Effortful control and parenting: Associations with HPA axis reactivity in early childhood

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Abstract

While activation of the hypothalamic-pituitary-adrenal (HPA) axis is an adaptive response to stress, excessive HPA axis reactivity may be an important marker of childhood vulnerability to psychopathology. Parenting, including parent affect during parent–child interactions, may play an important role in shaping the developing HPA system; however, the association of parent affect may be moderated by child factors, especially children’s emerging self-regulatory skills. We therefore tested the relationship between parent affectivity and 160 preschoolers’ cortisol reactivity during a laboratory visit, examining children’s effortful control (EC) as a moderator. Greater parent negative affectivity was related to greater initial and increasing cortisol over time, but only when children were low in EC. Higher parent positive affectivity was related to a higher baseline cortisol for children with low EC and lower baseline cortisol for children with high EC. Results indicate that children’s EC moderates the extent to which parent affect shapes stress reactive systems in early childhood.

Introduction

Cortisol reactivity to stress is an important marker of the hypothalamic-pituitary-adrenal (HPA) axis, a system that contributes to individual differences in vulnerabilities to psychopathology (Gunnar & Talge, 2008). The early environment appears to influence children’s cortisol reactivity (Gunnar & Donzella, 2002; Ouellet-Morin, Boivin, Dionne, Lupien, Arsenault, Barr, Pérusse & Tremblay, 2008; Saridjan, Huizink, Koetsier, Jaddoe, Mackenbach, Hofman, Kirschbaum, Verhulst & Tiemeier, 2010), thus potentially playing a causal role in elevating psychopathology risk via its influence on emerging individual differences in response to stress. In particular, available evidence suggests that parenting may be an especially critical factor (Blair, Granger, Kivlighan, Mills-Koonce, Willoughby, Greenberg, Hibell, Fortunato & the Family Life Project Investigators, 2008; Murray, Halligan, Goodyer & Herbert, 2010). For example, poor parental care has been linked to mal-adaptive cortisol functioning in animals (Coplan, Andrews, Rosenblum, Owens, Friedman, Gorman & Nemeroff, 1996), infants (Bugental, Martorell & Barraza, 2003) and preschool-aged children (Pendry & Adam, 2007).

With respect to more specific aspects of parenting, parent affect expressed during parent–child interaction may play an especially important role in children’s stress reactivity. Social referencing of parent affect by children is an adaptive behavior (Davies, 2011); for example, young children assess the safety of novel situations or persons in part by monitoring parental affective cues (Davies, 2011). Further, research indicates that children’s acceptance of a novel stranger or situation depends greatly on the affective tone that their parent conveys (Emde, 1992). Indeed, infants and young children...
frequently utilize both facial and vocal emotional cues from adults as a reference for how to respond to a variety of situations (Fernald, 1993; Camras & Sachs, 1991; Gunnar & Stone, 1984; Mumme, Fernald & Herrera, 1996; Walker-Andrews, 1997; Walker-Andrews & Lennon, 1991). Considered as a whole, this literature suggests that parental affect may serve as an important cue for the activation of children’s psychophysiological responses to novelty and stress.

With specific respect to cortisol, a small literature speaks to this notion, with positive parenting, including positive affective tone when parents interact with children, moderating the influence of negative life events on children’s cortisol reactivity (Barry & Kochanska, 2010; Bugental et al., 2003; Hagan, Roubinov, Gress-Smith, Luecken, Sandler & Wolchik, 2011; Pendry & Adam, 2007). This work suggests that parent affectivity may act in conjunction with other factors to shape children’s cortisol reactivity. In regard to parent negative affectivity, given its clear links to negative child outcomes (Carson & Parke, 1996; Kahen, Katz & Gottman, 1994), surprisingly few studies have examined its relationship to child cortisol reactivity, although measures of parenting that likely capture aspects of parental NA (e.g. hostility) are associated with children’s cortisol reactivity (Dougherty, Klein, Rose & Luptook, 2011). These findings, along with the larger literature implicating emotional tone during parent–child interaction as a critical element in child development and socialization (e.g. Eisenberg, Cumberland & Spinrad, 1998), suggest that parents’ affectivity with their children may be an important determinant of children’s early emerging cortisol reactivity. This is particularly important as research has demonstrated clear stability in observed parental affect, both negative and positive, expressed during parent–child interactions from ages 2 to 5 (Feng, Shaw, Skuban & Lane, 2007), a critical period for child development.

