
Permissive parenting	.07	.02	.02
Authoritative parenting x Sex	-.16	-.01	.02
Authoritarian parenting x Sex	-.003	.000	.03
Permissive parenting x Sex	.06	.01	.04
Age 6 Maternal Parenting			
Sex	-.06	-.13	.13
Race (white/non-white)	.10	.38	.20
BMI	.17**	.06	.02
Authoritative parenting	.003	.001	.01
Authoritarian parenting	-.07	-.02	.02
Permissive parenting	.04	.02	.02
Authoritative parenting x Sex	-.01	-.001	.02
Authoritarian parenting x Sex	.25	.05	.03
Permissive parenting x Sex	-.31	-.08	.05
Age 6 Paternal Parenting			
Sex	-.07	-.17	.14
Race (white/non-white)	-.01	-.05	.25
BMI	.17**	.06	.02
Authoritative parenting	.02	.003	.01
Authoritarian parenting	.08	.02	.02
Permissive parenting	-.04	-.02	.03
Authoritative parenting x Sex	-.08	-.004	.01
Authoritarian parenting x Sex	.22	.02	.02
Permissive parenting x Sex	-.21	-.03	.03

*p<.05, ** p<.01, *** p<.001

Table 12
Hierarchical linear regression models using parental marital satisfaction and depressive disorders to predict age 9 DHEA levels

	β	B	SE
Age 3 Maternal DAS			
Sex	-.16*	-.36	.14
Race (white/non-white)	.02	.09	.38
BMI	.22***	.08	.02
DAS	.03	.01	.02
DAS x Sex	.01	.003	.04
Age 3 Paternal DAS			
Sex	-.07	-.16	.15
Race (white/non-white)	.03	.15	.37
BMI	.19**	.06	.02
DAS	-.03	-.01	.02
DAS x Sex	-.06	-.03	.04
Age 6 Maternal DAS			
Sex	-.08	-.18	.14
Race (white/non-white)	.02	.09	.24
BMI	.20***	.08	.02
DAS	.02	.01	.02
DAS x Sex	-.06	-.03	.03
Age 6 Paternal DAS			
Sex	-.07	-.17	.15
Race (white/non-white)	-.03	-.12	.27
BMI	.16*	.06	.02
DAS	-.06	-.02	.02
DAS x Sex	-.05	-.02	.04
Maternal Lifetime Depressive Disorder			
Sex	-.06	-.15	.13
Race (white/non-white)	.06	.24	.21
BMI	.17***	.06	.02
Depressive disorder	.02	.05	.13
Depressive disorder x Sex	.25	.40	.27
Paternal Lifetime Depressive Disorder			
Sex	-.07	-.18	.13
Race (white/non-white)	.05	.19	.22
BMI	.18***	.06	.02
Depressive disorder	-.03	-.10	.17
Depressive disorder x Sex	-.01	-.02	.33

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 13
Hierarchical linear regression models using child psychopathology to predict age 9 DHEA levels

	β	B	SE
K-SADS lifetime dimensional symptom scores			
Sex	-.09	-.21	.13
Race (white/non-white)	.07	.27	.20
BMI	.19***	.06	.02
Depression	-.04	-.01	.02
Anxiety	-.03	-.004	.01
ADHD	.08	.01	.01
ODD	-.05	-.02	.02
Depression x Sex	-.19	-.06	.06
Anxiety x Sex	.21	.02	.02
ADHD x Sex	-.01	-.001	.02
ODD x Sex	.13	.04	.05
K-SADS lifetime diagnoses			
Sex	-.05	-.13	.15
Race (white/non-white)	.07	.28	.20
BMI	.19***	.06	.02
Depression	-.05	-.47	.50
Anxiety	.15*	.44	.22
ADHD	-.20	-.74	.43
ODD	.05	.39	.93
Depression x Sex	-.01	-.19	1.30
Anxiety x Sex	-.17*	-.70	.32
ADHD x Sex	.26*	1.13	.49
ODD x Sex	-.03	-.24	1.03
Maternal report of CBCL-Internalizing/Externalizing score			
Sex	-.07	-.16	.12
Race (white/non-white)	.08	.30	.20
BMI	.19***	.02	.06
CBCL-I	-.03	-.01	.02
CBCL-E	-.02	-.01	.02
CBCL-I x Sex	.20	.03	.03
CBCL-E x Sex	.10	.02	.03
Paternal report of CBCL-Internalizing/Externalizing score			
Sex	-.09	-.21	.13
Race (white/non-white)	-.03	-.13	.26
BMI	.21***	.08	.02
CBCL-I	-.03	-.01	.02
CBCL-E	-.01	-.004	.02
CBCL-I x Sex	-.13	-.01	.04
CBCL-E x Sex	.06	.02	.04

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 14
Hierarchical linear regression models using life stress to predict age 9 testosterone levels

	β	B	SE
Life Stress			
Sex	-.16**	-.15	.05
Race (white/non-white)	.03	.06	.11
BMI	.20***	.03	.01
Age 3 stress	-.01	-.002	.01
Age 6 stress	.02	.003	.01
Age 9 stress	.06	.02	.02
Age 3 stress x Sex	.15	.02	.02
Age 6 stress x Sex	-.07	-.01	.02
Age 9 stress x Sex	-.24	-.05	.03

