Received: 11 July 2017 | Accepted: 9 February 2018
DOI: 10.1002/dev.21625

RESEARCH ARTICLE

WILEY Developmental Psychobiology

Linking puberty and error-monitoring: Relationships between self-reported pubertal stages, pubertal hormones, and the error-related negativity in a large sample of children and adolescents

Julia Y. Gorday | Alexandria Meyer

Department of Psychology, Florida State University, Tallahassee, Florida

Correspondence
Alexandria Meyer, Department of Psychology, Florida State University, 1107 W Call St., Tallahassee 32304, FL.
Email: meyer@psy.fsu.edu

Funding information
National Institute of Mental Health, Grant number: R01 MH097767

Abstract

The error-related negativity (ERN) is a negative deflection in the event-related potential occurring when individuals make mistakes. The ERN has been proposed as a biomarker for anxiety and a substantial amount of research suggests the ERN increases across development. Further, the ERN may relate to individual differences and the development of cognitive control. Despite the large quantity of research on this topic, there have been no studies focusing on the relationship between pubertal hormones and the ERN. Previous work suggests developmental increases may begin sooner for girls than for boys, suggesting that puberty may impact the ERN. The current study examined the relationship between pubertal hormones and the ERN amplitude in a sample of 99 females between 8 and 14 years old. Each participant and the parent who accompanied them completed the Pubertal Developmental Scale (PDS) to assess the degree to which pubertal indicators are present. Participants also completed a Go/NoGo Task while EEG was recorded and participants provided saliva samples for hormone assays. Results indicated that ERN was significantly related to both the dehydroepiandrosterone (DHEA) hormone and PDS scores. A simultaneous multivariate regression suggested that DHEA levels significantly predict the ERN, even when controlling for age, behavioral variables, and PDS. These findings suggest that ERN amplitude is related to DHEA levels, further linking puberty to developmental increases in the ERN. Future research should examine this relationship in the context of developmental increases in anxiety symptoms.

KEYWORDS
adolescence, dehydroepiandrosterone, development, DHEA, EEG, ERN, error-related negativity, hormones, puberty, pubertal hormones, response monitoring

1 | INTRODUCTION

Adolescence is a unique period of development marked by dramatic alterations in hormone levels, physical appearance, behavior, and emotional functioning (Blakemore, Burnett, & Dahl, 2010; Forbes & Dahl, 2010). Moreover, adolescence is characterized by a transition away from parental dependence and by increased self-reliance (Casey, Duhoux, & Cohen, 2010), as well as maturation in physical, cognitive, social, and psychological domains (Blakemore et al., 2010). Differences in male and female developmental trajectories in these domains have led researchers to hypothesize about the role of puberty in neural development.

To understand the neurodevelopmental processes underlying the changes that occur during adolescence, one line of research has focused on the error-related negativity (ERN). The ERN is a fronto-central negative deflection in the waveform occurring when an individual makes an error of commission (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), and is thought to reflect the activation of a generic error detection system (Gehring et al., 1993; van Veen & Carter, 2002). The ERN is generated in the anterior cingulate cortex (ACC), a region of the brain where information about pain, threat, and punishment is integrated to alter subsequent behavior (Shackman et al., 2011).[1]

The ERN has been proposed as a neural biomarker of anxiety (Meyer, 2016) and has been shown to be increased in anxious individuals in over 40 studies to date (Moser, Moran, Schroder, Donnellan, & Yeung, 2013). Moreover, the ERN is increased in anxious children early in the course of development (Meyer et al., 2013). Additionally, the ERN appears to index *risk* for developing anxiety. For example, an increased ERN in young children predicts the onset of new onset anxiety disorders 3 years later, while controlling for baseline anxiety symptoms (Meyer, Hajcak, Torpey-Newman, Kujawa, & Klein, 2015).

Moreover, some work suggests the ERN may be related to individual differences in cognitive control (Coleman, Watson, & Strayer, 2017; Klawohn, Endrass, Preuss, Riesel, & Kathmann, 2016; Maier & Steinhauser, 2017; Miller, Watson, & Strayer, 2012), and some have suggested that changes in the ERN that occur during childhood may reflect the development of cognitive control (Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013). Considering the ERN indexes current anxiety and risk for anxiety, and perhaps individual variation in cognitive control, there is substantial interest in elucidating developmental trajectories of this neural marker.

The ERN has been linked to developmental changes throughout childhood and adolescence. For example, Davies, Segalowitz, and Gavin (2004) found that the magnitude of the ERN increased with age in a sample of children and adolescents ranging from 7 to 18 years-old. Since then, over 14 studies have found a similar pattern wherein the ERN increases as children age, reaching adult-like magnitude in mid to late adolescence (Tamnes et al., 2013). Consistent with these findings, an fMRI study including participants between the ages of 8 and 27 years-old found that error-related dorsal ACC activity increased across development (Velanova, Wheeler, & Luna, 2008).