While very little research has examined potential moderators of caregiver affect on children’s cortisol reactivity, a large literature examining child outcomes more broadly defined shows that not all children exhibit the same degree of responsivity to early caregiving (Luthar, 2006; Masten, 2007). One factor that may play a role in moderating the effects of parenting on children’s cortisol reactivity is children’s self-regulatory abilities. More specifically, temperamental effortful control (EC) may play a key role in shaping risk for an array of negative outcomes (e.g. Belsky & Beaver, 2011; Carver, Johnson & Joorman, 2008). This is not surprising as the capacity to effectively regulate one’s emotions and behavior is critical to adaptive development (Gottfredson & Hirschi, 1990; Vazsonyi & Huang, 2010).

Psychopathologists have proposed that children’s emerging EC may moderate other forms of risk for psychopathology across development (e.g. Carver et al., 2008; Kochanska, Barry, Jimenez, Hollatz & Woodard, 2009; Martel & Nigg, 2006; Verstraeten, Vasey, Raes & Bijttebier, 2009). For example, Verstraeten and colleagues (2009) found that, in a sample of 304 youth followed from grade seven to ten, child negative affect was associated with a ruminative response style and, in turn, with depressive symptoms, but only for children with low EC. In addition, child EC has been shown to moderate the association between low levels of child guilt and later disruptive outcomes (Kochanska et al., 2009). This suggests the intriguing possibility that children’s EC might mitigate any risk for increased cortisol reactivity associated with parent affectivity, particularly negative affectivity. Main effects of EC on cortisol reactivity are also suggested by the literature, indicating that EC serves as an important protective factor by influencing children’s negative emotional reactivity to stressful stimuli (Compas, Connor-Smith & Jaser, 2004; Lengu, 2008; Spinrad, Eisenberg, Granger, Eggum, Sallquist, Haugen, Kupfer & Hofer, 2009).

With this literature in mind, we examined whether parental positive and negative affectivity during parent–child interactions was related to children’s cortisol reactivity during a standardized battery of stress-eliciting tasks. While previous work shows that early parenting is associated with children’s cortisol reactivity (Blair et al., 2008; Dougherty et al., 2011), considerable variability remains in children’s cortisol responses to stress, suggesting the possibility that child factors moderate the influence of parenting on children’s cortisol. As previous work highlights the role of children’s self-regulation as a marker of children’s responsivity to their early environments (Carver et al., 2008; Kochanska et al., 2009; Martel & Nigg, 2006; Verstraeten et al., 2009), we posited that children with low levels of EC would display especially heightened cortisol reactivity in the context of parental negative affect. Although EC has several facets, including attentional regulation, inhibitory control, and activational control, we chose to examine one widely studied and readily observed behavioral manifestation of this trait; namely, inhibitory control, or the ability to plan and suppress inappropriate approach responses (Rothbart, 1989). We elected to focus on this specific aspect of EC as it has been linked to an array of important child outcomes (Carlson & Moses, 2001; Kochanska & Aksan, 2006; Schachar, Mota, Logan, Tannock & Klim, 2000).

With respect to positive parent affect, our hypotheses were more exploratory; children’s self-regulation skills might be less critical in the context of positive parenting
behaviors. Hence, parent positive affect might be expected to show an association with reduced child cortisol that is unmoderated by children’s EC. However, we considered two additional, tentative hypotheses. First, children with high EC, by virtue of their more efficient self-regulatory skills, might be better positioned to reap the benefits of positive caregiving, thus showing especially low levels of some aspects of reactivity in the context of greater parent positive affect. In addition, children with low EC might benefit from a less stimulating caregiving environment with respect to parent affectivity; if so, this would suggest that even positive aspects of parental affect might be associated with elevated cortisol in these children.

