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Relationships Between Anxiety, Hormones, and Pubertal Developmental Stage in Adolescent Females

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RELATIONSHIPS BETWEEN ANXIETY, HORMONES, AND PUBERTAL DEVELOPMENTAL STAGE IN ADOLESCENT FEMALES

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Introduction

Anxiety disorders are the most common form of psychopathology and often arise early in development (Beesdo, Knappe, & Pine, 2009, Beesdo, 2010). Anxiety symptoms increase during adolescence (Giedd, Keshavan, & Paus, 2008), and some work suggests that pubertal onset may be a sensitive period in which youths are specifically at risk for developing anxiety (Fuhrmann, Knoll, & Blakemore, 2015).

While age is the most widely studied metric of development, specific measures of puberty may provide unique insight into mechanisms associated with the neurodevelopment of anxiety related to puberty. Pubertal development can be measured via self-report and by pubertal hormones collected in saliva. Common self-report methods for measuring puberty include: the Tanner stages (Tanner & Whitehouse, 1976) and Pubertal Development Scale (PDS: Petersen, Crockett, Richards, & Boxer, 1988).

While most previous work focusing on the development of anxiety has primarily relied on "age" as an indicator, some work has examined more specific pubertal measures. In a previous study, Blanton et al. found relationships between Tanner pubertal stages and the size of the right hippocampus, as well as the amygdala (2012). Considering that the amygdala and hippocampus may play important roles in anxiety (Shin & Liberzon, 2009), these results are promising insofar as they link puberty and neural development. In addition, age was not a significant predictor of hippocampus or amygdala size, reinforcing the notion that neurodevelopment may be attributed to puberty, but not age (Blanton et al., 2012). The significance of a relationship between pubertal progression and anxiety symptoms has also been supported by earlier studies that have found associations between Tanner stages of puberty and

anxiety in adolescent females (Huerta & Brizuela-Gamiño, 2002). and shown self-reported social anxiety in adolescents to increase with pubertal stage (Deardorff et al., 2007).

The hormone DHEA (dehydroepiandrosterone) has been found to have a relationship with a variety of anxiety disorders, as well as its counterpart DHEAS, which is the hormone with an added sulfate component (Maninger, Wolkowitz, Reus, Epel, & Mellon, 2009). Previous studies have found that DHEA is related to bodily changes that occur during puberty and levels of the hormone are found to be highest before the onset of puberty (Korth-Schutz, Levine, New, & Chow, 1976). Researchers have found an overall increase in the DHEA concentration in the saliva of females of pubertal age while engaging in stress-inducing events, and those with increased anxiety symptoms displayed the largest increase in DHEA (Shirtcliff, Zahn-Waxler, Klimes-Dougan, & Slattery, 2007). Additionally, past studies have suggested a relationship between DHEA and social anxiety nearing the time of sexual maturation (Murray et al., 2016).

Estrogen levels have also been linked to anxiety symptoms. Decreased estrogen has been shown to correlate with greater anxiety levels in female rats (Imwalle, Gustafsson, & Rissman, 2005). Female rats which have had their ovaries surgically removed have exhibited lower levels of anxious and depressed conduct while performing exercises following exposure to estrogen at specific hippocampal receptor sites, finding that estrogen may have both properties capable of combating depressive and anxious tendencies (Walf & Frye, 2007). These observable effects were also reproduced in a study examining exercise performance among different genetically modified variants of rats, though the ERβ knockout, or βERKO, variation of rat did not experience the same anxiety-reducing effects among all exercises (Walf, Koonce, & Frye, 2008).

Low progesterone levels have been linked to anxiety in adult women (Seeman, 1997). In addition, exposure to progesterone when levels are low has been correlated with lowered anxious symptoms (1997). Results from a study on female rats suggested that allopregnanolone, a product of the hormone progesterone, related to higher anxiety levels in rats of pubertal-age (Rapkin, Morgan, Goldman, Brann, Simone, & Mahesh, 1997; Shen, Gong, Aoki, Yuan, Ruderman, Dattilo, Williams, & Smith, 2007). This correlation was not exhibited in older female rats, proposing varying biological functions of progesterone among different stages of sexual maturation (Shen, Gong, Aoki, Yuan, Ruderman, Dattilo, Williams, & Smith, 2007).