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 15
Hierarchical linear regression models using parenting styles to predict age 9 testosterone levels

	β	B	SE
Age 3 Maternal Parenting			
Sex	-.17**	-.16	.05
Race (white/non-white)	.04	.09	.12
BMI	.20***	.03	.01
Authoritative parenting	.07	.01	.004
Authoritarian parenting	-.02	-.002	.01
Permissive parenting	-.02	-.003	.01
Authoritative parenting x Sex	-.35	-.02	.01
Authoritarian parenting x Sex	.24	.02	.01
Permissive parenting x Sex	-.26	-.03	.02
Age 3 Paternal Parenting			
Sex	-.20**	-.18	.06
Race (white/non-white)	-.01	-.02	.13
BMI	.17**	.02	.01
Authoritative parenting	-.02	-.001	.003
Authoritarian parenting	.02	.002	.01
Permissive parenting	.03	.01	.01
Authoritative parenting x Sex	-.10	-.003	.01
Authoritarian parenting x Sex	.04	.003	.01
Permissive parenting x Sex	.13	.01	.02
Age 6 Maternal Parenting			
Sex	-.17**	-.15	.05
Race (white/non-white)	.11*	.16	.07
BMI	.21***	.03	.01
Authoritative parenting	.30	.02	.01
Authoritarian parenting	-.50**	-.06	.02
Permissive parenting	.35*	.06	.03
Authoritative parenting x Sex	-.23	-.01	.01
Authoritarian parenting x Sex	.40*	.03	.01
Permissive parenting x Sex	-.30	-.03	.02
Age 6 Paternal Parenting			
Sex	-.16**	-.15	.05
Race (white/non-white)	.04	.06	.09
BMI	.19**	.03	.01
Authoritative parenting	.03	.002	.004
Authoritarian parenting	.12	.01	.01
Permissive parenting	-.04	-.01	.01
Authoritative parenting x Sex	-.06	-.002	.01
Authoritarian parenting x Sex	-.21	-.01	.01
Permissive parenting x Sex	.22	.02	.02

*p<.05, ** p<.01, *** p<.001

Table 16

Hierarchical linear regression models using parental marital satisfaction and depressive disorders to predict age 9 testosterone levels

	β	B	SE
Age 3 Maternal DAS			
Sex	-.17**	-.16	.05
Race (white/non-white)	.04	.09	.12
BMI	.20***	.03	.01
DAS	.05	.01	.01
DAS x Sex	-.04	-.01	.01
Age 3 Paternal DAS			
Sex	-.17*	-.15	.06
Race (white/non-white)	-.02	-.05	.15
BMI	.18**	.03	.01
DAS	.06	.01	.01
DAS x Sex	.06	.01	.02
Age 6 Maternal DAS			
Sex	-.17**	-.16	.05
Race (white/non-white)	.05	.07	.09
BMI	.23***	.03	.01
DAS	.04	.004	.01
DAS x Sex	-.05	-.01	.01
Age 6 Paternal DAS			
Sex	-.14*	-.13	.06
Race (white/non-white)	.02	.03	.10
BMI	.21**	.03	.01
DAS	-.01	-.001	.01
DAS x Sex	-.09	-.02	.02
Maternal Lifetime Depressive Disorder			
Sex	-.52**	-.48	.14
Race (white/non-white)	.08	.12	.08
BMI	.21***	.03	.01
Depressive disorder	-.21**	-.20	.07
Depressive disorder x Sex	.38*	.24	.10
Paternal Lifetime Depressive Disorder			
Sex	-.17**	-.16	.05
Race (white/non-white)	.07	.10	.08
BMI	.21***	.03	.01
Depressive disorder	-.02	-.02	.06
Depressive disorder x Sex	.10	.07	.12

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 17
Hierarchical linear regression models using child psychopathology to predict age 9 testosterone levels

	β	B	SE
K-SADS lifetime dimensional symptom scores			
Sex	-.16**	-.15	.05
Race (white/non-white)	.09	.13	.07
BMI	.22***	.03	.01
Depression	-.08	-.01	.01
Anxiety	.02	.001	.003
ADHD	.01	.000	.003
ODD	-.08	-.01	.01
Depression x Sex	-.20	-.02	.02
Anxiety x Sex	-.19	-.01	.01
ADHD x Sex	.01	.000	.01
ODD x Sex	.26	.03	.02
K-SADS lifetime diagnoses			
Sex	-.12*	-.11	.05
Race (white/non-white)	.08	.11	.08
BMI	.21***	.03	.01
Depression	-.06	-.22	.19
Anxiety	.14	.15	.08
ADHD	-.15	-.22	.16
ODD	-.03	-.09	.35
Depression x Sex	-.01	-.08	.49
Anxiety x Sex	-.17*	-.27	.12
ADHD x Sex	.15	.24	.18
ODD x Sex	.04	.12	.39
Maternal report of CBCL-Internalizing/Externalizing score			
Sex	-.15**	-.14	.05
Race (white/non-white)	.08	.12	.07
BMI	.21***	.03	.01
CBCL-I	.000	.000	.01
CBCL-E	-.14*	-.01	.01
CBCL-I x Sex	-.10	-.01	.01
CBCL-E x Sex	.26	.01	.01
Paternal report of CBCL-Internalizing/Externalizing score			
Sex	-.17**	-.16	.05
Race (white/non-white)	.02	.03	.10
BMI	.22***	.03	.01
CBCL-I	.08	.01	.01
CBCL-E	-.10	-.01	.01
CBCL-I x Sex	-.15	-.03	.02
CBCL-E x Sex	.16	.02	.01

