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Predicting Developmental Changes in Internalizing Symptoms: Examining the Interplay Between Parenting and Neuroendocrine Stress Reactivity

ABSTRACT: In this study, we examined whether parenting and HPA-axis reactivity during middle childhood predicted increases in internalizing symptoms during the transition to adolescence, and whether HPA-axis reactivity mediated the impact of parenting on internalizing symptoms. The study included 65 children (35 boys) who were assessed at age 5, 7, and 11. Parenting behaviors were assessed via parent report at age 5 and 11. The child's HPA-axis reactivity was measured at age 7 via a stress task. Internalizing symptoms were measured via teacher reports at age 5 and 11. High maternal warmth at age 5 predicted lower internalizing symptoms at age 11. Also, high reported maternal warmth and induction predicted lower HPA-axis reactivity. Additionally, greater HPA-axis reactivity at age 7 was associated with greater increases in internalizing symptoms from age 5 to 11. Finally, the association between age 5 maternal warmth and age 11 internalizing symptoms was partially mediated by lower cortisol in response to the stress task. Thus, parenting behaviors in early development may influence the physiological stress response system and therefore buffer the development of internalizing symptoms during preadolescence when risk for disorder onset is high. © 2013 Wiley Periodicals, Inc. *Dev Psychobiol* 56: 908–923, 2014.

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Elevated internalizing symptoms, including depressed mood, social withdrawal and worrying, during the transition to adolescence have been associated with negative outcomes such as increased risk for mood disorders (Kovacs & Lopez-Duran, 2010), poor social

functioning (Pope & Bierman, 1999), and poor academic performance (Hughes & Coplan, 2010). Therefore, during the last several decades, researchers have identified a number of factors that increase or mitigate the development of internalizing symptoms during this period. Among these, parenting behaviors, such as discipline practices and parental warmth, have been the focus of much attention (e.g., Bender et al., 2007; Laskey & Cartwright-Hatton, 2009), and several psychosocial mechanisms have been identified to explain how parenting may impact internalizing symptoms (e.g., Berkien, Louwerson, Verhulst, & van der Ende, 2012). Yet, less is known about potential biological mechanisms at play. In this longitudinal study, we examined whether parenting behaviors at age 5 influenced

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changes in internalizing symptoms in preadolescence (age 11) through their impact on the child's physiological stress response system, the hypothalamic–pituitary–adrenal (HPA)-axis.

Parenting behaviors can play detrimental or protective roles in the development of internalizing symptoms. For example, harsh parenting (e.g., physical punishment) has emerged as a robust predictor and risk factor for high internalizing symptoms, both concurrently (Laskey & Cartwright-Hatton, 2009) and longitudinally (Leve, Kim, & Pears, 2005). This is not surprising given that harsh parenting has been linked to internalizing symptoms during multiple developmental stages (Bender et al., 2007; Bugental, Martorell, & Barraza, 2003). Among protective behaviors, nurturing/responsive parenting (e.g., parental warmth) has been linked concurrently to lower internalizing symptoms in early childhood (e.g., Bayer, Sanson, & Hemphill, 2006), the transition to adolescence (Roelofs, Meesters, ter Huurne, Bamelis, & Muris, 2006), and adolescence (Muris, Meesters, & van den Berg, 2003). Likewise, inductive discipline—a component of “effective parenting” that involves providing age-appropriate explanations for rules (i.e., reasoning) and nonaggressive redirection (Grusec & Goodnow, 1994) has been identified as a parenting characteristic that protects against internalizing symptoms (Conger & Conger, 2002).