We tested these questions in a sample of preschool-aged children and their primary caregivers, using standardized observational measures of children’s EC and parent affectivity during parent–child interactions. We chose to study preschool-aged children because children’s EC skills show large and meaningful individual differences at this age (Eisenberg, 2005; Kochanska, Murray & Harlan, 2000; Posner & Rothbart, 1998).

Method

Participants

Participants were 160 children and their primary caregivers who were part of a larger study of temperament in preschool-aged children. Participants from the larger project were recruited from a commercial mailing list. Participants in our subsample identified themselves as Caucasian (N = 150; 93.7%), African-American (N = 2; 1.2%), Asian (N = 1; .6%), and mixed or other race (N = 7; 5.1%); 10 participants identified themselves as Hispanic (6.2%). The majority of the families were middle class, as measured by Hollingshead’s Four Factor Index of Social Status (Hollingshead, 1975; M = 46.14; SD = 10.29). The mean age of parents was 36.5 years (SD = 3.8) for mothers and 38.7 years (SD = 4.6) for fathers. The mean age of child participants was 3.6 years (SD = .2), and 50.0% (N = 80) were female. The majority (77.9%) of the children came from two-parent homes, and 52.5% of the mothers worked outside the home part- or full-time; 16.7% worked more than 35 hours per week. Children were administered the Peabody Picture Vocabulary Test (PPVT; Dunn & Dunn, 1997) to screen for gross cognitive impairment and to assess task comprehension (M = 105.31, SD = 14.08, range = 61–139). In addition, the videos of children with lower scores (albeit greater than 60) were further reviewed by trained study personnel to ensure that they appeared to understand the tasks; no children were excluded based on this review.

Procedure

All children attended a laboratory session approximately 2.5 hours in duration, during which they were video-taped while participating with a female experimenter in 12 standardized tasks selected from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith, Reilly, Lemery, Longley & Prescott, 1995). Lab-TAB tasks are designed to elicit behavioral expressions of a broad range of emotional and other temperamental traits. Episodes were ordered so as to prevent carry-over effects in that no episodes presumed to evoke similar affective responses occurred consecutively, and each was followed by a brief play break. Two episodes were coded for EC and are described below. All other Lab-TAB episodes are described in detail elsewhere (Hayden, Klein, Sheikh, Olino, Dougherty, Dyson, Durbin & Singh, 2010).

Tower of patience

A female experimenter and child took turns building a tower using large cardboard blocks. The experimenter waited a series of increasing delays (5, 10, 15, 20, 30 s) before placing her block on the tower, thus forcing the child to wait increasingly longer periods of time before being given a turn. Two towers were built over the course of the task.

Snack delay

The experimenter placed a chocolate candy underneath a transparent cup, telling the child that (s)he must wait until the experimenter rang a bell before picking up the cup and eating the candy. The experimenter adhered to a series of delays of increasing length (5, 10, 20, 30 s), forcing the child to wait longer each time to eat the candy.

As an index of EC, each task was coded for prompting behavior to get the experimenter to place their block or ring the bell (e.g. verbally prompting, ‘ring it’ or ‘it’s your turn’, or physically prompting, pointing at the block or bell; see Carlson, 2005; Kochanska, Murray, Jacques, Koenig & Vandengeest, 1996; Kochanska & Knaack, 2003, for similar procedures). Prompts were averaged across each delay and then across tasks to create an aggregate EC scale. As more prompting reflects lower EC, scores were reverse-coded to ease interpretation. Coders were unaware of children’s parenting and
cortisol reactivity. Raters had to reach at least 80% agreement with a ‘master’ rater before coding independently. To examine interrater reliability, eight videotapes were independently coded by a second rater (ICC = .98).

Cortisol sampling procedure

Salivary cortisol was collected four times during the laboratory assessment. Saliva for cortisol assay was obtained by having the children dip 2-inch-long cotton dental rolls into small cups containing approximately .025 g of sugar-sweetened cherry Kool-Aid® drink mix. Children were then instructed to chew the cotton rolls until they were saturated with saliva. Previous work shows that the use of Kool-Aid® does not compromise the quality of the assays when used sparingly as it does not significantly alter the pH of the saliva (Talge, Donzella, Kryzer, Gierens & Gunnar, 2005). In addition, its use promotes saliva flow and makes the sampling pleasant for the child. The collection of each sample took approximately 1–2 minutes. After each sample was collected, the saliva was expelled from the cotton roll into a labeled micro tube, and stored at −20°C until assayed.