In children and adolescents, studies using source-localization suggest the ERN may be generated in the dorsal ACC or posterior cingulate cortex (Buzzell et al., 2017; Ladouceur, Dahl, & Carter, 2007; Santesso & Segalowitz, 2008). In line with studies finding developmental increases in the ERN, diffusion tensor imaging (DTI) studies have found that the cingulum bundle (a white matter tract that underlies the ACC) matures at a slower rate than most other tracts (Lebel & Beaulieu, 2011; Lebel et al., 2012). A study utilizing resting state functional connectivity suggests that in children, the dorsal ACC is relatively disconnected from a cingulo opercular control network that has been identified in adult populations (Fair et al., 2007). Recent work suggests that error-related activity within the insula, inferior frontal gyrus, and orbitofrontal cortex increase with age (Buzzell et al., 2017). Together, these findings suggest that error-related neural activity undergoes normative changes as children transition from childhood to adolescence.

Additionally, Davies et al. (2004) observed a quadratic relationship between the ERN and age, wherein the ERN amplitude appeared to transiently decrease around the time of pubertal onset, and then subsequently rise until reaching adult-like levels. Notably, Davies et al. (2004) observed an interaction between this trajectory and gender, indicating that the ERN began increasing sooner in girls compared to boys (Davies et al., 2004). This finding suggests that developmental increases in the ERN may be related to pubertal onset, and not just age, as girls experience puberty sooner than boys.

Despite these findings suggesting a link between the ERN and puberty, no study has yet examined the developmental increase in the ERN in relation to self-reported stages of puberty. Furthermore, the ERN has not yet been examined in relation to other indicators of puberty, such as levels of pubertal hormones. Indeed, while evidence from animal studies suggest that pubertal hormone levels exert significant effects on neural maturation (Blakemore et al., 2010; Sisk, Schulz, & Zehr, 2003; Sisk & Zehr, 2005), and some studies have found links between puberty and neural activity related to reward processing (Op de Macks et al., 2011), no studies have yet examined the role of puberty and error-processing. Additionally, some work has suggested that specific pubertal hormones may be related to developmental increases in anxiety via their impact on neural development (Murray et al., 2016)—raising the possibility that pubertal hormones may impact the ERN, a neural marker of anxiety. Moreover, while a commonly used measure of puberty is based on self-report (Tanner & Whitehouse, 1976), it may be beneficial to utilize hormonal assays due to the fact that hormone circulation occurs upstream of external or physical manifestation of puberty (Blakemore et al., 2010). Thus, using hormone measures, in addition to self-report, may be the optimal method of examining the impact of puberty on neural development.

The onset of puberty is associated with the following endocrine events: adrenarche and gonadarche (Dorn, 2006; Spear, 2000). Adrenarche begins with the activation of the hypothalamic-pituitary-adrenal axis, and typically begins earlier than gonadarch—between the ages of 6 and 9 years-old in females (Dorn, 2006). Adrenal hormones (dehydroepiandrosterone and progesterone) begin to increase early in development and continue well into late adolescence and early adulthood (early 20s) and are associated with secondary sexual characteristics such as pubic hair and changes in sweat or body odor (Dorn, 2006). Gonadarche, begins with the activation of the hypothalamic-pituitary-gonadal axis and typically begins between the ages of 8 and 14 years-old in females. This process begins with the release of gonadotropin-releasing hormone, which stimulates luteinizing hormone and follicle-stimulating hormone, which activate changes in the gonads (ovaries or testes). The gonads then begin producing gametes and secreting testosterone and estrogen—which trigger additional changes and the acquisition of secondary sexual characteristics (Spear, 2000).

In the current study, we examine the relationship between the ERN and child and parent-report of puberty using the Pubertal Development Scale (PDS; Carskadon & Acebo, 1993) in a large sample of child and adolescent females ($N = 99$) between the ages of 8 and 14 years-old. Additionally, we examine adrenarche and gonadarche pubertal hormone levels in relation to the magnitude of the ERN. We examine the following puberty-related hormones: dehydroepiandrosterone (DHEA), testosterone, estrogen, and progesterone. Based on previous work suggesting a link between puberty and the ERN (Davies et al., 2004), we predicted that both self-reported stages of puberty and pubertal hormones would relate to developmental increases in the ERN magnitude.

2 | METHOD

2.1 | Recruitment of participants

Participants in the proposed research included 99 females between the ages of 8 and 14 who are part of a larger and longitudinal ongoing NIMH-funded R01 study examining reward and depression across adolescence. We recruited children and adolescents using a commercial mailing list of families that have an 8–14 year-old female living at home. We sent letters describing the study prior to an initial call and screened families based on the following criteria: the child must live with at least one biological parent, the child and caretaker must speak English, and the child must not have a significant developmental or medical disability.

2.2 | Protocol

During the lab visit, when families arrived in the laboratory, parents and children were consented by a graduate student. The assessment consisted of a number of behavioral and psychophysiological measures that were part of a larger study, as well as the Go/NoGo task described below. During the laboratory visit, children and parents both completed the PDS and saliva samples were collected from children during the lab visit to measure hormone levels.

