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The influence of maternal care and overprotection on youth adrenocortical stress response: A multiphase growth curve analysis

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Abstract

We examined the associations between 2 dimensions of maternal parenting style (care and overprotection) and cortisol response to an acute laboratory-induced stressor in healthy youth. Forty-three participants completed the Parental Bonding Instrument and an adapted version of the Trier Social Stress Test-Child (TSST-C). Nine cortisol samples were collected to investigate heterogeneity in different phases of youth's stress response. Multiphase growth-curve modeling was utilized to create latent factors corresponding to individual differences in cortisol during baseline, reactivity, and recovery to the TSST-C. Youth report of maternal overprotection was associated with lower baseline cortisol levels, and a slower cortisol decline during recovery, controlling for maternal care, puberty, and gender. No additive or interactive effects involving maternal care emerged. These findings suggest that maternal overprotection may exert a unique and important influence on youth's stress response.

Keywords

Cortisol; Parenting Style; Maternal Care; Maternal Overprotection; Stress Response; Youth

Both maternal and paternal parenting style can exert a powerful influence on youth adjustment and maladjustment; however the literature has primarily examined the role of mothers because mothers tend to be the primary caregivers in families. In this study we focus on how different maternal parenting styles may have varying effects on development. For example, maternal care refers to a style of parenting characterized by individual differences in the degree to which mothers engage in caring and responsive behaviors toward their child. High levels of maternal care are thought to promote youth's self and world exploration (Parker, Tupling & Brown 1979; Rohner 1986). Maternal overprotection refers to a style of parenting characterized by controlling, demanding, intrusive, highly supervising and vigilant behaviors by the mother; which may encourage youth infantilization and

discourage independent emotions/behaviors (Parker 1983; Thomasgard & Metz 1993, 1997). Past research suggests that the influence of parenting style is crucial in understanding the development of youth internalizing and externalizing disorders (Albano, Chorpita & Barlow 2003; Roelofs, Meesters, ter Huurne, Bamelis & Muris 2006). In particular, previous research has shown that maternal overprotection is associated with youth anxiety disorders and behavior problems (Gere, Villabø, Torgersen & Kendall 2012; McLeod, Wood & Weisz 2007) and that the synergistic effect of low maternal care and high maternal overprotection is associated with externalizing behaviors (Weaver & Plelow 2005). Furthermore, there is research to suggest that maternal care can serve as a protective factor against youth maladjustment (e.g., Skopp, McDonald, Jouriles & Rosenfield 2007). However, the mechanisms by which these two maternal parenting styles (care and overprotection) influence youth adjustment are not well understood.

Maternal parenting style may relate to youth adjustment by influencing youths' pattern of cortisol response to stress (Blair, Granger, Willoughby & Kivlighan 2006; Del Giudice, Ellis & Shirtcliff 2011; Cicchetti & Rogosch 2001). When an individual experiences a stressful event, the hypothalamic-pituitary-adrenocortical (HPA) axis is activated and releases glucocorticoids (namely, cortisol) from the adrenal cortex. Cortisol mediates the physiological changes that occur in response to stress (e.g., increased glucose/energy/blood flow; engages the central nervous system) and aids homeostatic regulation (Kemeny 2003). However, there are between-person differences in the degree to which these processes are activated by stress, and we (and others) propose that specific maternal parenting behaviors may alter youths' stress response; which in turn may lead to different youth adjustment outcomes. In support of this view, animal studies have demonstrated that rat pups who received high maternal care (more licking and grooming) during the first week of life displayed decreased response to stress, and less behavioral fearfulness that lasted into adulthood (for a review see Kaffman & Meaney 2007). Similarly, translational human research has shown that individuals who experienced adverse environments early in life, such as maltreatment, neglect, abuse, and poor parenting, show altered acute stress responses (i.e., HPA axis dysregulation; for a review see Gunnar & Quevedo, 2007), though evidence of the direction of these effects is mixed.