Higher testosterone levels have been linked to decreases in anxiety in females (McHenry, Carrier, Hull, & Kabbaj, 2014). A study involving a large sample of adult females demonstrated a correlation between low amounts of testosterone in samples of saliva and the prevalence of several mental health disorders, including generalized anxiety disorder (Giltay et al., 2012).

Although several studies have noted the effects of specific hormones on anxiety levels, the body of literature examining relationships between pubertal development as measured by correlations between hormones and symptoms of anxiety among adolescent females is sparse. Moreover, few studies have examined the developmental trajectories of *different facets* of anxiety. It is possible that the onset of puberty may be linked to rises in specific types of anxiety. For example, it is possible that during pubertal development, social anxiety may increase due to rapid bodily changes, which then account for "embarrassing social situations" that can diminish future successful social involvement (Blumenthal et al., 2011), while at the same time, *decreases* in separation anxiety may be linked to puberty. Despite these possibilities, no study has yet examined specific subscales of anxiety in relation to pubertal development.

The current study aimed to examine the relationships between pubertal hormones and both parent and child-reported pubertal development in relation to symptoms of panic, generalized anxiety, separation anxiety, social anxiety, and school phobia using the Screen for Child Anxiety Related Emotional Disorders (SCARED) (Birmaher et al., 1997) in a large sample of adolescent females ranging from 8 to 14 years-old ($N = 267$). To do so, we conducted partial correlations between measures of puberty (i.e., Pubertal Developmental Stage) and all anxiety subscales, while controlling for age.

Additionally, we conducted partial correlations between pubertal hormones and all anxiety subscales, while controlling for age. To examine specificity regarding anxiety symptom subscales and puberty, we then conducted two stepwise linear regressions predicting pubertal development wherein all anxiety subscales were entered as predictors.

Method

Recruitment of participants

The current study utilized a sample of 267 adolescent females aged 8 to 14. Participants from a mailing list were sent letters providing information on the study. After receiving the letters, families were called and asked questions to ensure they were qualified to participate. Eligible families had adolescent females who were fluent in English, lived with one or more biological parent, and were not diagnosed with any notable disabilities. Parents also needed to be fluent in English to participate.

Protocol

Graduate students had parents and children sign consent forms after arriving in the lab. Families completed EEG measures, adolescents had their saliva swabbed for hormone levels, and both parent and adolescent answered a series of questions on their behaviors and emotions for a separate study. Adolescents and their parents completed the SCARED and PDS (Pubertal Developmental Stage) questionnaires.

Anxiety Measures

The current study measured anxiety symptoms by utilizing the Screen for Child Anxiety Related Emotional Disorders (SCARED) (Birmaher et al., 1997). The C-SCARED, or child-SCARED, was completed by each adolescent female and the P-SCARED, or parent-SCARED was completed by their parent. The SCARED includes questions that accurately reflect anxiety subscales of panic, generalized anxiety (GAD), separation anxiety, social phobia, and school avoidance. Responses were: 0 “not true or hardly ever true”, 1 for if a statement were “sometimes true”, or 2 for if a statement were “true or often true”. The highest score that could be obtained on both the P-SCARED and C-SCARED was 76, and 38 statements were included in both versions of the questionnaire.

Hormones

Samples of saliva were taken from the adolescents using an enzyme immunoassay kit (Salimetrics, State College, PA) in order to determine their levels of estradiol, progesterone, dehydroepiandrosterone, and testosterone. All tests reported in the current study were completed using the average level of each hormone for the sample. The minimum detection limits for

estradiol, progesterone, and testosterone were 0.1 pg/mL (range from 1- 32 pg/mL), 5 pg/mL (range from 10-2430 pg/mL), and 0.1 pg/mL (range from 6.1-600 pg/mL), respectively.

The mean intra-assay coefficient of variation for estradiol was 7%, and the mean inter-assay coefficient of variation was 7.5%. The mean intra-assay coefficient of variation for progesterone was 4%, and the average inter-assay coefficient of variation was 5.5%. The mean intra-assay coefficient of variation for testosterone was 2.5%, and the average inter-assay coefficient of variation was 5.6%.

