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# Pubertal maturation and cortisol level in response to a novel social environment among female adolescents

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A B S T R A C T

Research indicates changes in HPA-axis activity across puberty. The current study extends existing work by evaluating pubertal status and cortisol level in a novel social environment (research laboratory) while controlling for several important biological, behavioral, and psychological variables. Participants were 30 girls (ages 9–16 years) from the United States. Pubertal status was assessed via the Pubertal Development Scale. Salivary samples were collected at laboratory-introduction and a matched at-home period; laboratory-introduction levels were regressed on basal cortisol levels to create standardized residual scores. After controlling for covariates, pubertal status was positively associated with residualized cortisol values. Findings indicate more advanced puberty related to greater cortisol response to the laboratory; data are discussed in terms of future research and building biopsychosocial models of the puberty-psychopathology linkage.


A relatively brief period of profound biopsychosocial development, advancing puberty is associated with enhanced emotional lability, increased susceptibility to negative affectivity (Brooks-Gunn, Graber, & Paikoff, 1994; Larson & Ham, 1993; Susman, Dorn, & Chrousos, 1991), and increased risk for the development of anxiety/mood psychopathology (e.g., depression; Angold, Costello, Erkanli, & Worthman, 1999). Moreover, particularly among girls, enhanced psychological vulnerability is linked to pubertal maturation specifically, as opposed to increasing age generally (Reardon, Leen-Feldner, & Hayward, 2009). A pressing question pertains to identifying aspects of this normative biopsychosocial process that contribute to enhanced risk for psychopathology.

Contemporary models seeking to understand adolescent vulnerability not only emphasize relevant social (Biro & Dorn, 2006) and cognitive (Remmel & Flavell, 2004) features of puberty, but also biological facets, including maturation of pertinent neuroendocrine axes and resultant hormone production (Angold & Costello, 2006; Granger, Weisz, McCracken, Ikeda, & Douglas, 1996). For example, adrenarche, the first phase of puberty, involves activation and maturation of the hypothalamic-

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pituitary-adrenal (HPA) axis (Fechner, 2003). The HPA-axis is of particular interest to puberty-psychopathology researchers given well-documented HPA-axis dysregulation among individuals with specific mood/anxiety disorders (e.g., Barden, 2004; Yehuda & LeDoux, 2007). One downstream output of HPA-axis activity is the hormone cortisol. Cortisol evidences characteristic diurnal variation, is assessed easily via salivary sampling, and remains stable under less-than-optimal storage conditions (e.g., temperature variations; Granger et al., 2012; Kirschbaum & Hellhammer, 1994). Although salivary cortisol values are significantly lower than that found in blood, research consistently indicates that salivary cortisol is highly correlated with serum and plasma concentrations (e.g., *r*'s range from .71 to .92; Kirschbaum & Hellhammer, 1994), and thus provides a valid and reliable alternative to more invasive sampling techniques. Conceptualized as a "stress system," two dimensions of HPA-axis activity reflected in cortisol levels may be relevant to understanding psychological vulnerability: (a) basal levels (overall concentration/fluctuation patterns), and (b) "stress reactivity" (e.g., in the context of novelty/uncertainty/challenge; Kirschbaum & Hellhammer, 1994).

In regard to the first dimension, a growing body of work indicates that puberty is related to sex-specific changes in cortisol output at selected sample times (e.g., early evening, Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Netherton, Goodyer, Tamplin, & Herbert, 2004) resulting in subtle, but significant shifts in total output (e.g., Kiess et al., 1995) and diurnal rhythm (e.g., Shirtcliff et al., 2011). For example, in a large sample of adolescents followed from age 9–15 years, Shirtcliff et al. (2011) identified a "U-shaped" pattern of total cortisol output as a function of advancing pubertal stage, including sex-specific alterations to both the shape and slope of diurnal rhythm. Collectively, extant work supports the contention that pubertal development is associated with complex, meaningful changes in basal HPA-axis activity which may, in turn, be related to enhanced psychological vulnerability, particularly among girls (see also Adam, 2006; Legro, Lin, Demers, & Lloyd, 2003; Rosmalen, Oldehinkel, Ormel, De Winter, Buitelaar, & Verhulst, 2005; Shirtcliff & Essex, 2008).