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 18
Multilevel models investigating race as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.06***	.03
Coupling parameter, γ_{10}	.25***	.06	.78***	.12
Effect of moderators on cortisol level				
Race, γ_{01}	.05	.09	.06	.09
Effect of moderators on coupling				
Race, γ_{11}	-.19	.15	.06	.32
Random effects				
Cortisol level variance, u_0	.28***	.53	.30***	.55
Coupling level variance, u_1	.30***	.55	1.09***	1.04
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 19
Multilevel models investigating sex as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.09***	.03
Coupling parameter, γ_{10}	.16***	.03	.62***	.07
Effect of moderators on cortisol level				
Sex, γ_{01}	-.01	.06	.03	.06
Effect of moderators on coupling				
Sex, γ_{11}	-.04	.05	.04	.14
Random effects				
Cortisol level variance, u_0	.20***	.45	.17***	.41
Coupling level variance, u_1	.06***	.24	.47***	.69
Residual, r	.23	.48	.17	.41

*p<.05, ** p<.01, *** p<.001

Table 20
Multilevel models investigating BMI as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.23***	.05	.78***	.11
Effect of moderators on cortisol level				
BMI, γ_{01}	-.02	.01	-.02*	.01
Effect of moderators on coupling				
BMI, γ_{11}	.05***	.01	.06*	.03
Random effects				
Cortisol level variance, u_0	.27***	.52	.30***	.54
Coupling level variance, u_1	.29***	.54	1.05***	1.02
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 21
Multilevel models investigating PDS as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.23***	.06	.81***	.12
Effect of moderators on cortisol level				
PDS, γ_{01}	-.02	.03	-.02*	.03
BMI, γ_{02}	-.01	.01	-.01	.01
Effect of moderators on coupling				
PDS, γ_{11}	.05	.04	-.05	.10
BMI, γ_{12}	.03	.02	.06	.03
Random effects				
Cortisol level variance, u_0	.27***	.51	.29***	.53
Coupling level variance, u_1	.23***	.48	1.09***	1.05
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 22

Multilevel models investigating age 3 maternal parenting as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.04
Coupling parameter, γ_{10}	.21***	.06	.74***	.12
Effect of moderators on cortisol level				
Age 3 maternal authoritative parenting, γ_{01}	-.004	.005	-.004	.005
Age 3 maternal authoritarian parenting, γ_{02}	.01	.01	.01	.01
Age 3 maternal permissive parenting, γ_{03}	-.03*	.01	-.03*	.01
BMI, γ_{04}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Age 3 maternal authoritative parenting, γ_{11}	-.03	.01	-.01	.02
Age 3 maternal authoritarian parenting, γ_{12}	.01	.01	-.01	.02
Age 3 maternal permissive parenting, γ_{13}	-.02	.02	-.04	.05
BMI, γ_{14}	.04*	.02	.06	.03
Random effects				
Cortisol level variance, u_0	.29***	.53	.31***	.56
Coupling level variance, u_1	.29***	.54	1.13***	1.06
Residual, r	.18	.42	.13	.37

*p<.05, ** p<.01, *** p<.001

Table 23

Multilevel models investigating age 3 paternal parenting as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.09***	.04	2.09***	.04
Coupling parameter, γ_{10}	.24***	.06	.82***	.11
Effect of moderators on cortisol level				
Age 3 paternal authoritative parenting, γ_{01}	.0004	.004	-.0003	.003
Age 3 paternal authoritarian parenting, γ_{02}	.01	.01	.01	.01
Age 3 paternal permissive parenting, γ_{03}	.004	.02	.003	.02
BMI, γ_{04}	-.03*	.01	-.03*	.01
Effect of moderators on coupling				
Age 3 paternal authoritative parenting, γ_{11}	-.003	.01	-.03*	.01
Age 3 paternal authoritarian parenting, γ_{12}	.003	.01	-.05*	.02
Age 3 paternal permissive parenting, γ_{13}	-.02	.03	.01	.05
BMI, γ_{14}	.04**	.01	.07*	.03
Random effects				
Cortisol level variance, u_0	.30***	.55	.29***	.54
Coupling level variance, u_1	.26***	.51	.47***	.68
Residual, r	.13	.36	.14	.37