Nominal levels of cross-reactivity were detected for the estradiol samples in regard to both estriol and estrone, and no significant levels of cross-reactivity occurred regarding antibodies and progesterone, DHEA, or testosterone.) There is minimal cross-reactivity for the progesterone assay to corticosterone and no detected cross-reactivity to estradiol, testosterone, or DHEA. There is minimal cross-reactivity for the testosterone assay to progesterone or estradiol and no detected cross-reactivity to corticosterone or DHEA.

Development Measures

The PDS (Petersen, Crockett, Richards, & Boxer, 1988) measure was completed by parents and children. Responses were utilized to determine the pubertal stage of the adolescent based on reports of a variety of traits present in puberty. The five traits mentioned in the PDS were scored on a scale of 1 – 4, with one indicating “no development” in that area and 4 indicating “completed development”. Reported scores for each trait were averaged to create a total score for both the parent and child-reported PDS.

Statistical Approach

SPSS (Version 17.0) general linear model software was utilized in order to conduct the bivariate correlations and stepwise linear regressions, with Greenhouse-Geisser correction applied to *p* values with multiple degrees of freedom, repeated-measures comparisons when necessitated by violation of the assumption of sphericity. In SPSS, partial correlations were conducted to determine significant relationships between parent and child-reported SCARED subscales of anxiety and pubertal hormones, while controlling for age. Partial correlations were also completed while controlling for age in relation to z-scored and combined parent and child PDS. Two stepwise linear regressions were performed as well, one for both parent and child reported anxiety symptoms. The regressions were utilized to determine which subscale of anxiety best predicted pubertal development. An overall "puberty variable" was created for these analyses, which included PDS, DHEA, progesterone, and testosterone. The overall puberty variable was utilized as the dependent variable, while predictors for each regression were anxiety subscales.

Results

Descriptive Statistics

The average total score of the child-SCARED was 20.69, *SD*= 10.13. The average total score of the parent-SCARED was 11.16, *SD*= 8.18. The average child-reported scores for subscales of panic, generalized anxiety, separation anxiety, social anxiety, and school avoidance were 4.55, *SD*= 3.90 for panic; 5.60, *SD*= 3.96 for generalized anxiety; 3.90, *SD*= 2.92 for separation anxiety; 5.14, *SD*= 3.50 for social anxiety; and 1.51, *SD*= 1.54 for school avoidance. The average parent-reported scores for subscales of panic, generalized anxiety, separation anxiety, social anxiety, and school avoidance were 1.37, *SD*= 2.41 for panic; 3.60, *SD*= 3.50 for

generalized anxiety; 1.77, *SD*= 2.27 for separation anxiety; 3.57, *SD*= 3.41 for social anxiety; and .86, *SD*= 1.18 for school avoidance.

Overall, the average age of the child participants was 11.87, *SD*= 1.51. The average parent-reported PDS was 2.62, *SD*= .71. The average child-reported PDS was 2.63, *SD*= .71. The average level of testosterone among the participants was 48.16, *SD*= 21.27. The average progesterone level was 101.82, *SD*= 92.12. The average estrogen level was 1.82, *SD*= .85. The average DHEA level was 97.09, *SD*= 68.55.

Bivariate Correlations

Bivariate correlations are presented in Table 1. As can be seen in the table, all hormones correlated with one another. As would be expected, age correlated with PDS stage. Age was not significantly correlated with either parent- or child-reported SCARED total scores. The parent- and child-SCARED total scores correlated with one another. Additionally, both the parent- and child-SCARED total scores correlated with the PDS. Moreover, PDS and age both correlated with all hormones.

Generally, greater hormone levels related to increased anxiety symptoms reported on the SCARED. Specifically, DHEA levels correlated with both parent- and child-SCARED total scores, while higher progesterone related to the parent-SCARED overall score. Additionally, testosterone related to an increased child-SCARED score. Estrogen did not correlate to either the child- or parent-SCARED total score.

Associations controlling for age

We conducted partial correlations between the child- and parent-SCARED subscales and pubertal development (i.e., PDS), *controlling for age*. As can be seen in Tables 2a and 2b, PDS was significantly correlated with both parent- and child-reported SCARED subscales of panic and school avoidance. PDS was also related to the child-reported SCARED generalized anxiety and social anxiety subscales.