2.3 | Measure of puberty

Parents and children were given the Pubertal Development Scale (PDS) to assess the degree to which several indicators of puberty (e.g., body hair, growth in height, skin changes, deepening of voice in males, breast development in females) were present in the children (Petersen, Crockett, Richards, & Boxer, 1988). The PDS has been shown to reliably index puberty noninvasively and inexpensively (Bond et al., 2006) and is highly correlated with pubertal stages based on physical exam and basal hormone levels (Shirtcliff, Dahl, & Pollak, 2009). The PDS consists of five items (on a scale from 1 to 4; 1 = *no development*, 2 = *development had barely begun*, 3 = *development was definitely under way*, 4 = *development was complete*) that are averaged together to create a summary score. Parent and child scores on the PDS were *z*-scored and summed to form a combined self-report measure of pubertal status.

2.4 | Hormones

Participants provided two saliva samples for hormone assays during the lab visit, adjacent in time, and mean values across samples were used in analyses. Participants were instructed that they could not eat or drink at least 30 min before providing a saliva sample during the lab visit. It should be noted that lab visits took place at different times of the day and evening; as well as the fact that we did not control for variation in menstrual cycle. All samples were assayed for salivary estradiol, progesterone, dehydroepiandrosterone, and testosterone using an enzyme immunoassay kit (Salimetrics, State College, PA). For estradiol, the test has a minimum detection limit of 0.1 pg/mL (range from 1 to 32 pg/mL), and average intra- and inter-assay coefficients of variation were 7% and 7.5% respectively. For progesterone the test has a minimum detection limit of 5 pg/mL (range from 10 to 2430 pg/mL), and average intra- and inter-assay coefficients of variation were 4% and 5.5% respectively. For testosterone, the test has a minimum detection limit of 0.1 pg/mL (range from 6.1 to 600 pg/mL), and average intra- and inter-assay coefficients of variation were 2.5% and 5.6% respectively. There is minimal cross-reactivity for the estrasiol assay to estriol and estrone, and no detectable cross-reactivity of this antibody with progesterone, testosterone, cortisol, or DHEA. 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.

2.5 | Go/NoGo task

We utilized a Go/NoGo task to measure error-related brain activity. During this task, the Go/NoGo stimuli are presented for 200 ms, followed by an inter-trial interval (ITI) varying randomly between 600 and 1000 ms. The task takes approximately 10 min and participants could take breaks between blocks. The stimuli are green equilateral triangles presented randomly, 1.5 cm on each side, presented on a black background, in three different orientations; 80% of the triangles are vertically aligned and pointed up ("Go" stimuli) and 20% of the triangles are slightly tilted to the right or left ("No/Go" stimuli). Children are instructed to respond by clicking a mouse button to upward-pointing triangles and to withhold responses to slightly tilted triangles, and to be as fast and accurate as possible. There are a total of 420 trials (7 blocks of 60 trials each). At the end of each block, children receive feedback based on performance. If performance was 75% correct or lower, the message "Please try to be more accurate" was displayed; if performance was above 90% correct, the message "Please try to respond faster" was displayed; otherwise the message "You're doing a great job" was displayed.

2.6 | EEG recording and data reduction

Continuous EEG recordings were collected using an elastic cap and the ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands) and

data was processed offline with Brain Vision Analyzer (Brain Products, Gilching, Germany). Thirty-four electrode sites were used, as well as two electrodes on the left and right mastoids. Electrooculogram (EOG) generated from eye movements and eyeblinks was recorded using four facial electrodes: horizontal eye movements were measured via two electrodes located approximately 1 cm outside the outer edge of the right and left eyes. Vertical eye movements and blinks were measured via two electrodes approximately 1 cm above and below the right eye. The EEG signal was pre-amplified at the electrode to improve the signal-to-noise ratio and amplified with a gain of one by a BioSemi ActiveTwo system. The data was digitized at a 24 bit resolution with a sampling rate of 1024 Hz using a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz. Each active electrode was measured online with respect to a common mode sense (CMS) active electrode producing monopolar (non-differential) channel. Offline, all data was referenced to the average of the left and right mastoids, and band-pass filtered between 0.1 and 30 Hz; eye-blink and ocular corrections were conducted per Gratton, Coles, and Donchin (1983).

A semi-automatic procedure was employed to detect and reject artifacts. The criteria applied were a voltage step of more than 50.0 μV between sample points, a voltage difference of 300.0 μV within a trial, and a maximum voltage difference of less than .50 μV within 100 ms intervals. These intervals were rejected from individual channels in each trial. Visual inspection of the data were then conducted to detect and reject any remaining artifacts.

The EEG were segmented for each trial, beginning 300 ms before the response and continuing for 1,000 ms after the response. The response-locked ERPs were averaged separately for each trial type (e.g., correct and incorrect responses), and baseline correction was performed using the interval from −500 to −300 ms. Average amplitude at Fz between 0 and 100 ms after response was exported for each subject. In order to obtain a measure of differentiation between errors and correct responses, the average amplitude related to correct responses was subtracted from the average amplitude related to errors (i.e., the ΔERN).

Behavioral measures included both the number of error trials for each subject, as well as accuracy expressed as a percentage of all valid trials. Average reaction times (RTs) on error and correct trials were calculated separately, as well as RTs on correct trials following correct and error trials to evaluate post-error RT slowing (RTs on post-error trials minus RTs on post-correct trials; Gehring & Knight, 2000; Hajcak, McDonald, & Simons, 2003; Kerns et al., 2004).