The timing at which the laboratory cortisol samples were obtained was determined based on the presumed stress of the Lab-TAB episode and on previous studies using a similar paradigm with children close to this age (Luby, Heffelfinger, Mrakotsky, Brown, Hessler & Spitznagel, 2003). The principle that cortisol levels are believed to reflect the level of stress experienced about 20–40 minutes prior (Dickerson & Kemeny, 2004) was also taken into account. Based on these considerations, the first sample was taken upon arrival at the laboratory after parents in the study had completed informed consent for both themselves and their child. Cortisol levels at the time of this sample reflect levels prior to the assessment, when the child was with a parent either at home or en route to the laboratory. While this was not hypothesized to be a particularly stressful time, such samples may still have relevance for children’s adaptive development as they may reflect ‘resting’ state cortisol levels (Gunnar & Talge, 2008), and will therefore be examined as a dependent variable in analyses. This sample will subsequently be referred to as baseline. The second sample was collected 30 minutes following the Stranger Approach task of the Lab-TAB, the most stressful episode in the battery, during which the child was separated from his/her parent and a stranger entered the room. The third salivary cortisol sample was taken 30 minutes after Transparent Box, a frustration-inducing task in which the child is unable to unlock a box with a desirable toy inside. The fourth and final sample was collected 20 minutes after the completion of all Lab-TAB tasks. The second, third, and fourth samples reflect cortisol reactivity to the tasks and therefore will be referred to as cortisol reactivity. To control for non-stress related elevations of cortisol, laboratory assessments were conducted at either 10 am (69% of the assessments) or 2 pm. Families were instructed prior to coming to the laboratory that the child should not eat within one hour before their scheduled lab visit, and that children should avoid caffeine for at least two hours, and dairy products for at least 15 minutes, prior to arrival.

Samples were assayed using a time-resolved fluorescence immunoassay with fluorometric end point detection (DELFIA). All samples were assayed in duplicate. Samples yielding values above 44 nanomoles per liter (nmol/L) were excluded, which applied to four laboratory samples from four different individuals. The correlation between duplicates was .99. The inter- and intra-assay coefficients of variation (CV) were between 7.1% and 9.0% and 4.0% and 6.7%, respectively. As is typically found (Gunnar & Talge, 2008), the cortisol values in this sample were positively skewed. A log10 transformation of the raw cortisol values yielded unskewed cortisol values that were used in all analyses.

Parent affectivity

One hundred and forty-nine parent-child dyads completed an observational measure of parent–child interactions for preschool-aged children during a second laboratory visit approximately two weeks later. We used observational measures of parenting as these have been shown to have strong predictive validity for child outcomes (Zaslow, Weinfelder, Gallagher, Hair, Ogawa, Egeland, Tabors & De Temple, 2006). Children and a caregiver (typically the mother; N = 143; 96.0%) engaged in six tasks derived from the Teaching Tasks battery (Egeland, Weinfelder, Hiester, Lawrence, Pierce & Chipendale, 1995), which included book reading, block building, naming objects with wheels, matching shapes, completing a maze using an etch-sketch, and gift presentation. The battery (approximately 25 minutes in duration) was videotaped for subsequent coding on a number of parenting variables. The present study used two scales based on Weinfeld, Egeland and Ogawa (1998) that reflect parent affectivity during interaction with their child. The parental positive affectivity scale (range = 1–3) was based on the parent’s expression of positive regard and emotional support to the child and on general positive affectivity expressed during the battery (e.g. smiling, laughing, speaking in a pleasant tone). Higher scores indicate greater positive affectivity.
expressed throughout interaction with the child. The parental negative affectivity scale (range = 1–3) was based on the parent’s expression of negative regard and punitive behavior toward the child and on general negative affectivity (e.g. facial, bodily, and vocal expressions of anger, frustration, and or annoyance). Higher scores indicate greater negative affectivity expressed throughout interaction with the child. These two scales were significantly inversely correlated ($r = -0.35$, $p < .01$).