Several psychosocial explanations for the effect of parenting on internalizing symptoms have been proposed. Harsh parenting may facilitate the development of internalizing problems by creating an unpredictable and potentially unsafe environment, where a heightened state of physiological arousal encourages the persistence of negative emotional states with little opportunity for regulation (Sturge-Apple, Davies, Martin, Cicchetti, & Hentges, 2012). In contrast, positive parenting behaviors may promote emotion regulation and stress coping strategies, thus helping the child to learn adaptive regulation skills and reducing risk for adolescent internalizing disorders (Silk et al., 2007). These behaviors are also protective against the effects of parent psychopathology (Leckman-Westin, Cohen, & Stueve, 2009) and exposure to violence (Howell, Graham-Bermann, Czyz, & Lilly, 2010) suggesting that they may reduce risk for psychopathology by mitigating the effect of stress during key developmental transitions.

Given the potential of parenting behaviors to increase or mitigate the effects of stress, parental impact on the child's physiological stress response system, the HPA-axis, is a likely biological mechanism through which parenting exerts a detrimental or protective influence. Not surprisingly, anomalies in HPA-axis stress functioning (i.e., stress reactivity and diurnal

functioning) have been associated with both internalizing symptoms (Smider et al., 2002; Turner-Cobb, Rixon, & Jessop, 2008), and depressive disorders (Guerry & Hastings, 2011; Lopez-Duran, Kovacs, & George, 2009; Rao, Hammen, & Poland, 2010). Likewise, HPA-axis functioning is highly influenced by parenting behaviors during childhood (Gunnar & Donzella, 2002). For example, exposure to harsh parenting, maltreatment and parental withdrawal has been linked to long-term anomalies in diurnal HPA-axis functioning during childhood (Essex, Klein, Cho, & Kalin, 2002; Hessl et al., 1998; Shea, Walsh, MacMillan, & Steiner, 2005) as well as adolescence (Murray, Halligan, Goodyer, & Herbert, 2010; Roisman et al., 2009). The few studies that have examined the association between harsh parenting and HPA-axis reactivity to acute stress have been mostly limited to infants and young children. For example, mothers who engaged in frequent spanking had infants who demonstrated greater reactivity to separation (Bugental et al., 2003). This is consistent with experiments conducted with animals, where early stress in the form of maternal separation or neglectful rearing results in HPA-axis hyper-reactivity (Ladd et al., 1999; Sanchez, 2006). While infancy may be a particularly sensitive period for the development of the HPA-axis, there is evidence to suggest that contextual and environmental factors continue to impact HPA-axis functioning throughout childhood and into adulthood (e.g., Elzinga et al., 2008). For example, a recent finding suggests that intrusive and controlling parenting during the preschool years is related to elevated tonic cortisol at age 6 (Taylor et al., 2012).

Studies have also identified parenting behaviors that may have a positive impact on HPA-axis functioning. For example, bereaved adolescents were more likely to have lower cortisol reactivity to acute stress if exposed to positive parenting such as effective discipline and high warmth (Hagan et al., 2011). Similar findings have been noted in younger children, where supportive and warm parenting are linked to lower, more adaptive, reactivity to social stressors (Bugental, 2004; Ellenbogen & Hodgins, 2009; Kertes et al., 2009). These findings are also consistent with animal studies suggesting that parental care behaviors (e.g., licking and grooming) facilitate adaptive HPA-axis development (Suchecki, Rosenfeld, & Levine, 1993) as well as mitigate the negative impact of stress on HPA-axis functioning (Walker, 2010).

All in all, the existing data suggest that parenting behaviors can impact HPA-axis functioning and child internalizing symptoms, and that HPA-axis dysfunction is associated with internalizing symptoms. Yet, we found no longitudinal studies examining whether HPA-axis

functioning is a mechanism by which parenting impacts the developmental trajectory of internalizing symptoms. To that end, in this longitudinal study, we clarify the interplay between parenting and HPA-axis functioning as contributors to the development of internalizing symptoms. First, we examine whether parenting behaviors (age 5) and HPA-axis reactivity (age 7) predict increases in internalizing symptoms during preadolescence (age 11). We hypothesize that increases in internalizing symptoms will be predicted by the presence of harsh punishment, lower parental warmth, less inductive discipline, and greater HPA-axis stress reactivity. Second, we examine whether the effects of parenting at age 5 on the development of internalizing symptoms 6 years later are mediated by HPA-axis reactivity. We hypothesize that the presence of harsh punishment, lower parental warmth and low inductive discipline impact internalizing symptoms through their negative impact on HPA-axis reactivity.