Statistical analyses were conducted using SPSS (Version 17.0) General Linear Model software, with Greenhouse-Geisser correction applied to *p* values associated with multiple-df, repeated-measures comparisons when necessitated by the violation of the assumption of sphericity. The Pearson correlation coefficient (*r*) was used to examine bivariate relationships between puberty hormones, PDS, age, behavioral data, as well as the ERN, CRN, and ΔERN. A repeated-measures ANOVA was utilized to examine error-related brain activity. To examine the unique impact of pubertal hormones on the ΔERN, we conducted a simultaneous multivariate regression wherein all significant factors—age, PDS, correct, and error reaction time, as well as all pubertal hormones were entered predicting the ΔERN.

3 | RESULTS

3.1 | Self-report

Overall, the average parent-reported PDS score across the sample was 2.54, *SD* = .79 with a range from 1 to 4. Child-reported PDS scores had a mean of 2.53, *SD* = .87, with a range from 1 to 4. Because parent and child-reports of pubertal status (PDS) were highly correlated, $r(97) = .89$, $p < .001$, we *z*-scored and combined parent and child PDS scores to obtain a single self-report measure of pubertal development. All subsequent analyses focus on this combined PDS score. Age and pubertal status were moderately correlated, $r(97) = .73$, $p < .001$.

3.2 | Hormones

Children were tested for concentration levels of pubertal hormones. Data on pubertal hormones was available for 99 participants within this sample. There was an average DHEA concentration of *M* = 76.87, *SD* = 51.15. The average testosterone concentration was found to be *M* = 50.54, *SD* = 20.23. The average concentration of estradiol was *M* = 2.21, *SD* = 0.94. There was an average progesterone concentration of *M* = 75.51, *SD* = 52.02. Overall, all hormones were normally distributed—that is, skewness and kurtosis ≤ 1. As can be seen in Table 1, age was moderately correlated to all pubertal hormones: DHEA, $r(97) = .38$, $p < .001$, testosterone, $r(97) = .43$, $p < .001$, estradiol, $r(97) = .33$, $p < .001$, and progesterone, $r(97) = .26$, $p < .001$. Additionally, PDS scores were moderately correlated to the following pubertal hormones: DHEA, $r(97) = .39$, $p < .001$, testosterone, $r(97) = .39$, $p < .001$, and estradiol, $r(97) = .28$, $p < .001$. PDS scores related to progesterone at a trend level, $r(97) = .18$, $p = .08$.

3.3 | Behavioral data

Overall, participants committed an average of 40.28, *SD* = 13.18, range = 10–81 errors, and made an average of 233.32, *SD* = 4.3, correct responses. Consistent with previous work, participants were faster on error trials, *M* = 329.64 ms, *SD* = 70.20, compared to correct trials, *M* = 395.44, *SD* = 82.53, $F(1,98) = 358.67$, $p < 001$. Reaction time

TABLE 1 Correlations between pubertal hormones and main study variables

	DHEA	Testosterone	Estradiol	Progesterone
Age	.38**	.43**	.33**	.26**
PDS total	.39**	.39**	.28**	.18
RT correct	−.15	−.24*	−.09	−.05
RT error	−.18	−.29*	−.14	−.08
Accuracy	.08	.02	.04	.00
Post-error slowing	.14	.11	.02	.10
ERN	−.17	−.19	−.03	.02
CRN	.12	.01	.03	.03
ΔERN	−.29**	−.23*	−.06	.00

PDS, pubertal development scale; RT, reaction time; ERN, error-related negativity; CRN, correct-related negativity; ΔERN, error minus correct.

did not differ on trials that occurred after an error $M = 319.16$, $SD = 73.69$, compared to trials that occurred after a correct response, $M = 311.70$, $SD = 56.02$, $F(1, 98) = 2.31$, $p = .13$. As can be seen in Table 1, testosterone levels were significantly correlated with reaction time on both error and correct trials. However, there were no other significant correlations between hormones and behavioral data. Age was significantly related to both error and correct reaction time, $r(97) = -.39$, $p < .05$, and $r(97) = -.38$, $p < .05$, respectively, as well as accuracy, $r(97) = .23$, $p < .05$. Puberty was significantly related to both error and correct reaction time, $r(97) = -.33$, $p < .05$, and $r(97) = -.33$, $p < .05$, respectively.

We then conducted partial correlations between puberty hormones, PDS and behavioral data, while controlling for age. Results suggested that when controlling for age, there were no significant correlations between hormones or PDS and behavioral data, all ps > .10.

3.4 | Error-related brain activity

Overall, the ERN, $M = -3.60$, $SD = 6.76$, was more negative than the CRN, $M = -.22$, $SD = 4.41$, $F(1, 98) = 33.67$, $p < .001$. While neither the ERN nor the CRN alone related to pubertal hormones, the ΔERN significantly related to both DHEA and testosterone, $r(97) = -.29$, $p < .01$, and $r(97) = -.23$, $p < .05$, respectively. All subsequent analyses focus on the ΔERN. The ΔERN was significantly related to both PDS, $r(97) = -.26$, $p < .05$, and age, $r(97) = -.37$, $p < .001$, as well as both correct and error reaction time, $r(97) = .24$, $p < .05$, and $r(97) = .30$, $p < .05$, respectively. The ΔERN was not significantly related to accuracy or post-error slowing, all ps > .10.