Despite the mixed findings in human research, there is supporting evidence that maternal parenting behaviors early in life may contribute to systematic variations in offspring HPA axis regulation. For example, among 3-month olds, higher quality maternal care was associated with a faster cortisol decline during recovery from the daily bathing routine: an acute stressor (Albers, Riksen-Walraven, Sweep & de Weerth 2008). In adults, retrospective reports of both low and high parental care in childhood (compared to medium levels of parental care) were associated with reduced cortisol secretion in response to an acute laboratory stressor (Engert, Efanov, Dedovic, Duchesne, Dagher & Pruessner 2010). In a study of early life stress, Gunnar and colleagues (2009) compared 10-12 year old children adopted from orphanages at age one or older (defined as group exposed to severe early life stress), children adopted from foster care before eight months of age (defined as group exposed to moderate early life stress), and controls (defined as non-adopted born and raised with birth families). They found that the groups exposed to moderate early life stress exhibited decreased cortisol activity in response to the Trier Social Stress Test for Children

(TSST-C): an acute stressor (Gunnar et al., 2009). This finding is surprising in light of research that suggests that exposure to early life stress is associated with increased cortisol reactivity to stress (at least among children with psychiatric predispositions and disorders; e.g., De Bellis et al., 1999; Carrion et al., 2002). However, Gunnar and colleagues propose that their findings support the stress-inoculation hypothesis which suggests that some degree of early life stress exposure helps individuals cope with later life stress (Garmezy, 1991; Rutter, 1993). Together these studies suggest that maternal care behaviors may be related to a quick cortisol recovery and altered cortisol reactivity (support for both increased and decreased cortisol) to an acute stressor, but this effect may depend on: 1) the developmental phase of the offspring; and 2) severity of exposure to early life adversity.

The effects of maternal parenting styles on offspring stress response may be particularly important when examining the transition from childhood to adolescence, as this developmental phase is associated with increased independence from parents, self-identity formation, and increased stressors (Laursen & Collins 2009). Hackman, Betancourt, Brodsky, Kobrin, Hurt & Farah (2013) found that low parental responsivity (mostly, maternal responsivity; 92% of the sample assessed mothers) during early childhood was associated with blunted cortisol reactivity to a laboratory stressor in adolescence. Though, Ellenbogen and Hodgins (2009) found that low levels of maternal and paternal structure (e.g., organization and consistency) during middle childhood predicted increased cortisol levels following awakening and to the standard Trier Social Stress Test (TSST) among adolescents. These studies suggest that the effects of maternal parenting style on youth's acute stress response may depend on the specific style of parenting assessed. There is growing evidence in both animal and human studies that the presence of a parent (usually the mother) reduces or eliminates the cortisol stress response (e.g., parental social buffering effect) in children, but not in adolescents (e.g., see Doom, Hostinar, VanZomeren-Dohm & Gunnar 2015, Hostinar, Johnson, & Gunner, 2014; Hostinar, Sullivan, Gunner, 2014 for human research; see Hennessy, Kaiser, & Sachser, 2009 for animal research). Thus, the protective effects of maternal care and support may be diminished as children transition into adolescence. However, from this work it is unclear if the effects of other maternal parenting styles continue to have an impact.

Most of the literature examining maternal parenting has focused on maternal care. We propose that as children transition into adolescence, maternal overprotective behaviors may have a particular effect on youth's ability to regulate stress. One possible reason is that the developmental tasks associated with the transition from childhood to adolescence may be at odds with maternal overprotective behaviors; maternal overprotective behaviors may shelter youth from experiencing stressors in terms of the number of stressors, the frequency of stressors, and the perceived severity of stressors. Consequently, when offspring do experience stressors, they may have difficulty coping with stress. Liu (2015) recently argued that a component of the stress-inoculation hypothesis would suggest that youth who experience low levels of early life stressors (relative to moderate stressors) may be more likely to exhibit greater stress sensitivity (akin to those exposed to high levels of early life stressors). In line with this hypothesis, we propose that the sheltering behaviors of maternal overprotection may lead to low levels of stress exposure, and thus increased reactivity to the small number of encountered stressors. Although speculative, we hypothesize that

physiologically, increased exposure to maternal overprotection may manifest as an exaggerated secretion of cortisol and then difficulty “turning-off” the release of cortisol in response to an acute stressor (i.e., slow return of cortisol levels to baseline).