A convergence of theoretical and empirical work further suggests that increases in the *stress reactivity* of the HPA-axis also may occur across the course of puberty (e.g., Hastings et al., 2011; Ladouceur, 2012; Sanborn & Hayward, 2003; Susman et al., 1991). Indeed, animal models consistently suggest puberty-related differences in stress-related cortisol output (Romeo, 2010); however, data from human research are not uniform. For example, in a sample of 30 adolescents, Buske-Kirschbaum et al. (1997) found that parental report of pubertal events (e.g., pubic hair) was not related to cortisol output in response to the Trier Social Stress Test (TSST; a psychosocial stressor recognized for consistent, robust increases in cortisol output; Gunnar, Talge, & Herrera, 2009; Kirschbaum, Pirke, & Hellhammer, 1993; Martel et al., 1999). Similarly, Gunnar, Wewerka, et al. (2009) found marginal but non-significant associations between self-reported maturation and TSST response in a sample of 13-year-olds for whom pubertal data was available ($n = 20$). Conversely, in the largest study to date to directly examine human HPA-axis reactivity as a function of pubertal status (cf. pubertal *timing*; Natsuaki, Klimes-Dougan, Ge, Shirtcliff, Hastings, & Zahn-Waxler, 2009), and the only to address the role of sex in this relation, Stroud, Papandonatos, Williamson, and Dahl (2004) found that girls (but not boys) evidence increases in total cortisol following a corticotrophin-releasing hormone challenge across advancing pubertal stage. To date, extant data suggest that puberty may be related to increased HPA-axis activity in response to emotional/physical challenge among *female* adolescents (e.g., Gunnar, Wewerka, et al., 2009; Stroud et al., 2004); however, available evidence is mixed and no work has examined this relation in the context of more subtle stressors such as a novel (cf. universally stressful) social environment.

Extending available work to understanding HPA-axis activity in the context of novelty is important because acute responding to isolated, extreme stressors may not be necessary or sufficient for dysregulation of the HPA-axis (e.g., van der Kolk, 2003). Further, in addition to increased exposure to stressful events (e.g., traumatization; Copeland, Keeler, Angold, & Costello, 2007), adolescence is characterized by exposure to novel social environments (e.g., classroom transitions, new social/romantic encounters); thus responsivity to more subtle stressors, including unique environments, is particularly relevant to the etiology of risk during this period (Turner-Cobb, 2005). Work utilizing animal models has identified significant, lasting neuroendocrine and behavioral effects of single and repeated mild stress exposure during adolescence (e.g., Haas & George, 1988; Pohl, Olmstead, Wynne-Edwards, Harkness, & Menard, 2007; Yuen et al., 2012). Although acute responding to mild stressors initially may be minimal, repeated exposure may sensitize the system, resulting in enhanced responding (including corticosterone secretion) to subsequent stressors (e.g., Pohl et al., 2007; Schmidt et al., 2007). Thus, understanding more nuanced reactivity in adolescent humans may be important in identifying facets of puberty that may confer risk for long-term alterations and correlated psychopathology. Indeed, whereas research suggests that exposure to novelty does not elevate cortisol among young children (Gunnar, Talge, et al., 2009), age is positively associated with cortisol output in the context of a novel social environment among adolescents (Gunnar, Wewerka, et al., 2009). Of note, no work has yet examined this relation as a function of pubertal status. This gap in the literature is problematic, given consistent evidence for the unique incremental validity of pubertal variables, relative to age, in predicting psychosocial outcomes among youth (e.g., Angold & Costello, 2006).

Finally, HPA-axis reactivity is associated with a number of relevant psychological, behavioral, and environmental factors (e.g., Kudielka, Hellhammer, & Wüst, 2009). For example, atypical HPA-axis activity has been linked to depression (Barden, 2004), social anxiety (Condren, O'Neill, Ryan, Barrett, & Thakore, 2002), and panic (Abelson, Khan, Liberzon, & Young, 2007); all of which evidence linkages with puberty among girls (Angold et al., 1999; Deardorff et al., 2007; Hayward et al., 1992). Further, changes in cortisol secretion have been linked with nicotine and medication use (e.g., dietary, allergy), pregnancy, recent nutrient intake (Kudielka et al., 2009) and traumatization (Lipschitz, Morgan, & Southwick, 2002). In order to identify the *unique* relation between puberty and cortisol output, these variables must be taken into account.