METHODS

Participants

Participants for this study included 65 children (35 boys) representing a subsample of families participating in a larger longitudinal study of the development of externalizing problems in childhood ($N=220$; Olson, Sameroff, Kerr, Lopez, & Wellman, 2005). The children participating in the original study were recruited in early childhood (age 3–4) and at the time represented the full range of externalizing and internalizing symptom severity on the Child Behavior Checklist/2–3 (Achenbach, 1992). These participants were originally recruited through newspaper and community advertisements as well as referrals from preschool teachers and pediatricians. Children with chronic medical conditions, mental retardation, or pervasive developmental disorders were excluded from the study. Families were paid for their participation and were representative of the local community including African-American (5.5%), Hispanic American (2.5%), and Asian American families (1%). The majority (87.9%) of the children resided in two-parent families. The median annual family income was \$52,000, ranging from \$20,000 to over \$100,000.

For the current study, 203 of the available original families were contacted by phone or postal mail and were invited to participate in an *additional* study examining HPA-axis reactivity in children. Seventy-eight families agreed to participate in the additional study. These 78 families did not differ from the original sample in age (in months; subsample: $M=63.2$; $SD=2.87$ vs. full sample: $M=63.5$; $SD=2.61$; $p=.52$), family income level (subsample: $M=9.72$; $SD=2.65$ vs. full sample: $M=9.18$; $SD=3.16$; $p=.211$), mother's education (subsample: $M=6.23$; $SD=.72$ vs. full sample: $M=6.11$; $SD=.90$; $p=.32$), age 5 teacher reported internalizing behavior problems (subsample: $M=2.09$; $SD=2.99$ vs. full sample: $M=2.71$; $SD=4.17$; $p=.29$), age 5

teacher reported externalizing behavior problems (subsample: $M=4.48$; $SD=7.00$ vs. full sample: $M=4.34$; $SD=8.65$; $p=.91$), age 5 mother reported warmth (z -scores; subsample: $M=-.218$; $SD=1.81$ vs. full sample: $M=.109$; $SD=1.64$; $p=.19$), induction (z -scores; subsample: $M=-.113$; $SD=1.77$ vs. full sample: $M=.06$; $SD=1.93$; $p=.53$), or physical punishment (subsample: $M=.167$; $SD=.249$ vs. full sample: $M=.150$; $SD=.261$; $p=.66$). Eight of these 78 participants were used as validity check controls to evaluate the experimental protocol (Lopez-Duran, Hajal, Olson, Felt, & Vazquez, 2009) and 5 did not complete all the necessary tasks to be included; therefore, the final sample included in the present analyses consisted of 65 children.

Data from this study include information collected across three time points. Measures of parenting and internalizing symptoms were completed at age 5 ($M=5.79$ years, $SD=.3$). Between 1 and 2 years later, the participants completed a task designed to measure HPA-axis reactivity (age 7; $M=7.48$ years, $SD=.7$). Finally, measures of internalizing symptoms were completed in preadolescence (age 11; $M=10.5$ years, $SD=.46$).

Measures

Internalizing Problems. Child internalizing symptoms were measured at age 5 and 11 via the global internalizing scale of the Teacher's Report Form-6-18 (TRF; Achenbach, 1991). The TRF is a 114-item teacher-reported measure of internalizing (e.g., sadness, withdrawal) and externalizing (e.g., aggression) behaviors. Informants were asked to report on a scale of 0–2, whether the given behavior is "not true," "sometimes true," "often or very true" of their child. Some examples of internalizing items are "Would rather be alone than with others" (social withdrawal), and "Cries a lot" (depression). The TRF internalizing scale displays high internal consistency within this sample (age 5 Cronbach's $\alpha=.81$; age 11 Cronbach's $\alpha=.88$). Teacher reports were used in this study for two reasons: childhood diagnoses are under-identified in the absence of teacher reports (Ford, Goodman, & Meltzer, 2003), and to minimize shared-variance between parent reports of their own and their child's behavior.

