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The roles of puberty and age in explaining the diminished effectiveness of parental buffering of HPA reactivity and recovery in adolescence

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Summary Parental support is a powerful regulator of stress and fear responses for infants and children, but recent evidence suggests it may be an ineffective stress buffer for adolescents. The mechanisms underlying this developmental shift are not well-understood. The goal of the present study was to examine the independent and joint contributions of pubertal status and chronological age in explaining this shift. A sample of 75 typically developing youth (*M* age = 12.95 years, *SD* = 0.70, range = 11.7—14.6 years; 37 females) was recruited to complete a modified Trier Social Stress Test (TSST-M) in the laboratory. Participants were recruited in such a way as to disentangle pubertal stage and chronological age by phone screening for markers of pubertal stage and then recruiting roughly equal numbers of younger and older, pre/early and mid/late pubertal youth who were then randomly assigned within groups to condition. The TSST-M was used as the stressor and youth prepared either with their parent or stranger (parent condition: *N* = 39). Pubertal stage was confirmed by the Petersen Pubertal Development Scale at the time of testing and treated, along with chronological age, as a continuous variable in the analyses. The results revealed an interaction of pubertal stage and support condition for cortisol reactivity to the TSST-M such that preparing for the speech with the parent became a less potent buffer of the HPA axis as pubertal stage increased. Age did not interact with condition in predicting cortisol reactivity. In contrast, the parent's presence during speech preparation decreased in its effectiveness to hasten recovery of the HPA axis as children got older, but pubertal stage was not predictive of recovery rate. These patterns were specific to cortisol and

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were not observed with salivary alpha-amylase levels or subjective stress ratings for the task. These analyses suggest that the switch away from using parents as social buffers may be the result of neurobiological mechanisms associated with puberty.

1. Introduction

For the young of many mammalian species including our own, contact and proximity with the attachment figure is critical to survival. When threatened or stressed, young animals and children seek this proximity and the presence of the attachment figure provides a powerful buffer of the infant's fear and stress systems (Hennessy et al., 2009). Termed parental social buffering, this regulating effect of the attachment figure's presence on key stress mediating systems, such as the hypothalamic—pituitary—adrenocortical (HPA) system, has been demonstrated multiple times in infants and toddlers (Gunnar et al., 1996; Nachmias et al., 1996; Spangler and Schieche, 1998; Ahnert et al., 2004).

Stress buffering has been observed in adults as well. Among adults, romantic relationship partners (Kirschbaum et al., 1995) and close friends (Fontana et al., 1999; Uno et al., 2002) have been shown to reduce cortisol and autonomic responses to social stressors. Furthermore, the presence of another person is not sufficient for stress buffering; instead, it seems that a degree of intimacy with the person providing the buffering is required (Kirschbaum et al., 1995). Indeed, for adults, a period of self-disclosure that enhances intimacy is sufficient to increase the value of a stranger's presence as a source of social buffering of the HPA axis (Smith et al., 2009).

Until recently, there had been little work examining parental social buffering beyond infancy. One research group did show that among 7- to 12-year old females, recovering from a social stressor task with their mother reduced cortisol to baseline faster than doing so without any maternal contact, and even talking to their mother on the phone provided some benefit (Seltzer et al., 2010). This study also showed that contact with the mother, in person or by phone, increased the production of oxytocin (Seltzer et al., 2010). In work with adults, nasal oxytocin has been shown to produce effects comparable to a partner's presence in reducing cortisol responses to a social stressor task (Heinrichs et al., 2003). Evidence of oxytocin as a potential mediator or correlate of social buffering underscores the importance of the bond between social partners as critical to the buffering potency of the other person's presence.

One question with regards to parental social buffering is how long in development the buffering potency of the parent lasts. If social buffering organized around parents is part of an immature mode of coping with danger and threat, then one would expect it to diminish in potency as the child approaches independence. Recently we examined the effectiveness of the parent as a social buffer of the HPA axis among children aged 9- and 10-years-old and adolescents aged 15- and 16-years-old (Hostinar et al., 2015). Among the children, the presence of the parent during the time the child prepared for the speech in the Trier Social Stress Test completely blocked elevations in cortisol despite the fact that the parent was not present during the speech and math section of the task. Among the adolescents the parent's presence had no effect. In addition, as would be expected, basal levels of cortisol were higher among the adolescents than the children (Dahl and Gunnar, 2009). In other work, researchers have shown that with adolescence, the mother's presence no longer buffers the amygdala responses to threat stimuli, allowing fear conditioning to occur even when the mother is present and not indicating fear of the conditioned stimulus (Gee et al., 2014).

Thus, there is evidence of a reduction of parental social buffering potency with the transition from childhood to adolescence. The question addressed in this study is the extent to which this reduction is associated with puberty or with age changes in processes that are unrelated to puberty. We hypothesized that the capacity of the parent's presence to reduce reactivity of the HPA axis to a social stressor would decrease in relation to pubertal stage and not child age. To provide a more fine-grained analysis, we differentiated reactivity and recovery components of the HPA response to the social stressor task as suggested by Juster and colleagues (2012). To help determine whether the parent's presence operated through reducing how frightened or anxious the children were, we obtained the children's self-reports of stress at different points in the procedure. We also collected measures of alpha-amylase, an indirect indicator of autonomic arousal, to examine whether this phenomenon is specific to the HPA axis or applies to both stress-mediating systems.

2. Methods

2.1. Participants

A total of 81 youth ages 11—14 were recruited from a department-maintained participant pool and were enrolled in the study. Exclusion criteria included the use of steroid or psychotropic medications, and diagnosis of Autism Spectrum Disorder, Fetal Alcohol Spectrum Disorder, or any other developmental disorder. Six adolescents were excluded from analysis for taking medications that likely affect cortisol levels (e.g., corticosteroids, diabetes injections, vasopressin analogs, antidepressant, testosterone injections, beta-blockers, immunosuppressants). Participant data below is reported without these 6 excluded participants. A total of 75 typically developing youth (M age = 12.95, SD = 0.70, range = 11.67—14.58 years; 37 females) were included in all analyses. Approximately half of each sex was assigned to prepare for the stressor task with their parent present (23 males/16 females) and the others prepared with the stranger present (15 males/21 females). To balance pubertal status between the sexes, the average age of the males (M age = 13.27 years, SD = 0.69, range: 11.75—14.58) was about