Elucidating associations between specific maternal parenting styles and youth cortisol stress response has the potential to inform how different parental influences may affect youth adjustment. Furthermore, multiple parenting styles can co-occur. For example, displaying both maternal care and overprotection may be particularly likely as mothers are learning how to shift from parenting a child to parenting an adolescent. Thus, studies that only examine one maternal parenting style may obscure our understanding of the effects of maternal parenting style on youth stress response.

In sum, the majority of past research examining the association between maternal parenting style and offspring stress response has focused on: 1) infancy and early childhood; 2) adult retrospective reports of parenting; and 3) maternal care (versus other facets of parenting). The current study aims to address these gaps in the literature by examining the effects of maternal care and overprotection on youth stress response. We also extend prior literature by adopting an analytical approach (i.e., multiphase growth curve modeling) which allows us to estimate the associations between specific maternal parenting styles and discrete aspects of the stress response (i.e., baseline, reactivity, and recovery) as outlined by de Kloet, Joëls and Holsboer (2005).

Hypotheses

While there is no consensus on the definition of an “adaptive” stress response, de Kloet, Joëls and Holsboer (2005) suggest that an effective physiological stress response involves a rapid activation of the sympathetic nervous system, promoting the release of cortisol, and a quick return of cortisol levels to baseline once the stressor has subsided. In line with this view and the literature on the maternal parenting styles-youth stress response link we tested two main hypotheses. Specifically, we examined the hypothesis that youth report of high levels of maternal care would be associated with an adaptive cortisol response (steep or quick cortisol increase and a quick cortisol decline), while youth report of high levels of maternal overprotection would be associated with an overactive stress response (elevated cortisol increase and slow cortisol decline-suggesting difficulty recovering for a stressor). On an exploratory basis, we examined the interactive effect of youth's perceptions of maternal care and overprotection on stress response, as well as the interactive effects of youth pubertal development and maternal parenting style (both care and overprotection).

Methods

Participants

Participants in the current study included a subset of 43 healthy children with a mean age of 12.7 years ($SD = 2.4$; age range = 8 -17; 56% female) who were part of a larger study examining sex differences in stress response across puberty (NSF 0644171). Racial/ethnic distribution of the sample included 58% Non-Hispanic Caucasian, 30% Hispanic, 7% Asian, and 5% African-American children. Using the Hollingshead Four-Factor Index

(Hollingshead 1975), 29% of the sample came from high socioeconomic households (Hollingshead=1), 51% were from middle SES households (Hollingshead=2 or 3), and 20% were from low SES households (Hollingshead score=4 or 5). According to Tanner pubertal stage criteria, 44% (n=19) of the sample was in the early stages of puberty (score of 1-3) and 56% (n=24) of the sample was in the later stages of puberty (score of 3.5-5).

Recruitment Strategy

Participants were recruited for the study using community and online postings. Exclusion criteria included factors known to influence cortisol measurement, including use of oral contraceptives, thyroid medications, steroids, and psychotropic medications (Granger, Hibel, Fortunato & Kapelewski 2009). Additional exclusion criteria included diagnosis of autism or mental retardation, a history of psychological or behavioral problems, substance use, or current physical illness.

Assessment of Maternal Parenting Styles

The Parental Bonding Instrument (PBI; Parker, Tupling & Brown 1979) is a 25 item self-report measure that examines children's perceptions of their parents' caregiving styles. In the current study, children completed the PBI by reporting on their mothers' caregiving styles. The measure is designed to produce a two-factor model of childrearing consisting of a maternal care scale (12 items) and a maternal overprotection scale (13 items). Participants were given the following instructions: "Below is a list of statements that describe mothers. Please answer how much you disagree or agree with each statement about your MOTHER." Using a 4-point Likert scale, respondents rated each item based on how much they agreed or disagreed with statements about their mothers (1 = Disagree a Lot, 4 = Agree a Lot). Items on the maternal care scale included statements such as "My mother could make me feel better when I was upset," and "My mother spoke to me with a warm and friendly voice." Items on the maternal overprotection scale included statements such as "My mother tried to control everything I did," and "My mother invaded my privacy." The measure had good test-retest reliability (ranging from .60-.79), and convergent validity with interview-based ratings (Parker, Tupling & Brown 1979). It has also been validated on a variety of study populations, including youth (Klimidis, Minas & Ata 1992; Martin, Bergen, Roeger & Allison 2004; Wihelm, Niven, Parker & Hadzi-Pavlovic 2005). In the present study both the maternal care ($\alpha=.83$), and maternal overprotection ($\alpha=.78$) scales showed good internal consistency.