To examine the unique impact of pubertal hormones on the ΔERN, we conducted a simultaneous multivariate regression wherein all significant factors—age, PDS, correct, and error reaction time, as well as all pubertal hormones were entered predicting the ΔERN. As can be seen in Table 2, when all variables are entered simultaneously, only age and DHEA levels significantly predict the ΔERN. To depict this relationship, Figure 1 features a scatterplot of the relationship between the ΔERN and DHEA. As can be seen, an enhanced ΔERN is related to increases in DHEA hormone levels. Figure 2 depicts waveforms and topographical headmaps of activity during error and correct trials, as well as the difference (error minus correct). For purposes of illustration, a median-split was performed on DHEA hormone levels—children characterized by high DHEA levels are depicted on the top and children characterized by low DHEA levels are depicted on the bottom. As can be seen in both the waveforms and headmaps, the ΔERN was larger among children with increased levels of DHEA.

4 | DISCUSSION

Results from the current study suggest that both self-reported stages of puberty and pubertal hormone levels related to increased error-related neural activity in a large sample of child and adolescent females.

TABLE 2 Simultaneous multiple regression wherein age, PDS (self-reported puberty stage), correct and error RT (reaction time), and puberty hormones were entered predicting the ΔERN (error minus correct activity)

	△ERN		
Variables entered	***B***	**Std. error**	***t***
	***N* = 99**		
Age	−1.17	.47	−2.45*
PDS	.34	.41	.84
Correct RT	−.02	.02	−.94
Error RT	.03	.02	1.53
DHEA	−.04	.02	−2.34*
Testosterone	−.02	.05	−.49
Estradiol	.87	.89	.98
Progesterone	.03	.01	1.81
Overall model: total R-squared	.26**		
Adjusted R-squared	.19**		

As can be seen, age and the DHEA hormone are significant independent predictors. Unstandardized beta (*B*), standard error (Std. error) and *t* values (*t*) are presented.
*$p < .05$, **$p < .01$.

While the ERN related to both DHEA and testosterone levels, results suggested that increased levels of DHEA related to ERN magnitude uniquely, even when controlling for the impact of age and all other pubertal hormones (i.e., testosterone, estradiol, and progesterone). Results from the current study are novel insofar as self-report and biological indicators of puberty relate to developmental increases in the ERN.

A wealth of previous work has identified a developmental increase in error-related brain activity across childhood and adolescence (Tamnes et al., 2013). Additionally, Davies et al. (2004) observed developmental increases sooner in females relative to males—which led them to hypothesize that increases in the ERN may be linked to

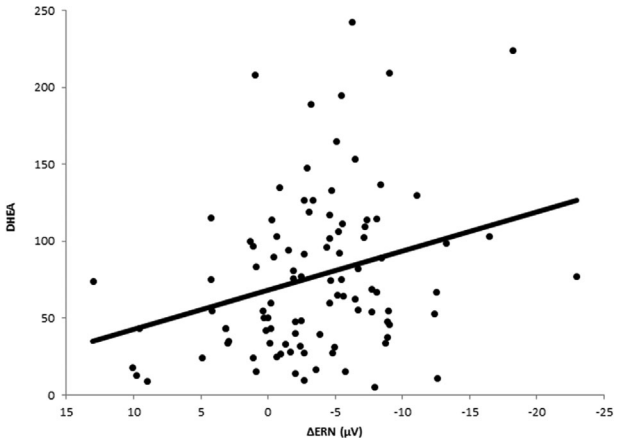

FIGURE 1 Scatterplot depicting the correlation between the error-related negativity (ΔERN) and a puberty hormone—that is, dehydroepiandrosterone (DHEA). As can be seen in the figure, as levels of DHEA increase, the magnitude of the ΔERN increases