We then conducted partial correlations between the child- and parent-SCARED subscales and pubertal hormones, *controlling for age*. As can be seen in Tables 3a and 3b, DHEA related to higher levels of parent-reported panic and separation anxiety, while increased progesterone also correlated with a significant increase in panic. DHEA correlated with every child-reported SCARED anxiety subscale with the exception of school avoidance. In relation to both the parent- and child-SCARED subscales, estrogen did not significantly relate to any anxiety subscales. Though testosterone was not correlated with any parent-SCARED subscales, higher levels of testosterone related to an increase in GAD and separation anxiety as reported by the child.

Regressions

To examine specificity regarding anxiety symptom subscales and puberty, we conducted two stepwise linear regressions predicting pubertal development. We z-scored and combined the PDS score, as well as all hormones with significant associations with anxiety (i.e, DHEA, progesterone, and testosterone) to create an overall measure of "pubertal development." We then conducted a stepwise linear regression, entering all child-reported SCARED subscales, as well as age, predicting pubertal development. Results suggested that age and child-reported GAD

significantly related to pubertal development, $B = .53$, $t = 9.22$, $p < .001$ and $B = .20$, $t = 3.46$, $p < .001$, respectively. All other subscales were excluded from the model.

We then conducted a stepwise linear regression, entering all parent-reported SCARED subscales, as well as age, predicting pubertal development. Results suggested that age and parent-reported panic significantly related to pubertal development, $B = .56$, $t = 9.77$, $p < .001$, and $B = .15$, $t = 2.58$, $p < .01$, respectively. All other subscales were excluded from the model.

Discussion

The results suggest that self-reported pubertal stage and pubertal hormones are associated with both child and parent-reported anxiety symptoms, even when controlling for age. Moreover, DHEA has the strongest relationship with child and parent-reported anxiety symptoms, above the influence of age. This finding is consistent with previous research that suggests more anxiety symptoms in adolescent females correlate with higher DHEA levels (Shirtcliff, Zahn-Waxler, Klimes-Dougan, and Slattery, 2007).

Results from the stepwise linear regressions indicate that when entering all anxiety subscales into a model predicting pubertal development, it is only child-reported generalized anxiety and parent-reported panic symptoms that remain significant – indicating that these subscales of anxiety may *uniquely* relate to puberty. The findings also indicate a potential discrepancy between the general anxiety symptoms that a child experiences and reports and the identifiable symptoms that a parent is able to recognize and report on. These results support previous findings that indicate that adolescents feel their symptoms are misconstrued and incorrectly interpreted by their parental figures (Bidaut-Russell et al., 1995), especially in the

case of correctly identifying child anxiety (Edelbrock, Costello, Dulcan, Conover, & Kala,1986). Future research will need to be performed in order to determine the extent to which this discrepancy affects the dynamic between parent and child, as well as any effects this may have on the progression of the adolescent's anxiety.

Prior studies have found self-reported social anxiety symptoms in adolescents to increase with pubertal stage (Deardorff et al., 2007), as well as relationships between DHEA and social anxiety nearing the time of sexual maturation (Murray et al., 2016). Results from the current study are consistent with these findings, suggesting a robust correlation between PDS and social anxiety. Additionally, results also suggested a significant relationship between high DHEA levels and greater child-reported social anxiety symptoms.

Previous researchers have noted increased anxiety symptoms in African-American and Mexican adolescent females with a higher pubertal status (Carter, 2014; Huerta & Brizuela-Gamiño, 2002). The current study is consistent with these findings insofar as anxiety related to pubertal development. However, the current study is novel in that self-reported levels from adolescents were utilized, but also reports of PDS and SCARED anxiety symptoms from their parents were examined for existing correlations to adolescent reports. In addition, the current study is novel in utilizing pubertal hormone levels in addition to PDS to create a measure of pubertal development.

Limitations to the current study include the absence of a physician's assessment of the pubertal stage of the adolescents. Due to reliance on self-report measures to determine each adolescent's PDS, it is possible parents or adolescents have biases in reporting pubertal developmental. Though previous researchers have found that self-report on PDS by adolescent

females of pubertal age and their mothers were correlated to reports of pubertal development completed by a medical professional (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987), other studies have found that adolescents may display bias in their reporting (Irwin, Turner, & Schlossberger, 1990).