Parenting Behaviors. The parenting dimensions inventory (PDI; Power, 1993) was used at age 5 to measure parenting behaviors at age 5. The PDI is a 47-item self-report measure of parenting behaviors. Each item allows the parent to indicate according to a scale from 1 to 6 how much the given item describes the parent's typical behavior towards the participating child (e.g., "once a month," "several times daily"). For the current study, we examined three subdomains derived from this scale. A parental warmth index was estimated by merging the Nurturance and Responsiveness subscales ($\alpha=.74$; see Kerr, Lopez, Olson, & Sameroff, 2004). A nonaggressive inductive discipline index was created by merging the Reasoning and Reminding subscales ($\alpha=.71$; see Kerr et al., 2004). Finally, harsh discipline was measured using the Physical Punishment subscale ($\alpha=.75$).

Stress Procedures (Age 7)

Approximately 1–2 years following the first visit, each child participated in a stress task at a preschool center located at a large, public university in the Midwest. Children were unfamiliar with the preschool center. All visits were conducted in the late afternoon (3:00 p.m. or 4:30 p.m.) on nonschool days. The stress task protocol consisted of a 30-min baseline phase, a 3-min stress task, and a 60-min regulation period for a total of 93 min.

Baseline Phase. A 30-min baseline phase was used to allow for the regulation of the stress response to any stressors that occurred prior to arrival and because the preschool center was novel to the children. During the baseline phase, each child met a research assistant (RA) who accompanied the child for the duration of the visit. The RA first directed the child to a playroom where he/she could play with Legos, a castle, puzzles, or draw. After the baseline procedures, the child was led into the experiment room by the RA for the stress task.

Stress Tasks. Upon arrival to the preschool, each child was randomly assigned to one of two stress task conditions: fear ($n=33$) or frustration ($n=32$). Both tasks were effective in producing a stress response (Lopez-Duran et al., 2009a). The mean peak response for youth in the fear condition was $.112 \mu\text{g/dl}$ ($SD=.023$) while the peak response for youth in the frustration condition was $.107 \mu\text{g/dl}$ ($SD=.023$), $t(63)=.70$, $p=.49$. See Figure 1 and Table 1 for cortisol reactivity following onset of the fear and frustration tasks separately. Therefore, the results of both tasks were merged into a single stress protocol, which are intended to broadly represent negative affective states and their associated neuro-endocrine responses.

Fear Task. A fear response was obtained using a 3-min fear paradigm based on Calkins's fear protocol (see Calkins,

Graziano, Berdan, Keane, & Degnan, 2008). The task took place in a semi-dark room that had an empty fish tank with a rubber snake partially covered with mulch. The RA and the child slowly approached the tank as the RA made specific statements according to a script, including "I have something that I want to show you. It's inside that tank. Let's be quiet so it doesn't wake up." The RA initially asked the child to keep away from the tank while they removed a blanket covering the top. Then the child was invited to approach the tank. When the child began to approach, the RA abruptly took the snake out of the tank while simultaneously indicating that the snake was fake. At the end of the task, the child was provided with a prize identical to that used in the frustration condition.

Frustration Task. A frustration response was obtained using a 3-min frustration paradigm (Calkins, 1997). After the baseline phase, the child was led into the experiment room. On a table in front of the child there was a clear Tupperware box with a gift card to Toys'R'Us inside. The RA instructed the child to open the box in under a minute with socks on their hands. The child was told that if they open the container while keeping their hands inside the socks, he/she would win the gift card. The RA then proceeded to demonstrate completion of the task while saying, "This is so easy, even a baby could do it." After the demonstration the RA discretely switched the box with an identical box that had been glued shut. The child was told that he/she had 1 min to open the box. The RA then left the room. After 1 min, the RA returned and explained to the child that the box was "probably broken" and gave the gift card to the child.