*p<.05, ** p<.01, *** p<.001

Table 24

Multilevel models investigating age 6 maternal parenting as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.08***	.03	2.08***	.03
Coupling parameter, γ_{10}	.21***	.05	.75***	.12
Effect of moderators on cortisol level				
Age 6 maternal authoritative parenting, γ_{01}	-.002	.01	-.001	.004
Age 6 maternal authoritarian parenting, γ_{02}	.01	.01	.01	.01
Age 6 maternal permissive parenting, γ_{03}	-.03	.02	-.03	.02
BMI, γ_{04}	-.01	.01	-.01	.01
Effect of moderators on coupling				
Age 6 maternal authoritative parenting, γ_{11}	-.01	.01	-.01	.01
Age 6 maternal authoritarian parenting, γ_{12}	-.01	.01	-.03	.03
Age 6 maternal permissive parenting, γ_{13}	-.02	.02	-.01	.05
BMI, γ_{14}	.04**	.01	.05*	.02
Random effects				
Cortisol level variance, u_0	.27***	.52	.29***	.54
Coupling level variance, u_1	.24***	.48	1.12***	1.06
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 25
Multilevel models investigating age 6 paternal parenting as a predictor of hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.04	2.07***	.04
Coupling parameter, γ_{10}	.23***	.06	.79***	.13
Effect of moderators on cortisol level				
Age 6 paternal authoritative parenting, γ_{01}	.00005	.004	-.00003	.01
Age 6 paternal authoritarian parenting, γ_{02}	-.01	.01	-.01	.01
Age 6 paternal permissive parenting, γ_{03}	.01	.01	.01	.01
BMI, γ_{04}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Age 6 paternal authoritative parenting, γ_{11}	-.01	.01	-.003	.01
Age 6 paternal authoritarian parenting, γ_{12}	.002	.01	-.04	.03
Age 6 paternal permissive parenting, γ_{13}	-.04*	.02	-.01	.04
BMI, γ_{14}	.05***	.01	.08**	.03
Random effects				
Cortisol level variance, u_0	.30***	.55	.33***	.58
Coupling level variance, u_1	.29***	.54	1.34***	1.16
Residual, r	.16	.39	.12	.35

*p<.05, ** p<.01, *** p<.001

Table 26

Multilevel models investigating associations between life stress at ages 3, 6, and 9 and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.24***	.06	.80***	.12
Effect of moderators on cortisol level				
Age 3 total stress, γ_{01}	-.01	.01	-.01	.01
Age 6 total stress, γ_{02}	.003	.01	.003	.01
Age 9 total stress, γ_{03}	-.01	.03	-.01	.03
BMI, γ_{04}	-.03	.01	-.03	.01
Effect of moderators on coupling				
Age 3 total stress, γ_{11}	-.01	.02	.05	.05
Age 6 total stress, γ_{12}	.01	.03	-.09	.07
Age 9 total stress, γ_{13}	.03	.04	.01	.10
BMI, γ_{14}	.04**	.01	.06**	.02
Random effects				
Cortisol level variance, u_0	.28***	.53	.30***	.55
Coupling level variance, u_1	.25***	.50	1.04***	1.02
Residual, r	.17	.41	.14	.37

*p<.05, ** p<.01, *** p<.001

Table 27

Multilevel models investigating associations between lifetime maternal depression and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.21***	.05	.77***	.11
Effect of moderators on cortisol level				
Maternal depression, γ_{01}	-.06	.07	-.06	.07
BMI, γ_{02}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Maternal depression, γ_{11}	-.06	.11	-.02	.22
BMI, γ_{12}	.04**	.01	.05*	.02
Random effects				
Cortisol level variance, u_0	.27***	.52	.29***	.54
Coupling level variance, u_1	.21***	.46	1.06***	1.03
Residual, *r*	.16	.41	.12	.35

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 28

Multilevel models investigating associations between lifetime paternal depression and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.08***	.03	2.07***	.03
Coupling parameter, γ_{10}	.21***	.05	.78***	.11
Effect of moderators on cortisol level				
Paternal depression, γ_{01}	.01	.09	.01	.09
BMI, γ_{02}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Paternal depression, γ_{11}	-.11	.14	-.34	.31
BMI, γ_{12}	.04**	.01	.05*	.02
Random effects				
Cortisol level variance, u_0	.27***	.52	.29***	.54
Coupling level variance, u_1	.22***	.47	1.05***	1.03
Residual, r	.16	.41	.12	.34

*p<.05, ** p<.01, *** p<.001

Table 29

Multilevel models investigating associations between age 3 maternal report of marital distress and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.11***	.04	2.11***	.04
Coupling parameter, γ_{10}	.18**	.07	.75***	.14
Effect of moderators on cortisol level				
Marital distress, γ_{01}	-.01	.01	-.01	.01
BMI, γ_{02}	-.01	.01	-.01	.01
Effect of moderators on coupling				
Marital distress, γ_{11}	-.01	.02	-.01	.04
BMI, γ_{12}	.03	.02	.06	.04
Random effects				
Cortisol level variance, u_0	.28***	.52	.30***	.55
Coupling level variance, u_1	.28***	.53	1.37***	1.17
Residual, r	.17	.42	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 30
Multilevel models investigating associations between age 3 paternal report of marital distress and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.10***	.04	2.10***	.04
Coupling parameter, γ_{10}	.23***	.06	.86***	.13
Effect of moderators on cortisol level				
Marital distress, γ_{01}	-.004	.01	-.004	.01
BMI, γ_{02}	-.02	.02	-.02	.02
Effect of moderators on coupling				
Marital distress, γ_{11}	.01	.02	.03	.04
BMI, γ_{12}	.03	.02	.08**	.02
Random effects				
Cortisol level variance, u_0	.27***	.52	.27***	.52
Coupling level variance, u_1	.25***	.50	.65***	.81
Residual, r	.14	.38	.14	.38