## Results

Table 1 presents correlations between mean cortisol levels at each sampling time and all major study variables. Cortisol levels at all four time points were highly positively correlated ($r_s = .29–.83$). Child EC scores were significantly associated with greater cortisol levels at the first and second samples post-stress. Parent negative affectivity expressed during the parent–child interaction task was also positively correlated with children’s cortisol reactivity levels. Child sex was negatively correlated with final cortisol levels, indicating that girls tended to have lower final cortisol levels than boys. Time of day was negatively correlated with baseline cortisol and the third sample taken post-stress, suggesting that children tested in the morning trended toward higher cortisol levels for these samples. Time of day was positively correlated with parent negative affectivity, Hollingshead scores, and child sex, indicating that children who were tested in the afternoon had parents who displayed more negative affectivity in interactions with them, were of a higher socioeconomic status, and were more likely to be female.

### Cortisol level comparisons across sample

As reported in Dougherty, Klein, Congdon, Canli and Hayden (2010), there was an observed decrease in child cortisol from baseline to the first sample taken after the beginning of the Lab-TAB. While counterintuitive, it is important to note that this pattern is frequently found in laboratory studies of cortisol reactivity in children (Gotlib, Joorman, Minor & Cooney, 2006; Luby et al., 2003; Talge, Bruce, Donzella & Gunnar, 2003), and may reflect stress-related increases related to anticipating the laboratory visit (Gunnar & Talge, 2008), such that the baseline sample is elevated and then declines. As evidenced by the significant positive quadratic effect (see Figure 1 for average trajectory of log 10 transformed data), average cortisol levels then began to increase steadily across the remaining two samples.

### Table 1 Correlations among variables

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<th>Table 1 Correlations among variables</th>
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<td>10. Child Sex in Months</td>
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Note: Cortisol levels are measured in nanomoles per liter (nmol/L). Child Sex: Male = 1 and Female = 2. Time of Day: am = 0 and pm = 1.* $p < .05$, **$p < .01$. 

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Examination of cortisol trajectories

To examine overall cortisol trajectories for the sample and predictors of individual differences in these trajectories, we used multi-level modeling (MLM) conducted with HLM 6 (Scientific Software International Inc., IL). MLM has many advantages, including the ability to model data at two levels (Level 1, describing within-individual change over time; and Level 2, relating predictors to any interindividual differences in change), and the ability to account for missing values at Level 1 (Singer & Willett, 2003). For the Level 1 model, cortisol time points (baseline, first, second and third reactivity samples) were nested in the Level 2 variable, participant. Log10 transformed cortisol values were the dependent variable. Because cortisol levels show a diurnal pattern of variation, time of assessment (i.e. morning versus afternoon) was controlled for in all analyses.

Between-subjects predictors of individual change were modeled to allow examination of cortisol levels at each sampling time for each individual, while taking into account between-persons predictors. For these analyses, Level 2 predictors were time of day, child EC, and parent negative and positive affectivity during the parent–child interaction task. As study hypotheses focused on whether child EC moderated associations between parenting and child cortisol reactivity, two-way interactions between the relevant between-subject variables were the focus of analyses. Time was anchored at baseline (time = 0) so that the cortisol intercepts (β00j) would reflect the average of individuals’ cortisol levels at baseline. All Level 2 between-person variables were centered at their grand mean. MLM is equipped to handle missing data at Level 1 by estimating the trajectory based on existing data for that participant (two children were missing a baseline cortisol sample and two children were missing the final sample). At Level 2, parenting data were missing for 11 participants, and EC data were missing for one participant. These participants were excluded from analyses. The analytic sample did not differ from the full sample with regard to cortisol reactivity or basic demographic characteristics. Descriptive statistics for the full sample are reported throughout.