Assessment of Puberty

Tanner pubertal stage was assessed by having participants select the sex-specific pictorial representations that most closely matched their perceived pubertal developmental stage: breasts (B) and pubic hair (PH) for girls, and genitals (G) and PH for boys (Marshall & Tanner, 1969, 1970). Self-selected scores ranged from 1-5 for both B/G and PH. The mean of the self-selected Tanner B/G and PH stage was then calculated to create a single score of pubertal development. Using these Tanner stage means, puberty was dichotomized such that early or pre-pubertal development represented Tanner B/G and PH mean scores of 1-3 and late or post-pubertal development represented Tanner B/G and PH mean scores of 3.5-5.

Stress Induction and Cortisol Collection

An adapted version of the Trier Social Stress Test-Child (TSST-C; Buske-Kirschbaum, Jobst, Wustmans, Kirschbaum, Rauh & Hellhammer, 1997; Stroud, Foster, Papandonatos, Handwerker, Granger, Kivlighan & Niaura 2009) was administered. Participants first completed a 20-minute baseline period in which they watched G-rated movies and television shows, followed by three stressors. The stressors included a speech (5 minutes preparation plus a 5 minute speech), a mental arithmetic task (5 minutes), and a mirror-tracing task (5 minutes) performed in front of a “stern” two-member audience. Following the stress tasks, participants engaged in a 60 minute recovery period in which they watched G-rated movies and television shows, as well as completed questionnaires. During the TSST-C, saliva samples were collected from each participant by passive drool (Granger, Kivlighan, Fortunato, Harmon, Hibel, Schwartz & Whembolua 2007). In order to control for variability in cortisol levels due to conditions outside of the TSST-C, we asked study participants not to eat or drink 2 hours prior to the session, consume caffeine 24 hours prior, exercise 24 hours prior, or use alcohol or medications 48 hours prior. To optimize timing for assessment of cortisol response to the stress tasks, participant saliva was taken 15-20 minutes after each period of interest for a total of nine saliva samples (2 during the baseline period, 1 after the speech preparation to assess anticipatory stress, 1 after the speech, 1 after the mental arithmetic problems, 1 after the mirror-tracing task, and 3 throughout recovery period). After collection, the samples were frozen at –80 degrees Celsius until they were shipped on dry ice to the laboratory of Clemens Kirschbaum, Ph.D. (Dresden University of Technology), where cortisol measurements were assessed using a chemiluminescence-immuno-assay (CLIA) with high sensitivity (0.16 ng/ml; IBL, Hamburg, Germany). Both intra- and inter-assay coefficients for cortisol were below 8%.

Procedures

Of relevance to the current study, participants completed a “rest” session and a “stress” session. Written informed consent was obtained from all parents and written assent was obtained from all youth included in the study at the first laboratory visit. The purpose of the rest session was twofold: 1) to have participants habituate to the novelty of the laboratory and saliva sampling to reduce laboratory effects on cortisol collection; and 2) to collect self-reports of demographic information and the PBI. In the stress session, participants completed the TSST-C. Each session occurred between 14:00 and 17:00 to control for circadian cortisol variation, and lasted approximately 2 hours. Following the adapted TSST-C, participants were debriefed. The current study was approved by the Lifespan Institutional Review Board prior to data collection.