Additionally, the current study is limited due to a lack of ethnic variation in the overall sample, which was primarily Caucasian. Though some studies have been conducted in order to examine how pubertal status affects anxiety levels in adolescents from several ethnic and cultural backgrounds including African-American (Carter, 2014), Iranian (Ahmadi, Anoosheh, Vaismoradi, & Safdari, 2009), and Spanish youth (Canals, Martí-Henneberg, Fernández-Ballart, Clivillé, & Domènech, 1992), further research will need to be completed to determine any variations in the correlations found in the current study due to differing cultural experience.

Future research will also need to be completed in order to generalize these results to adolescent males of pubertal age. Previous studies have shown results similar to the current study, indicating increased anxiety levels for those males who were further in their pubertal development (Susman, Dorn, & Chrousos, 1991). Potentially differing social experiences and biological processes during puberty may also have an effect on relationships between anxiety subscales, PDS, and hormones in adolescent male populations.

	1	2	3	4	5	6	7	8
1. Age	-							
2. PDS	.73**	-						
3. DHEA	.32**	.43**	-					
4. Estrogen	.30**	.31**	.34**	-				
5. Progesterone	.30**	.37**	.54**	.48**	-			
6. Testosterone	.41**	.42**	.70**	.63**	.54**	-		
7. Child-SCARED total	.06	.22**	.20**	.09	.08	.12*	-	
8. Parent-SCARED total	.11	.16*	.16**	.04	.12*	.09	.43**	-

* $p < .05$; ** $p < .01$

Table 1: Bivariate correlations

	1	2	3	4	5	6
1. PDS	-					
2. Child-SCARED panic	.23**	-				
3. Child-SCARED GAD	.26**	.62**	-			
4. Child-SCARED separation anxiety	.08	.47**	.49**	-		
5. Child-SCARED social anxiety	.18**	.37**	.50**	.32**	-	
6. Child-SCARED school avoidance	.16*	.53**	.49**	.44**	.31**	-

* $p < .05$; ** $p < .01$

Table 2A: Correlations between PDS and child-SCARED anxiety subscales, controlling for age.

	1	2	3	4	5	6
1. PDS	-					
2. Parent-SCARED panic	.17*	-				
3. Parent-SCARED GAD	.08	.48**	-			
4. Parent-SCARED separation anxiety	.07	.50**	.51**	-		
5. Parent-SCARED social anxiety	.03	.33**	.48**	.38**	-	
6. Parent-SCARED school avoidance	.19**	.48**	.42**	.47**	.41**	-

* $p < .05$; ** $p < .01$

Table 2B: Correlations between PDS and parent-SCARED anxiety subscales, controlling for age.

	1	2	3	4	5	6	7	8	9
1. DHEA	-								
2. Estrogen	.28**	-							
3. Progesterone	.51**	.42**	-						
4. Testosterone	.62**	.64**	.48**	-					
5. Child-SCARED panic	.16*	.05	.04	.05	-				
6. Child-SCARED GAD	.22**	.07	.10	.15*	.63**	-			
7. Child-SCARED separation anxiety	.19**	.14	.02	.15*	.47**	.50**	-		
8. Child-SCARED social anxiety	.20**	.01	.08	.09	.36**	.51**	.33**	-	
9. Child-SCARED school avoidance	.05	-.08	-.05	-.01	.53**	.50**	.47**	.34**	-

* $p < .05$; ** $p < .01$

Table 3A: Correlations between hormones and child-SCARED subscales, controlling for age.

	1	2	3	4	5	6	7	8	9
1. DHEA	-								
2. Estrogen	.28**	-							
3. Progesterone	.51**	.42**	-						
4. Testosterone	.62**	.64**	.48**	-					
5. Parent-SCARED panic	.16*	-.01	.14*	.07	-				
6. Parent-SCARED GAD	.11	.04	.06	.06	.48**	-			
7. Parent-SCARED separation anxiety	.17*	.01	.07	.02	.52**	.48**	-		
8. Parent-SCARED social anxiety	.08	-.03	.11	.06	.32**	.46**	.36**	-	
9. Parent-SCARED school avoidance	.04	-.11	-.03	-.03	.48**	.43**	.46**	.41**	-

* $p < .05$; ** $p < .01$

Table 3B: Correlations between hormones and parent-SCARED subscales, controlling for age.

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