A quadratic equation was built to examine the effects of Level 2 variables on the intercept, instantaneous rate of change (henceforth referred to as slope), and curvature. As reported by Dougherty and colleagues (2010), significant variation in intercept, slope, and curvature was found, confirming the appropriateness of testing Level 2 predictors. As evidence that a quadratic model best fit the data, a chi-square test of the deviance statistics between unconditional linear and quadratic models indicated that adding a quadratic term to the model resulted in a significant improvement in model fit, supporting results from a graphical representation indicating that most children showed an initial decrease in cortisol from baseline followed by an increase (see Figure 1; X²(1) = 36.03, p < .001).

To evaluate the model, the following function was specified to describe the data from each individual:

\[
\text{Level 1: } Y_{ij} = \beta_{0j} + \beta_{1j}(\text{Time}) + \beta_{2j}(\text{Time}^2) + r_{ij}
\]

\[
\text{Level 2: }
\begin{align*}
\text{Intercept: } & \beta_{0j} = \gamma_{00} + u_{0j} \\
\text{Instantaneous Rate of Change: } & \beta_{1j} = \gamma_{10} + u_{1j} \\
\text{Curvature: } & \beta_{2j} = \gamma_{20} + u_{2j}
\end{align*}
\]

(Equation 1)

Main effects and interactions

To examine the main effects and two-way interactions of Level 2 predictors on the intercept, slope, and curvature for individuals’ cortisol trajectories, interaction terms were created by centering continuous variables, then multiplying the terms to reflect the product of Level 2 predictors of interest (Aiken & West, 1991). The grand mean centered Level 2 predictor variables were then entered into the quadratic growth model followed by the two-way interaction terms (see Table 2). All demographic predictors (child sex, PPVT scores, child age, time of day, and Hollingshead scores) were included in the initial model and those not significantly related to child cortisol intercept, slope, or curvature were removed.
to conserve power. Utilizing this process, only time of day was retained in the final models.

Main effects of time of day, parent negative affect, and EC were found in predicting cortisol intercept, slope, and curvature. Time of day was significantly associated with a lower cortisol intercept, suggesting that children tested in the afternoon had lower cortisol levels at the first assessment. The main effects of child EC and parent negative affectivity on cortisol were qualified by a significant interaction between these two variables in predicting cortisol slope and curvature, while EC and parent positive affectivity interacted to predict child cortisol intercepts only. To interpret the interactions between parent affectivity and EC, EC scores were centered at values one standard deviation above and below the mean so that model coefficients would reflect the effects of parent negative and positive affectivity when child EC was at high and low levels (see Figures 2 and 3). For children with high EC, greater parent positive affectivity was associated with a significantly lower cortisol intercept (intercept: unstandardized coefficient \( b = -0.208, SE = 0.085, t = -2.426, p = .017 \)) compared to children with high EC and low parent positive affectivity. For children with low EC, high parent positive affectivity was associated with a significantly higher cortisol intercept (intercept: unstandardized coefficient \( b = 0.423, SE = 0.183, t = 2.301, p = .023 \)) compared to children with low EC and low parent positive affectivity.

With respect to parent negative affectivity, it was associated with a significantly steeper cortisol slope, and slower rate of curvature when children were low in EC (slope: unstandardized coefficient \( b = 0.605, SE = 0.146, t = 4.146, p < .001 \); curvature: unstandardized coefficient \( b = -0.186, SE = 0.043, t = -4.290, p < .001 \)). For children high in EC, parent negative affectivity was not significantly associated with any aspect of cortisol (ps > .26).

To further examine differences among cortisol levels at each time point for the EC-parent NA interaction, we re-centered our Level 1 predictor time at each sample post-stress to examine interaction effects at the level of the intercept. \(^1\) Consistent with the pattern suggested in Figure 2, these analyses showed a significant EC–parent NA interaction in predicting cortisol intercept at the second post-stress sample (\( p = .03 \)). The interaction was at a trend level for intercept and the first post-stress sample (\( p = .06 \)).