Analytic Plan

We used Mplus version 7 (Muthén & Muthén 2013) to test our models. To address issues of non-normally distributed data, robust full-information maximum likelihood estimation was used. To be able to test nuanced relationships between maternal parenting style and youth stress response we used multiphase growth curve modeling (Ram & Grimm 2007). Multiphase growth curve modeling facilitates the investigation of heterogeneity in response to stress by modeling three different phases of the response to stress: baseline levels of

cortisol, individual differences in the adrenocortical response to a stressor (reactivity), and individual differences in recovery from the stressor. Hence, multiphase growth curve modeling facilitates the investigation of HPA axis dysregulation by “dissecting” different facets of the individual's response to stress. This approach allowed us to simultaneously model the impact of intra-individual differences, as well as inter-individual differences, across these three phases. We created three latent factors from cortisol indicators corresponding to baseline, reactivity, and recovery phases. Before entering our hypothesized predictors and interactions, we assessed model fit by inspecting Root Mean Square Error Approximation (RMSEA), Tucker-Lewis Index (TLI), and Comparative Fit Index (CFI) (Little 2013).

We examined the independent, additive (covaried), and interactive associations between maternal parenting styles (care and overprotection) and stress response. First, we entered maternal care as a predictor of the latent baseline, the latent reactivity, and the latent recovery factors. Second, in a separate model we entered maternal overprotection as a predictor of the three stress responses phases (baseline, reactivity, and recovery). Third, we entered maternal care and overprotection as simultaneous predictions in the same model. This allowed us to examine the influence of maternal overprotection above and beyond any potential protective effect of maternal care. Fourth, we included the interaction between maternal care and overprotection on all three latent factors of stress response. For all models described above, youth puberty and gender were treated as covariates. Lastly, we examined the maternal care x puberty interaction and the maternal overprotection x puberty interaction while controlling for youth gender.

Results

Specifying the Stress Response Multiphase Growth Curve Model

For illustrative purposes, *Figure 1* shows the mean trajectory of cortisol levels across the administration of the TSST-C. This figure demonstrates that the observed data conforms to our a priori hypothesis that there are three distinct stress response phases: baseline, reactivity, and recovery. To test our hypotheses, several specifications were conducted when fitting the multiphase growth curve model. First, we modeled cortisol saliva samples 1 and 2 as the baseline phase, as these samples were collected before the stressors. Second, we modeled cortisol saliva samples 3, 4, 5, and 6 as the reactivity phase, as these samples corresponded to the experimental manipulation of stress. Finally, we modeled cortisol saliva samples 7, 8, and 9 as the recovery phase, as these samples corresponded to the cessation of the experimentally-induced stress (and theoretically expected decline in cortisol levels post-stressor). As shown in *Figure 2*, three latent factors corresponding to baseline, reactivity, and recovery phases were created. Our estimated model fit the data adequately ($\chi^2_{34, N=43} = 47.5, p < .05, RMSEA = .08, CFI = .97$, and $TFI = .97$). The baseline factor had a mean of 5.14 ($p < .01$) and a variance of 6.34 ($p < .01$); the reactivity factor had a mean of 3.49 ($p < .01$) and a variance of 33.17 ($p < .05$); and the recovery factor had a mean of -4.25 ($p < .01$), and a variance of 23.88 ($p < .01$). Thus, the factor means show that on average, cortisol levels increased from the baseline phase to the reactivity phase, and decreased from the reactivity phase to the recovery phase.

Maternal Parenting Styles

Maternal care ($M=35.3$, $SD=3.2$) and overprotection ($M=26.4$, $SD=5.9$) were moderately and negatively correlated ($r=-.44$, $p<.05$), suggesting that these constructs are inversely related and represent different parenting styles. For illustrative purposes, we presented the mean trajectory of cortisol levels across the stress session as a function of maternal parenting style (see *Figure 3*). We then conducted a series of models to examine if maternal parenting styles predicted individual differences in youth cortisol levels.

Independent Effect of Maternal Care on Youth Stress Response

After controlling for puberty and gender, maternal care was not significantly associated with baseline levels of cortisol ($\beta = -.01$, $p=.91$), but was significantly associated with a steeper rate of cortisol increase in response to stress ($\beta = .50$, $p=.03$), and faster decline in recovery ($\beta = -.52$, $p=.03$).