*p<.05, ** p<.01, *** p<.001

Table 31
Multilevel models investigating associations between age 6 maternal report of marital distress and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.08***	.03	2.08***	.03
Coupling parameter, γ_{10}	.22***	.06	.83***	.12
Effect of moderators on cortisol level				
Marital distress, γ_{01}	-.001	.01	-.001	.01
BMI, γ_{02}	-.01	.01	-.01	.01
Effect of moderators on coupling				
Marital distress, γ_{11}	-.02	.02	-.03	.03
BMI, γ_{12}	.03*	.02	.06	.03
Random effects				
Cortisol level variance, u_0	.27***	.52	.29***	.54
Coupling level variance, u_1	.25***	.50	1.11***	1.05
Residual, *r*	.17	.41	.13	.36

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 32
Multilevel models investigating associations between age 6 paternal report of marital distress and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.08***	.04	2.07***	.04
Coupling parameter, γ_{10}	.23***	.06	.79***	.14
Effect of moderators on cortisol level				
Marital distress, γ_{01}	-.01	.01	-.01	.01
BMI, γ_{02}	-.01	.01	-.01	.01
Effect of moderators on coupling				
Marital distress, γ_{11}	-.01	.02	-.02	.03
BMI, γ_{12}	.05*	.02	.11**	.04
Random effects				
Cortisol level variance, u_0	.30***	.55	.32***	.57
Coupling level variance, u_1	.32***	.56	1.33***	1.15
Residual, *r*	.16	.40	.12	.34

*$p<.05$, ** $p<.01$, *** $p<.001$

Table 33

Multilevel models investigating associations between lifetime K-SADS dimensional symptom scores and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.24***	.06	.79***	.11
Effect of moderators on cortisol level				
Age 9 dimensional depression, γ_{01}	.01	.01	.01	.01
Age 9 dimensional anxiety, γ_{02}	-.002	.01	-.003	.01
Age 9 dimensional ADHD, γ_{03}	-.02	.01	.003	.004
Age 9 dimensional ODD, γ_{04}	-.02	.02	-.02	.01
BMI, γ_{05}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Age 9 dimensional depression, γ_{11}	-.01	.03	-.02	.02
Age 9 dimensional anxiety, γ_{12}	-.01	.01	-.01	.02
Age 9 dimensional ADHD, γ_{13}	-.01	.01	-.01	.01
Age 9 dimensional ODD, γ_{14}	.01	.02	-.05	.05
BMI, γ_{15}	.05***	.01	.06**	.02
Random effects				
Cortisol level variance, u_0	.28***	.52	.30***	.55
Coupling level variance, u_1	.29***	.53	1.03***	1.02
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 34

Multilevel models investigating associations between lifetime K-SADS diagnoses and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.26***	.06	.84***	.12
Effect of moderators on cortisol level				
Any depressive disorder, γ_{01}	-.23	.55	-.23	.54
Any anxiety disorder, γ_{02}	-.002	.08	-.003	.08
Any ADHD, γ_{03}	-.01	.10	-.01	.10
ODD, γ_{04}	.07	.10	.07	.10
BMI, γ_{05}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Any depressive disorder, γ_{01}	-.79	.48	-1.47	1.28
Any anxiety disorder, γ_{02}	-.40**	.15	-.19	.32
Any ADHD, γ_{03}	-.18	.13	-.42	.25
ODD, γ_{04}	-.05	.20	-.28	.54
BMI, γ_{15}	.05***	.01	.06**	.02
Random effects				
Cortisol level variance, u_0	.28***	.52	.30***	.55
Coupling level variance, u_1	.29***	.53	1.02***	1.01
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 35
Multilevel models investigating associations between age 9 maternal CBCL scores and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.22***	.05	.82***	.12
Effect of moderators on cortisol level				
Age 9 CBCL-I, γ_{01}	.01	.01	.01	.01
Age 9 CBCL-E, γ_{02}	-.03	.01	-.02	.01
BMI, γ_{03}	-.02	.01	-.02	.01
Effect of moderators on coupling				
Age 9 CBCL-I, γ_{11}	-.002	.02	.03	.04
Age 9 CBCL-E, γ_{12}	.002	.02	-.03	.05
BMI, γ_{13}	.04*	.02	.06	.03
Random effects				
Cortisol level variance, u_0	.25***	.50	.27***	.52
Coupling level variance, u_1	.23***	.48	1.10***	1.05
Residual, r	.17	.41	.13	.36

*p<.05, ** p<.01, *** p<.001

Table 36
Multilevel models investigating associations between age 9 paternal CBCL scores and hormone coupling