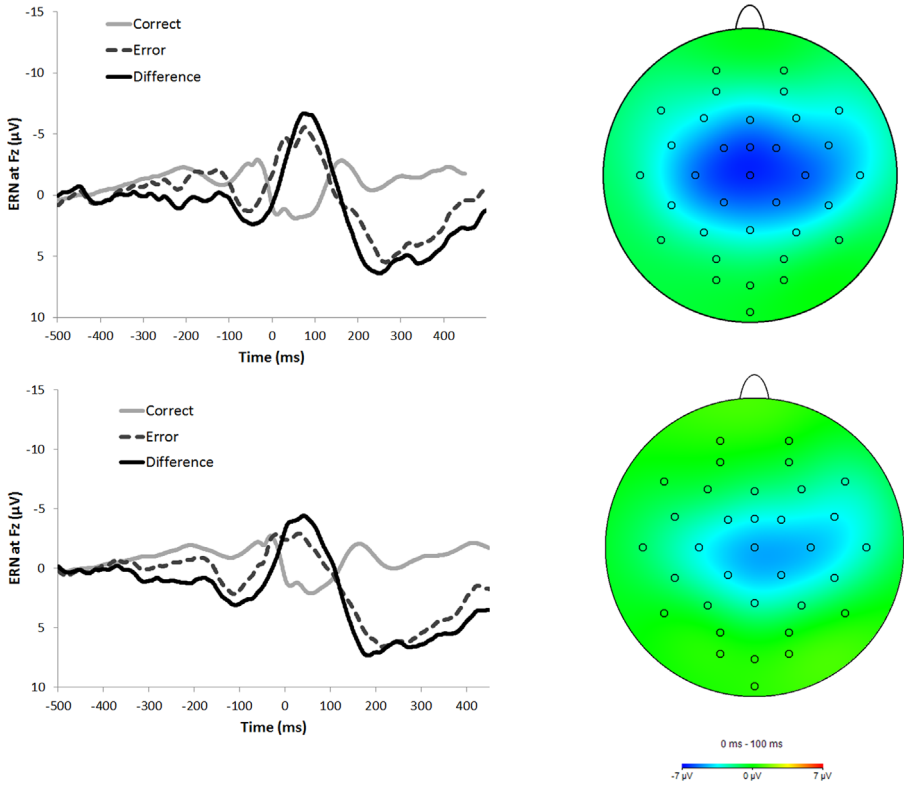

FIGURE 2 For purposes of illustration, a median-split was performed on DHEA hormone levels—children characterized by high DHEA levels are depicted on the top and children characterized by low DHEA levels are depicted on the bottom. On the left are waveforms depicting neural response (at electrode Fz) during error and correct trials, as well as the difference (error minus correct). On the right are topographical headmaps depicting for error minus correct during 0–100 ms after the response. As can be seen in both the waveforms and headmaps, the ΔERN was larger among children with increased levels of DHEA

pubertal onset. Despite these observations, no previous study has specifically examined the relationship between puberty and the ERN. Correlation analyses in the current investigation suggested that the ERN related to self-reported pubertal levels (i.e., PDS scores—combined parent and child report), age, DHEA, and testosterone. Follow-up regression analyses suggested that both age and DHEA have a unique relationship to increases in ERN magnitude, controlling for other pubertal hormones and behavior on the Go/No-Go task. Thus, puberty does appear to have a unique influence on the ERN.

Puberty is a foundational aspect of human development, resulting from an influx of hormones that set off a cascade of physical changes (i.e., menstruation, breast development, pubic, and axillary hair, sexually dimorphic changes in facial structure and muscle composition, and vocal changes). In addition to these physical changes, hormones also induce neurodevelopmental changes (Schulz, Molenda-Figueira, & Sisk, 2009). Puberty-induced physical and neural changes prepare individuals to gain increasing independence from caregivers and to navigate complex social interactions (Dahl, 2004). Specifically, the ability to monitor and control one's own behavior becomes increasingly important during this time. In line with this, the ERN may index increased cognitive control or concern over one's own behavior (Cavanagh & Shackman, 2014; Hajcak, 2012) and results from the current study implicate puberty as an important factor in the development of this neural marker. Moreover, the ERN has also been identified as a biomarker for anxiety symptoms (Meyer, 2016) —it is possible that the pubertally induced increases observed in the ERN in the current study underlie normative increases in anxiety during this developmental period. Future work is needed to clarify what specific psychological changes the developmental increase in the ERN may index.

Results from the current study suggested that DHEA may play a unique role in the development of the ERN. Dehydroepiandrosterone (DHEA) is a sex steroid hormone which works as a precursor for the synthesis of estrogens in females and androgens in males (Hopper & Yen, 1975). Typically, between the ages of 6 and 9 years old, in females, DHEA begins to increase and adrenarche begins. Adrenarche is associated with secondary sexual characteristics such as pubic hair and changes in sweat or body odor (Dorn, 2006). Along with its role in the production of sex hormones, increased levels of DHEA have also been linked to anxiety symptoms in developmental populations (Murray et al., 2016). For example, Murray et al. (2016) found that DHEA levels related to increased pituitary gland volume, which in turn, related to increased social anxiety symptoms in a large sample of children approximately 9 years old. Results from the current study are in line with this finding—suggesting that DHEA may play a unique role in the neurodevelopment process underlying increases in anxiety symptoms and self-monitoring that are commonly observed during the transition between childhood to adolescence.

One limitation of the current investigation is that the relationship between the ERN and DHEA was investigated only among females. Future studies should extend this work to examine whether pubertal hormones impact the development of the ERN in male populations as well. Moreover, the current investigation was conducted in sample characterized by a broad age-range. Although we statistically controlled for age in the analyses, future work could examine the relationship between the ERN and pubertal hormones in a more homogenous age-range to better control for age-related changes. Moreover, the current investigation did not include measures of cognitive control or anxiety symptoms. Future studies should explore to what extent DHEA-related changes in the ERN relate to psychological or cognitive changes across development.

The current findings raise the possibility that the functioning of the anterior cingulate cortex (ACC) during response monitoring may be potentiated by increases in DHEA. Indeed, previous work has implicated DHEA in the development of the *structure* of the ACC in developmental populations (Nguyen et al., 2013). This work paves the way for future translational work wherein the impact of hormones on specific neural mechanisms can be investigated and potentially provide novel targets for interventions. For example, work in animals may provide the basis for hormone-based pharmacological interventions aimed at reducing developmental increases in anxiety in at-risk populations.