Independent Effect of Maternal Overprotection on Youth Stress Response

After controlling for puberty and gender, maternal overprotection was marginally associated with lower baseline levels of cortisol ($\beta = -.11$, $p=.07$), a significant slower rate of cortisol increase in response to stress ($\beta = -.27$, $p=.01$), and a significant slower decline in recovery ($\beta = .33$, $p=.00$).

Additive and Interactive Effects of Maternal Care and Overprotection on Youth Stress Response

After controlling for puberty and gender, maternal overprotection was significantly associated with lower baseline levels of cortisol ($\beta = -.14$, $p=.04$), a non-significant slower rate of cortisol increase in response to stress ($\beta = -.19$, $p=.12$), and a significant slower decline in recovery ($\beta = .27$, $p=.04$). No effects of maternal care emerged for baseline ($\beta = .13$, $p=.24$), reactivity ($\beta = .34$, $p=.25$), or recovery ($\beta = .29$, $p=.31$). We then examined the maternal care x overprotection interaction term as an additional predictor; however, this effect was not statistically significant (p -values ranged from .10 to .51).

Interactive Effects of Maternal Care and Overprotection X Puberty on Youth Stress Response

We tested the maternal care x puberty interaction, and the maternal overprotection x puberty interaction on the different phases of the stress response (i.e., baseline, reactivity, and recovery). All analyses were null, p -values ranged from .15 to .97.

Discussion

The aim of the current study was to provide a preliminary examination of the influence of maternal parenting style, specifically youth report of maternal care and overprotection, on youth acute stress response. Consistent with animal and human studies, we found evidence that specific aspects of maternal parenting style may exert an important influence on stress response amongst youth. When maternal care and overprotection were examined as independent predictors of youth stress response we found divergent effects of these two

maternal parenting styles; however, our additive model showed that only maternal overprotection had a unique effect on youth stress response. Below we outline the nature of our findings and the implications.

When examined independently maternal care was associated with both a higher cortisol reactivity to stress, and a quicker recovery among youth. Taking both cortisol reactivity and recovery effects into account, it appears that maternal care is related to rapid activation (i.e., steep increase in cortisol levels) and recovery (i.e., steep decrease in cortisol levels) of the HPA axis among youth. This acute stress response profile is arguably indicative of an adaptive stress response (de Kloet, Joëls & Holsboer 2005). Although, it does run counter to animal work that suggest that maternal care is related to decreased stress reactivity (for a review see Kaffman & Meaney 2007). These contradictory findings may be due to differences in the developmental phase examined. For example, animal work that has found the maternal care-to-decreased stress reactivity link has focused on the first week of a rat pup's life, which is roughly equivalent to a 2 month-old human infant (Sengupta, 2013); while the current sample consisted of children and adolescents (versus infants). On the other hand, maternal overprotection was marginally associated with lower baseline levels of cortisol, a low reactivity to stress, and slow recovery among youth. Thus, it appears that maternal overprotection is related to an underactive or blunted youth stress response. Interestingly, our data suggest that even though maternal overprotection is associated with blunted youth cortisol reactivity, youth struggle to return to baseline levels of cortisol after experiencing an acute stressor. It is important to note that our results differed by statistical approach. Our most rigorous model, which accounted for both maternal care and overprotection, as well as puberty and gender, indicated that reports of maternal overprotection, but not maternal care, were associated with an altered cortisol stress response in youth.

In the current study, we found evidence that maternal overprotection was uniquely associated with low baseline levels of cortisol, and difficulty recovering from an acute stressor, above and beyond the effects of maternal care, youth pubertal status, and youth gender. In other words, youth who perceived their mothers to be controlling, demanding, and intrusive (i.e., overprotective mothers) displayed difficulty in their ability to effectively regulate their physiological responses to stress. Since most prior studies focused on the mother-youth HPA axis regulation link do not include assessments of maternal overprotection, it is difficult to compare our findings to past research. However, the current study highlights the importance of examining the influence of multiple maternal parental styles when investigating youth stress response. Much of the literature on maladjustment (e.g., psychopathology) and HPA axis regulation has yielded mixed results showing that maladjustment is associated with low baseline cortisol levels, and both elevated and blunted stress responses (e.g., McBurnett, Lahey, Rathouz & Loeber 2000; Fairchild, van Goozen, Stollery, Brown, Gardiner, Herbert & Goodyer 2008; Jezova, Makatsori, Duncko, Moncek & Jakubek 2004; Burke, Davis, Otte & Mohr 2005, Lopez-Duran, Kovacs & George 2009). This mixed data may indicate that various factors, for example distinct maternal parenting styles, differentially influence the youth stress response.