The stress tasks were conducted with the approval of the University's institutional review board. They were selected because they reflect naturalistic stressors that children encounter often in their daily life (i.e., completing very difficult tasks, encountering fear eliciting stimuli). The tasks are also very short (under 5 min), which mitigates the effects of prolonged exposure to the stressors. Furthermore, the HPA-axis activation produced by these tasks is significantly less

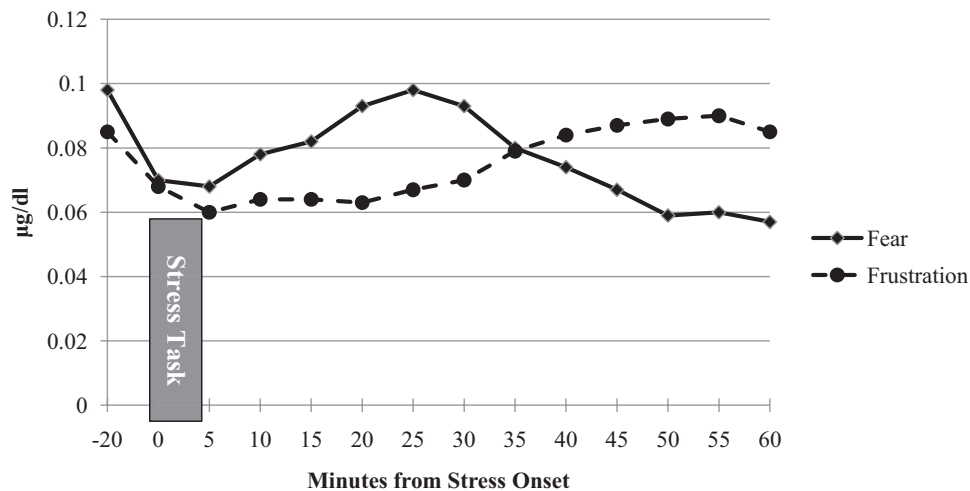


FIGURE 1 Unadjusted salivary cortisol response from onset of fear and frustration stress task conditions.

Table 1. Mean (SD) Cortisol Values (µg/dl) in Response to Each Psychosocial Stress Task (Fear and Frustration)

Condition	M (SD)													
	-20	0	5*	10**	15***	20***	25***	30***	35***	40*	45***	50***	55***	60***
Fear	.098 (.05)	.070 (.02)	.068 (.02)	.078 (.03)	.082 (.02)	.093 (.03)	.098 (.03)	.093 (.03)	.080 (.02)	.074 (.02)	.067 (.02)	.059 (.02)	.060 (.01)	.057 (.02)
Frustration	.085 (.02)	.068 (.02)	.060 (.02)	.064 (.02)	.064 (.02)	.063 (.02)	.067 (.02)	.070 (.03)	.079 (.02)	.084 (.02)	.087 (.03)	.089 (.03)	.090 (.03)	.085 (.02)

**p* < .10.
 ***p* < .05.
 ****p* < .01.

intense than that produced by the simple act of coming to the laboratory (Lopez-Duran et al., 2009a), which suggests that these tasks are very mild and less stressful than other common experiences children endure in their daily life (e.g., going to a doctor’s appointment).

Regulation Phase. Immediately following the stress task, the child was led into a new room with chairs, cushions and a television. The child watched two 30-min episodes of *Wallace and Gromit* from Aardman Animations (Episodes: “A Grand Day Out” and “A Close Shave”). These videos were selected for their popularity with children and their lack of emotionally arousing content (for methodological validation of these procedures, see Lopez-Duran et al., 2009a).