	DHEA		Testosterone	
Parameter	Estimate	SE	Estimate	SE
Fixed Effects				
Intercept, γ_{00}	2.07***	.03	2.07***	.03
Coupling parameter, γ_{10}	.22***	.06	.83***	.12
Effect of moderators on cortisol level				
Age 9 CBCL-I, γ_{01}	-.001	.01	-.001	.01
Age 9 CBCL-E, γ_{02}	-.01	.01	-.01	.01
BMI, γ_{03}	-.01	.01	-.01	.01
Effect of moderators on coupling				
Age 9 CBCL-I, γ_{11}	-.03	.02	.01	.04
Age 9 CBCL-E, γ_{12}	.02	.02	-.02	.03
BMI, γ_{13}	.03*	.01	.04	.03
Random effects				
Cortisol level variance, u_0	.27***	.52	.30***	.54
Coupling level variance, u_1	.27***	.52	1.14***	1.07
Residual, r	.18	.42	.13	.36

*p<.05, ** p<.01, *** p<.001

Figure 1
Interaction of age 3 maternal authoritative parenting and sex predicting cortisol levels

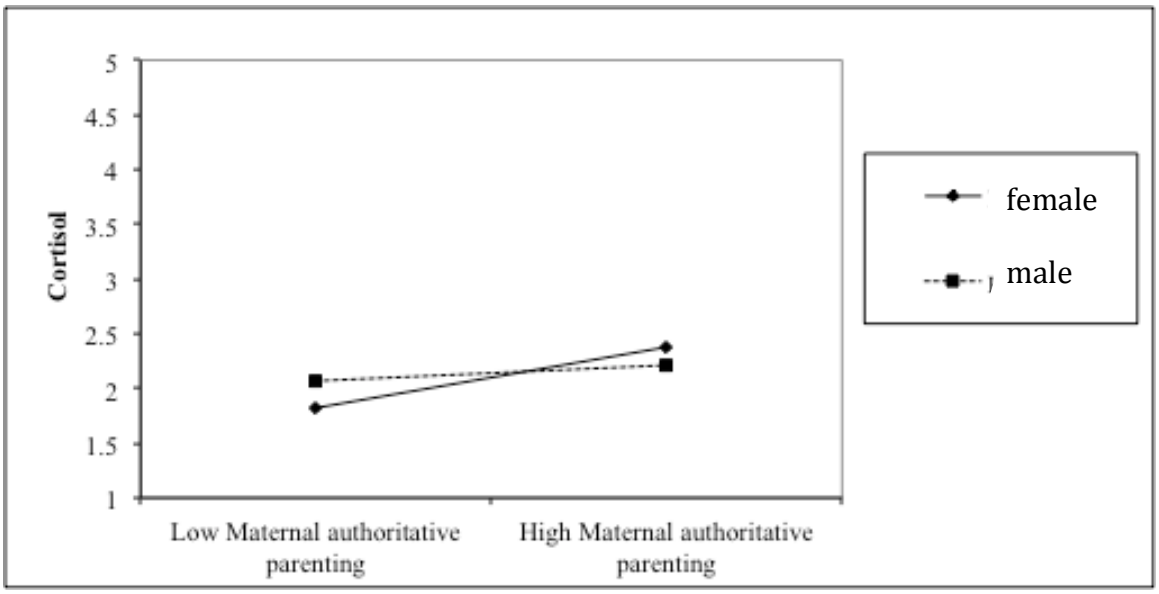

Figure 2
Interaction of depressive disorder diagnosis and sex predicting cortisol levels

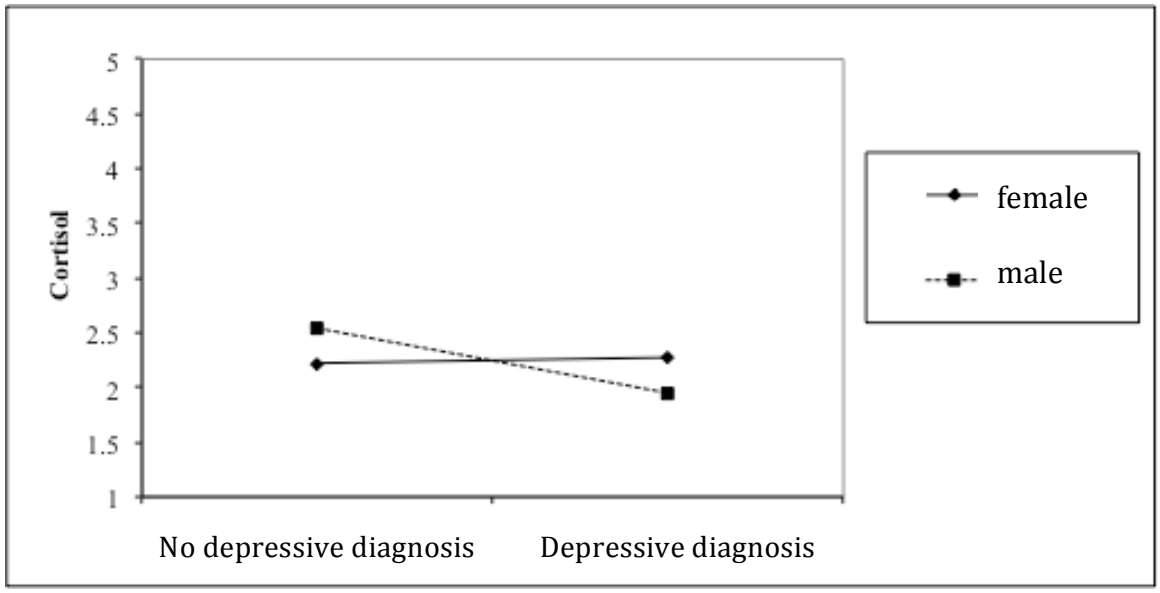

Figure 3
Interaction of anxiety disorder diagnosis and sex predicting DHEA levels