Some data point to an association between both anxiety and externalizing problems and blunted responses to stress. For example, a previous study found that children with conduct disorder have lower baseline levels of cortisol (McBurnett, Lahey, Rathouz & Leober, 2000). Similarly, anxiety and externalizing behavior problems have been correlated with lower cortisol levels in analyses that combine reactivity and recovery phases (e.g., Fairchild, van Goozen, Stollery, Brown, Gardiner, Herbert & Goodyer 2008; Jezova, Makatsori, Duncko, Moncek & Jakubek 2004). In contrast, depression has been characterized by increased stress reactivity (increased levels of cortisol; Burke, Davis, Otte & Mohr 2005; Lopez-Duran, Kovacs & George 2009).

Some researchers have suggested that HPA axis dysregulation, specifically during childhood/adolescence, may mediate the relationship between parenting behaviors and youth psychopathology (Allwood, Handwerger, Kivlighan, Granger & Stroud 2011; Doom & Gunnar 2013; Heim, Newport, Mletzko, Miller & Nemeroff 2008; Snoek, Van Goozen, Matthys, Buitelaar & Van Engeland 2004). The current findings indicate that perhaps maternal overprotection may lead to blunted responses to stress, which could potentially increase risk for anxiety and externalizing problems. This is consistent with research that has found parental overprotection, but not care, to be associated with youth anxiety and externalizing problems (e.g., Hoeve, Dubas, Eischelsheim, Van der Laan, Smeenk & Gerris 2009; Rork & Morris 2009). However, future prospective studies that include youth with internalizing and externalizing disorders are needed to determine the direction of effects between parenting, stress response, and offspring psychopathology.

Contrary to our hypotheses, we did not observe an effect of maternal care on youth responses to stress (in our most rigorous model). This was surprising in light of past research that has demonstrated maternal care to be associated with adaptive outcomes (e.g., Albers, Riksen-Walraven, Sweep & de Weerth 2008) or the inverse (low maternal care associated with maladaptive outcomes; e.g., Engert, Efanov, Dedovic, Duchesne, Dagher & Pruessner 2010; Hackman, Betancourt, Brodsky, Kobrin, Hurt & Farah 2013; Hanson & Chen 2010; Zalewski, Lengua & Fisher 2012). Maternal care and overprotection are not mutually exclusive, and it is possible for a parent to display both types of behaviors (e.g., caring and attentive, but also highly vigilant and controlling). The degree to which a mother displays both care and overprotection could be differentially associated with youth outcomes. Based on this logic, we examined the interactive effect of maternal care and overprotection on an exploratory basis. However, no synergistic effects emerged. We acknowledge that there was likely not sufficient power to detect interactions; however, our first order effects suggest that, regardless of whether a child perceives their mother to display caring behaviors, an overprotective maternal parenting style may be associated with youth hypoactivation of the stress response. Based on these results, future studies may aim to investigate how different maternal practices influence heterogeneity in cortisol response to stress.

One reason we may not have found additive effects of maternal care on adolescent stress response may be due to the developmental clash between maternal overprotective behaviors (e.g., control and intrusiveness) and youth's expanding need for independence and privacy. In other words, maternal care may not have protective effects in the context of simultaneous maternal overprotective behaviors. It may be that maternal overprotection may hamper the

ability to appropriately navigate the increasing social demands of this developmental period. Another explanation may be that maternal support has diminished effectiveness in buffering HPA axis stress responses around the beginning of puberty. Indeed, a recent study found that the presence of a parent during an acute laboratory stressor provided “social buffering” by aiding a quick recovery (i.e., regulation of the HPA axis and return to baseline) in children, but not in adolescents (Doom, Hostinar, VanZomeren-Dohm & Gunnar 2015). Other studies have found similar effects (e.g., Hennessy, Kaiser, & Sachser, 2009; Hostinar, Johnson, & Gunner, 2014; Hostinar, Sullivan, Gunner, 2014).