HPA-Axis Stress Reactivity. HPA-axis stress functioning was estimated from cortisol extracted from 17 saliva samples obtained during the stress protocol. To obtain cortisol samples, children spit directly into a salivette tube without the use of any agents (such as chewing gum) to facilitate saliva production. The first saliva sample was taken in the first minute of the baseline period. At this time, a stopwatch was started and all further samples were collected according to a strict schedule: 20, 10, 5, and 0 min prior to the stress task, one immediately following the stress task, then 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, and 60 min after the initiation of the stress task. The baseline (0 min) and all poststress samples were used in the analysis of stress reactivity. All salivettes were stored in a freezer at -20°C until assayed. Samples were assayed at a University of Michigan endocrinology laboratory within 6 months of collection in duplicate and averaged using a commercial enzyme immunoassay kit (Salimetrics, State College, PA). All cortisol reactivity methods are consistent with the recommendations of Clements (2012) for use of salivary cortisol in developmental research.

Data Analysis

We conducted a series of first-order autoregressive multiple regression models (Jöreskog, 1979) using a Generalized Linear Model framework via SAS PROC-GLM to test our hypothesis that parenting and HPA-axis reactivity would predict relative increases in internalizing symptoms during preadolescence. First, we predicted age 11 internalizing symptoms from age 5 internalizing symptoms (Step 1), and parental warmth, inductive discipline and physical punishment (Step 2). Second, we predicted age 11 from age 5 internalizing symptoms and baseline cortisol (Step 1) and HPA-axis reactivity (Step 2). We included baseline cortisol (the sample obtained immediately before the stressor) to control for the impact that baseline levels can have on HPA-axis reactivity (Kudielka, Gierens, Hellhammer, Wüst, & Schlotz, 2012). In analyses where HPA-axis reactivity is a predictor in the model, HPA-reactivity was calculated using Area Under the Curve-Increase (AUCi) via trapezoidal aggregation (Matthews, Altman, Campbell, & Royston, 1990) including baseline and all poststress samples. AUCi denotes the total cortisol produced after the stress task over and above the cortisol levels already present at baseline. It is preferable

to change scores from baseline to a specific time point as an index of reactivity because it accounts for individual variability in peak times (see Lopez-Duran et al., 2009a).

To test our mediation hypothesis, we first examined whether parenting behaviors predicted HPA-axis reactivity. For these analyses, AUCi was not used as an index of HPA-axis reactivity. Instead, we modeled the entire poststress cortisol curves using a growth curve modeling framework via SAS PROC MIXED, which is preferable to repeated measures because it does not assume independence of cortisol samples within individuals (Hruschka, Kohrt, & Worthman, 2005). However, due to significant individual variability in peak times, we applied a nonparametric curve-correction technique to the individual curves based on landmark registration (Molinari & Gasser, 2004; Ramsay & Li, 1998). Specifically, we anchored individual peak values upon a common peak time by aligning the individual growth curves on the horizontal (time) axis so that individual peak levels fall on the same time point, and the *x*-axis then reflects minutes before peak. This approach allows us to model the impact of predictors on true individual peaks (common intercept), as well as the reactivity slope towards the peak (acceleration before peak) while controlling for baseline levels. Finally, for those sets of variables in which the basic assumptions for mediation were met (Baron & Kenny, 1986), we conducted a mediation analysis using a standard bootstrap technique based on the SAS-PROC algorithm (Preacher & Hayes, 2004). Bootstrapping offers significant benefits over the more traditional Sobel approach since it makes fewer inaccurate assumptions about the shape of the sampling distribution of the indirect effect and has more power to identify indirect effects (Shrout & Bolger, 2002; Williams & MacKinnon, 2008).

RESULTS

Our sample includes complete data from 65 participants (35 boys) across three time points: age 5, 7, and 11. Table 2 presents descriptive statistics and correlations

between all parenting, HPA-axis reactivity and internalizing variables. As expected, internalizing symptoms increased significantly between age 5 and 11, $t(129) = -4.72$, $p < .001$; $d = .49$. Specifically, at the age 5 assessment, no participants in our sample had internalizing symptoms in the clinically significant range (T -scores range = 33–63), whereas at age 11, 9% of our sample had clinically significant internalizing symptoms ($T > 68$; range = 33–74).