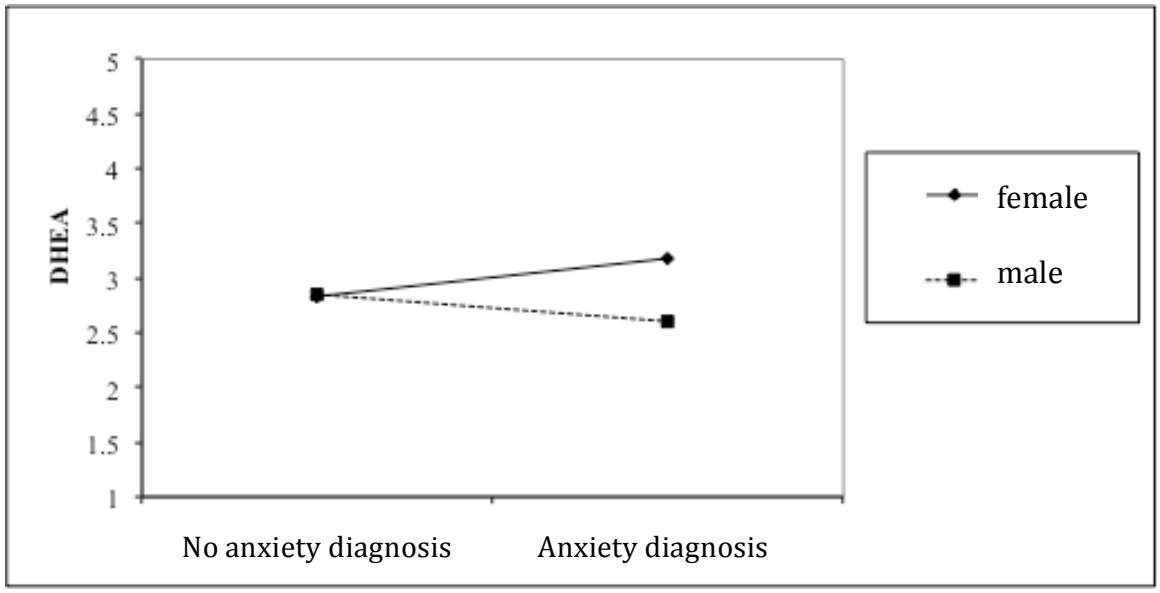

Figure 4
Interaction of ADHD diagnosis and sex predicting DHEA levels

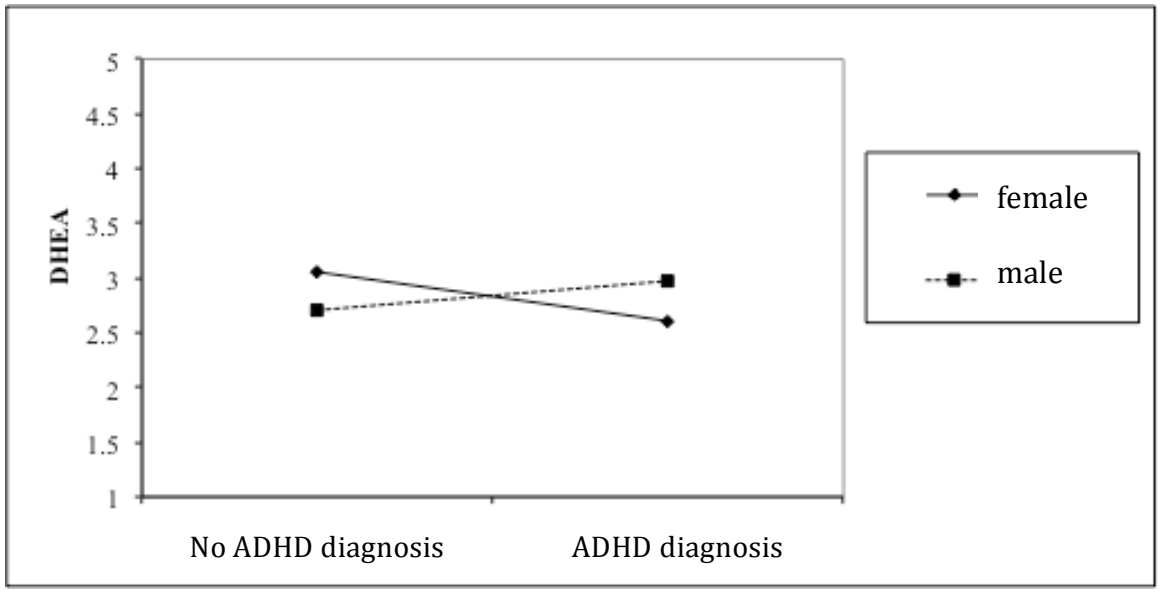

Figure 5

Interaction of age 6 maternal authoritarian parenting and sex predicting testosterone levels