**Discussion**

While early parenting is associated with children’s cortisol reactivity (Blair et al., 2008; Dougherty et al., 2011), variability in children’s cortisol responses to stress

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**Table 2**  
**HLM model effects**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient Intercept (SE)</th>
<th>t-value</th>
<th>Cortisol Instantaneous Rate of Change (SD)</th>
<th>t-value</th>
<th>Cortisol Curvature (SD)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time of Day</td>
<td>-1.11 (-.053)</td>
<td>.210*</td>
<td>.062 (.055)</td>
<td>1.12</td>
<td>-.023 (.016)</td>
<td>-1.432</td>
</tr>
<tr>
<td>Parent Negative Affectivity</td>
<td>.214 (.278)</td>
<td>.767</td>
<td>.397 (.126)</td>
<td>3.144**</td>
<td>-.128 (.044)</td>
<td>-2.942**</td>
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<tr>
<td>Parent Positive Affectivity</td>
<td>.108 (.086)</td>
<td>1.257</td>
<td>.092 (.082)</td>
<td>1.112</td>
<td>-.029 (.023)</td>
<td>-1.256</td>
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<tr>
<td>Child Effortful Control (EC)</td>
<td>-.036 (.037)</td>
<td>-.981</td>
<td>-.093 (.040)</td>
<td>-2.363*</td>
<td>.036 (.013)</td>
<td>2.871**</td>
</tr>
<tr>
<td>Child Effortful Control (EC) \times Parent Negative Affectivity</td>
<td>-.225 (.289)</td>
<td>-.779</td>
<td>-.291 (.130)</td>
<td>-2.232*</td>
<td>.080 (.044)</td>
<td>1.857</td>
</tr>
<tr>
<td>Child Effortful Control (EC) \times Parent Positive Affectivity</td>
<td>-.440 (.161)</td>
<td>-2.741**</td>
<td>.177 (.160)</td>
<td>1.108</td>
<td>-.028 (.042)</td>
<td>-6.61</td>
</tr>
</tbody>
</table>

Time of Day: am = 0 and pm = 1 * \( p < .05 \); ** \( p < .01 \); † \( p < .10 \).

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**Figure 2**  
Cortisol as a function of child EC and parent negative affectivity. Note: parent affectivity 1 SD above and below the mean; EC = Effortful Control; NA = Negative Affectivity.

\(^1\) As the parent positive affectivity–child EC interaction was significant for the intercept only, we did not conduct similar additional analyses for that model.
suggests that child factors may moderate the influence of parenting on children’s cortisol. Children’s EC may be a marker of their responsivity to the early environments (Kochanska et al., 2009; Martel & Nigg, 2006; Verstraeten et al., 2009). Consistent with this idea, we found that the effects of parent positive and negative affectivity predicted cortisol reactivity differently depending on children’s EC.

More specifically, we found that children lower in this trait showed a significantly steeper cortisol slope and slower rate of quadratic curvature when parent negative affectivity expressed during parent–child interactions was high. In other words, as hypothesized, children with lower EC showed a relatively pronounced cortisol response to laboratory stressors when their parents showed more negative emotions during parent–child interaction. Previous research has reported main effects of negative parenting behavior (Azar, Paquette, Zoccolillo, Baltzer & Tremblay, 2007) and low levels of EC (Oldehinkel, Hartman, Nederhof, Riese & Ormel, 2011) on children’s psychophysiological reactivity; however, our findings suggest further complexity in the relationship between parenting and child characteristics in predicting child cortisol function, and indicate that high parental negativity may exert the most profound impact when children have relatively low self-regulation. Also consistent with our hypotheses, greater cortisol reactivity in the context of high parent negative affectivity was not found for children with high EC, raising the possibility that greater self-regulation skills mitigate the effects of parental expressions of negative emotions on children’s cortisol. Such a process might be one means through which EC promotes adaptive child development (Gottfredson & Hirschi, 1990; Vazsonyi & Huang, 2010). This finding is also consistent with recent research demonstrating that EC moderates other risk factors across development (e.g. Carver et al., 2008; Kochanska et al., 2009; Martel & Nigg, 2006; Verstraeten et al., 2009).