NOTES

There are no conflicts of interest to report. All authors are affiliated with the Florida State University psychology department. This research was supported by Grant Number R01 MH097767 from the National Institute of Mental Health.

ENDNOTE

[1] It should be noted that the ACC activity has been observed in processes other than error processing (Bush, Luu, & Posner, 2000; Devinsky, Morrell, & Vogt, 1995; MacDonald, Cohen, Stenger, & Carter, 2000; Vogt, Finch, & Olson, 1992).

ORCID

Julia Y. Gorday http://orcid.org/0000-0002-1040-7934
Alexandria Meyer http://orcid.org/0000-0002-1298-5922

REFERENCES

Blakemore, S.-J., Burnett, S., & Dahl, R. E. (2010). The role of puberty in the developing adolescent brain. *Human Brain Mapping*, *31*(6), 926–933. https://doi.org/10.1002/hbm.21052

Bond, L., Clements, J., Bertalli, N., Evans-Whipp, T., McMorris, B. J., Patton, G. C., . . . Catalano, R. F. (2006). A comparison of self-reported puberty using the Pubertal Development Scale and the Sexual Maturation Scale in a school-based epidemiologic survey. *Journal of Adolescence*, *29*(5), 709–720. https://doi.org/10.1016/j.adolescence.2005.10.001

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, *4*(6), 215–222.

Buzzell, G. A., Richards, J. E., White, L. K., Barker, T. V., Pine, D. S., & Fox, N. A. (2017). Development of the error-monitoring system from ages 9–35: Unique insight provided by MRI-constrained source localization of EEG. *Neuroimage*, *157*, 13–26.

Carskadon, M. A., & Acebo, C. (1993). A self-administered rating scale for pubertal development. *Journal of Adolescent Health*, *14*(3), 190–195.

Casey, B. J., Duhoux, S., & Cohen, M. M. (2010). Adolescence: What do transmission, transition, and translation have to do with it? *Neuron*, *67*(5), 749–760. https://doi.org/10.1016/j.neuron.2010.08.033

Cavanagh, J. F., & Shackman, A. J. (2014). Frontal midline theta reflects anxiety and cognitive control: Meta-analytic evidence. *Journal of Physiology-Paris*, *109*(1-3), 3–15.

Coleman, J. R., Watson, J. M., & Strayer, D. L. (2017). Working memory capacity and task goals modulate error-related ERPs. *Psychophysiology*, *55*(3). https://doi.org/10.1111/psyp.12805

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. Keynote address. *Annals of the New York Academy of Sciences*, *1021*(1), 1–22.

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004). Development of response-monitoring ERPs in 7- to 25-year-olds. *Developmental Neuropsychology*, *25*(3), 355–376.

Devinsky, O., Morrell, M. J., & Vogt, B. A. (1995). Contributions of anterior cingulate cortex to behaviour. *Brain*, *118*(1), 279–306.

Dorn, L. D. (2006). Measuring puberty. *Journal of Adolescent Health*, *39*(5), 625–626.

Fair, D. A., Dosenbach, N. U., Church, J. A., Cohen, A. L., Brahmbhatt, S., Miezin, F. M., . . . Schlaggar, B. L. (2007). Development of distinct control networks through segregation and integration. *Proceedings of the National Academy of Sciences,*, *104*(33), 13507–13512.

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and Clinical Neurophysiology*, *78*(6), 447–455.

Forbes, E. E., & Dahl, R. E. (2010). Pubertal development and behavior: Hormonal activation of social and motivational tendencies. *Brain and Cognition*, *72*(1), 66–72. https://doi.org/10.1016/j.bandc.2009.10.007

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science (Wiley-Blackwell)*, *4*(6), 385–390.

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience*, *3*(5), 516–520.

Gratton, G., Coles, M. G., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, *55*(4), 468–484.

Hajcak, G. (2012). What we've learned from mistakes. *Current Directions in Psychological Science*, *21*(2), 101–106. https://doi.org/10.1177/0963721412436809.

Hajcak, G., McDonald, N., & Simons, R. F. (2003). To err is autonomic: Error-related brain potentials, ANS activity, and post-error compensatory behavior. *Psychophysiology*, *40*(6), 895–903.

Hopper, B., & Yen, S. (1975). Circulating concentrations of dehydroepiandrosterone and dehydroepiandrosterone sulfate during puberty. *The Journal of Clinical Endocrinology & Metabolism*, *40*(3), 458–461.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*(5660), 1023–1026.

Klawohn, J., Endrass, T., Preuss, J., Riesel, A., & Kathmann, N. (2016). Modulation of hyperactive error signals in obsessive-compulsive disorder by dual-task demands. *Journal of Abnormal Psychology*, *125*(2), 292.

Ladouceur, C. D., Dahl, R. E., & Carter, C. S. (2007). Development of action monitoring through adolescence into adulthood: ERP and source localization. *Developmental Science*, *10*(6), 874–891.

Lebel, C., & Beaulieu, C. (2011). Longitudinal development of human brain wiring continues from childhood into adulthood. *The Journal of Neuroscience*, *31*(30), 10937–10947.