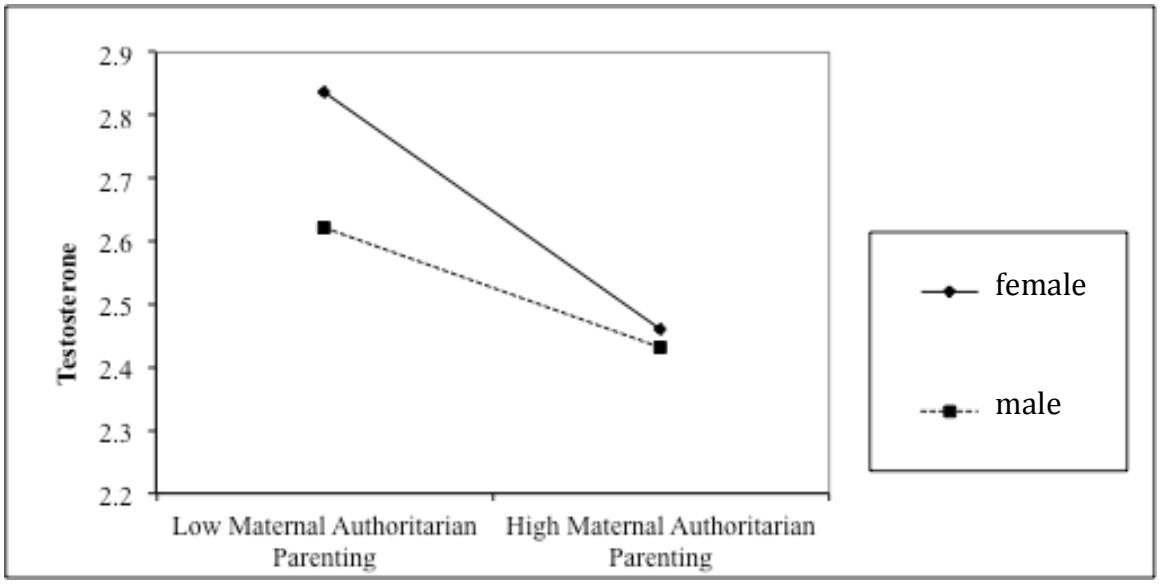

Figure 6
Interaction of anxiety diagnosis and sex predicting testosterone levels

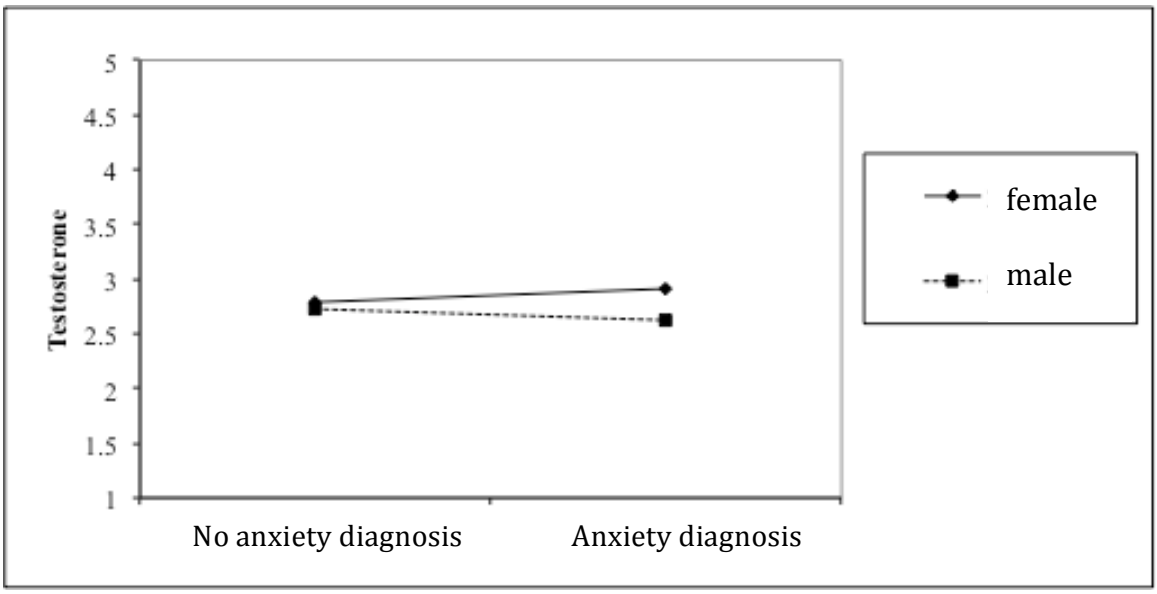

Figure 7
Interaction of maternal history of depression and sex predicting testosterone levels