A significant interaction was also found between parent positive affectivity and child EC such that for children with higher EC, greater parent positive affectivity was associated with lower baseline cortisol levels. Given the nature of the timing of collection of this sample, any effects must be interpreted with caution. However, this effect could mean that greater EC increases the extent to which children can derive benefits from parents’ expressions of positive affect. More specifically, children with high EC may have a greater capacity to internalize parental warmth expressed during interaction, resulting in more efficient regulation of stress coping. In contrast, for children with lower EC, greater parent positive affectivity was associated with a higher baseline cortisol levels. Baseline cortisol levels can be interpreted as a reflection of ‘resting state’ cortisol activity (Gunnar & Talge, 2008). Alternatively, research has demonstrated that children’s cortisol samples obtained at arrival at a novel laboratory setting are elevated relative to samples obtained at home at the same time of day as a lab visit, which suggests that initial lab samples may reflect a response to novelty (Gunnar & Talge, 2008). Regardless, both heightened baseline cortisol and exaggerated reactivity to a relatively benign stressor, such as arrival at the lab, may be indicative of risk (Gunnar & Talge, 2008; Halligan, Herbert, Goodyer & Murray, 2004). Our results suggest that lower parental emotional expression in general is associated low cortisol levels in children with low EC either at baseline (positive parent affect) or following stress (negative parent affect). Future research assessing cortisol reactivity using procedures to obtain more interpretable measures of baseline cortisol (e.g. Kryski, Smith, Sheikh, Singh & Hayden, 2011) will be needed to better explore this possibility.

Our study had several strengths, including observational measures of parenting (Zaslow et al., 2006) which have good predictive validity for important child outcomes, and objective measures of child EC based on a coding scheme that showed good interrater reliability.

However, a number of limitations should be acknowledged. While we focused on EC as a moderator of parental influences on cortisol reactivity, other temperament traits may also moderate responses to environmental factors (e.g. positive emotionality; Wichers, Kenis, Jacobs, Myin-Germeys, Schruers, Mengelers, Delespaul, Derom, Vlietinck & van Os, 2008). In addition, we examined only a narrow-band facet of EC (Rothbart & Bates, 2006) reflecting inhibitory control, despite the fact that other facets of this trait may also moderate contextual influences on cortisol reactivity. Replication of our

Figure 3 Cortisol as a function of child EC and parent positive affectivity. Note: parent affectivity 1 SD above and below the mean; EC = Effortful Control; PA = Positive Affectivity.
findings in larger samples and with other EC measures is important to better understand the complexity of these interactions. Due to the cross-sectional nature of this study we are unable to claim directionality in our models. For example, it is possible that child cortisol reactivity may shape both EC development and parenting practices. Longitudinal studies are necessary to disentangle the direction of this relationship.

Another possible limitation of this study is the way in which cortisol reactivity was indexed. Variability in the time at which samples were collected (Dickerson & Kemeny, 2004) and child reactivity to the novelty of the laboratory testing conditions (Gunnar & Talge, 2008) may have reduced the accuracy with which children’s cortisol reactivity was assessed. For example, we found an average decrease in child cortisol from baseline to second sample. However, it is important to note that such a pattern is quite frequently reported in the literature (Gotlib et al., 2006; Luby et al., 2003; Talge et al., 2003); indeed, the majority of developmental studies of cortisol reactivity in children fail to find an increase in children’s cortisol in response to laboratory stressors (Gunnar, Talge & Herrera, 2009). It has been posited that this may reflect developmental differences in how children’s HPA axis systems respond to stress, and that such hyporesponsivity may be the normative response to stress in early to middle childhood. Conversely, this pattern may also reflect methodological issues, such as children’s heightened reactivity to novel laboratory settings and procedures (Kryski et al., 2011). As cortisol reactivity and contextual factors that elicit it change over the course of development, studies exploring these relationships across development are warranted. Lastly, our sample was primarily Caucasian and middle class. While it is unclear what, if any, influence this had on the findings obtained, future work exploring these relationships in a more diverse sample is recommended as these findings cannot be generalized beyond the scope of a primarily Caucasian and middle-class sample.

Overall, the results of this study indicate that child EC influences how parenting shapes children’s cortisol responses to stress. In order to more fully understand the development of psychophysiological indices of stress sensitivity, developing and testing models that include both intrinsic child factors and contextual variables is likely critical.

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References