Lebel, C., Gee, M., Camicioli, R., Wieler, M., Martin, W., & Beaulieu, C. (2012). Diffusion tensor imaging of white matter tract evolution over the lifespan. *Neuroimage*, *60*(1), 340–352.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science*, *288*(5472), 1835–1838.

Maier, M. E., & Steinhauser, M. (2017). Working memory load impairs the evaluation of behavioral errors in the medial frontal cortex. *Psychophysiology*, *54*(10):1472–1482.

Meyer, A. (2016). Developing psychiatric biomarkers: A review focusing on the error-Related negativity as a biomarker for anxiety. *Current Treatment Options in Psychiatry*, *3*(4):1–9.

Meyer, A., Hajcak, G., Torpey-Newman, D. C., Kujawa, A., & Klein, D. N. (2015). Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. *Journal of Abnormal Psychology*, *124*(2), 266.

Meyer, A., Hajcak, G., Torpey, D. C., Kujawa, A., Kim, J., Bufferd, S., & Klein, D. N. (2013). Increased error-related brain activity in six-year-old children with clinical anxiety. *Journal of Abnormal Child Psychology*, *41*(8), 1257–1266.

Miller, A. E., Watson, J. M., & Strayer, D. L. (2012). Individual differences in working memory capacity predict action monitoring and the error-related negativity. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *38*(3), 757.

Moser, J. S., Moran, T. P., Schroder, H. S., Donnellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. *Frontiers in Human Neuroscience*, *7*, 466.

Murray, C. R., Simmons, J. G., Allen, N. B., Byrne, M. L., Mundy, L. K., Seal, M. L., & Whittle, S. (2016). Associations between dehydroepiandrosterone (DHEA) levels, pituitary volume, and social anxiety in children. *Psychoneuroendocrinology*, *64*, 31–39.

Nguyen, T.-V., McCracken, J. T., Ducharme, S., Cropp, B. F., Botteron, K. N., Evans, A. C., & Karama, S. (2013). Interactive effects of dehydroepiandrosterone and testosterone on cortical thickness during early brain development. *Journal of Neuroscience*, *33*(26), 10840–10848.

Op de Macks, Z. A., Moor, B. G., Overgaauw, S., Güroğlu, B., Dahl, R. E., & Crone, E. A. (2011). Testosterone levels correspond with increased ventral striatum activation in response to monetary rewards in adolescents. *Developmental Cognitive Neuroscience*, *1*(4), 506–516. https://doi.org/10.1016/j.dcn.2011.06.003.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, *17*(2), 117–133.

Santesso, D. L., & Segalowitz, S. J. (2008). Developmental differences in error-related ERPs in middle-to late-adolescent males. *Developmental Psychology*, *44*(1), 205.

Schulz, K. M., Molenda-Figueira, H. A., & Sisk, C. L. (2009). Back to the future: The organizational-activational hypothesis adapted to puberty and adolescence. *Hormones and Behavior*, *55*(5), 597–604. https://doi.org/10.1016/j.yhbeh.2009.03.010

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews Neuroscience*, *12*(3), 154–167.

Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). Pubertal development: Correspondence between hormonal and physical development. *Child Development*, *80*(2), 327–337.

Sisk, C. L., Schulz, K. M., & Zehr, J. L. (2003). Puberty: A finishing school for male social behavior. *Annals of the New York Academy of Sciences*, *1007*(1), 189–198. https://doi.org/10.1196/annals.1286.019

Sisk, C. L., & Zehr, J. L. (2005). Pubertal hormones organize the adolescent brain and behavior. *Frontiers in Neuroendocrinology*, *26*(3), 163–174.

Spear, L. P. (2000). The adolescent brain and age-related behavioral manifestations. *Neuroscience & Biobehavioral Reviews*, *24*(4), 417–463.

Tamnes, C. K., Walhovd, K. B., Torstveit, M., Sells, V. T., & Fjell, A. M. (2013). Performance monitoring in children and adolescents: A review of developmental changes in the error-related negativity and brain maturation. *Developmental Cognitive Neuroscience*, *6*(0), 1–13. https://doi.org/10.1016/j.dcn.2013.05.001

Tanner, J. M., & Whitehouse, R. H. (1976). Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Archives of Disease in Childhood*, *51*(3), 170–179.

van Veen, V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: FMRI and ERP studies. *Physiology & Behavior*, *77*(4-5), 477–482. https://doi.org/10.1016/s0031-9384(02)00930-7

Velanova, K., Wheeler, M. E., & Luna, B. (2008). Maturational changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. *Cerebral Cortex*, *18*(11), 2505–2522.

Vogt, B. A., Finch, D. M., & Olson, C. R. (1992). Functional heterogeneity in cingulate cortex: The anterior executive and posterior evaluative regions. *Cerebral Cortex*, *2*(6), 435–443.

How to cite this article: Gorday JY, Meyer A. Linking puberty and error-monitoring: Relationships between self-reported pubertal stages, pubertal hormones, and the error-related negativity in a large sample of children and adolescents. *Dev Psychobiol*. 2018;1–8. https://doi.org/10.1002/dev.21625