We note several limitations to the current study. First, we used a small sample size leading to lower power to detect effects, especially for the interactions. Second, our sample consisted of participants who spanned a wide age range and pubertal development. It is possible that the degree to which parenting style influences youth stress response may differ by pubertal stage. We addressed this limitation by including puberty as a covariate in all analyses, as well as by examining the interactive effect of maternal care, overprotection, and puberty. However, we did not find that puberty moderated the effects of either maternal parenting style. Though, again, we acknowledge that due to our small sample size we may not have had sufficient power to examine our hypotheses stratified by puberty. Third, our sample consisted of primarily Caucasian participants, which prevents generalization of our findings to minority populations. Future studies need to investigate if similar mechanisms operate in diverse samples, and/or if cultural factors influence these associations. For example, it is possible that the level of parent acculturation to American culture may influence maternal parenting style or moderate the effect of parenting on youth stress response. Fourth, we assessed youth's perceptions of maternal parenting style versus utilizing behavioral observations of mother-youth interactions. While youth reports of maternal behaviors may provide useful information on maternal parenting style, this type of assessment is subject to reporter bias. On a related note, our study focused on maternal parenting styles and does not generalize to paternal styles. It will be important for future studies to examine the potential effects of fathers. Lastly, in addition to replicating the current findings in a larger sample and testing potential pubertal differences, it may be important for future studies to assess youth stress response to “real world” stressors (versus a laboratory stress) to examine the influence of parenting style to more ecologically valid experiences.

Despite these limitations, we highlight several strengths of the current investigation that expand on past research. First, we included assessment of both older children and adolescents, whereas the majority of prior research has focused on infancy/young childhood or adult retrospective accounts. Second, we investigated the potential additive and synergistic effects of maternal care and overprotection on youth stress response. Third, we examined the relationship between maternal parenting style and youth stress response unconfounded by current psychopathology. The current findings suggest that community parent education programs may benefit from teaching parents how to deter overprotective behaviors in order to shape more autonomous capabilities to respond to stress. Lastly, multiphase growth curve modeling was utilized to characterize individual variation in cortisol baseline, reactivity, and recovery, levels expanding on previous statistical models focusing on area under the curve or difference scores. Our more nuanced statistical approach

may more adequately “carve nature at the joints” of individual differences in adrenocortical stress response.

Conclusions

In sum, the current findings add to the body of literature suggesting that specific maternal parenting behaviors may impact the youth adrenocortical stress response (Tarullo & Gunnar 2006). Our results provide initial evidence that maternal overprotection may lead to blunted physiological reactivity and difficulty recovering from acute stress. Results warrant replication in a larger sample with a tighter pubertal range. Moreover, future studies are needed to investigate if the physiological stress response mediates the association between parenting behaviors and youth adjustment outcomes, such as psychopathology.

Acknowledgments

Declaration of Interest

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Figure 1.

Trajectory of cortisol response to the adapted TSST-C. The figure shows the mean trajectory of cortisol levels across the administration of the adapted TSST-C for the whole sample ($n = 43$). It demonstrates that there are three distinct stress response phases: baseline, reactivity (in gray), and recovery.

Figure 2.

Multiphase growth curve model of cortisol stress response to the adapted TSST-C.

Unstandardized loadings coefficients are presented, standardized correlations between latent constructs are presented, * = $p < .05$, ** = $p < .05$.

Figure 3.

Cortisol response to the adapted TSST-C as a function of maternal care and overprotection. Although continuous values were included in the statistical analyses, a median split on parental care and overprotection was used to categorize participants for visual display purposes. This figure demonstrates the average cortisol level across the stress session as function of the following groups: Low care (<36) and low overprotection (<27); Low care (<36) and high overprotection (>27); high care (>36) and low overprotection (<27); high care (>36) and high overprotection (>27).