Predictors of Internalizing Symptoms

Predicting Change in Internalizing Symptoms From Parenting Behaviors. First, we modeled age 11 internalizing symptoms as predicted by age 5 internalizing symptoms (AIC = 345.9). Age 5 internalizing symptoms were associated with internalizing symptoms at age 11, $\beta = .63$, $t(52) = 2.10$, $p < .05$. We then added the effects of maternal warmth, physical punishment, and induction at age 5. This model demonstrated improved fit over our first model (AIC = 321.8), where maternal warmth demonstrated a main effect on age 11 internalizing symptoms, $p < .001$. Neither induction nor physical punishment exhibited a main effect on age 11 internalizing symptoms, $p = .95$ and $p = .83$, respectively. See Table 3 for parameter estimates for adjusted models predicting age 11 internalizing symptoms.

Predicting Change in Internalizing Symptoms From HPA-Axis Reactivity. Here, we modeled age 11 internalizing symptoms as predicted by age 5 internalizing symptoms and baseline cortisol (AIC = 340.2). As expected, our base model indicated that internalizing symptoms during middle childhood were associated with internalizing symptoms at age 11, $\beta = .63$, $t(51) = 2.08$,

Table 2. Means, Standard Deviations, and Correlations Between All Parenting, HPA-Axis and Internalizing Symptom Variables

	1	2	3	4	5	6	7	8	Mean	SD
Time 1										
1. Age	1.00								5.79	.31
2. Internalizing ^a	.391*	1.00							1.98	2.86
3. M. warmth	-.246	-.394*	1.00						5.39	.48
4. M. reasoning ^a	.040	-.330	-.146	1.00					1.96	.40
5. M. punishment ^a	.032	.240	.037	-.467**	1.00				.16	.23
Endocrine										
6. Baseline ^a	.049	-.100	.009	.173	.112	1.00			.069	.018
7. Reactivity (AUCi) ^a	.101	.133	-.361**	.108	-.091	.348**	1.00		3.70	.82
Time 2										
8. Age	.627**	.364	-.363**	-.028	.095	-.048	.023	1.00	10.49	.45
9. Internalizing ^a	.214	.344	-.515**	.041	.033	.089	.296*	.125	4.25	5.85

^aVariables were log transformed due to skewness and/or kurtosis, raw means are reported.

* $p < .05$.

** $p < .01$.

Table 3. Parameter Estimates From Models With HPA-Axis and Parenting Variables Predicting Age 11 Internalizing Symptoms

Model	Fixed Effects	β	SE	t-Value
Parenting	Age 5 internalizing	.28	.30	.94
	M. warmth	-1.45**	.40	-3.60**
	M. induction	-.03	.49	.95
HPA-axis	M. punishment	-.80	3.69	-.22
	Age 5 internalizing	.44	.30	1.49
	Baseline	-5.98	7.06	-.85
	AUCi	2.51	1.04	2.41*

* $p < .05$.
 ** $p < .01$.

$p < .05$, and that baseline cortisol did not predict age 11 internalizing symptoms, $\beta = .02$, $t(51) = .00$, $p = .99$. The inclusion of HPA-axis reactivity (AUCi) improved the model fit (AIC = 332.9). Specifically, greater cortisol reactivity (AUCi) predicted higher internalizing symptoms in preadolescence, $p < .05$ (see Tab. 3).

Parenting Predictors of HPA-Axis Reactivity

We first examined conditional linear and quadratic growth models of poststress cortisol using peak values

as the intercept. All of our models include prestress cortisol as a control. The quadratic model was the best fit to the data (linear model AIC = -2,264.6 vs. quadratic model AIC = -2,033.0). For this quadratic model, the average predicted peak (Intercept) was significantly greater than 0, $\beta = 21.54$, $t(61) = 13.67$, $p < .001$. The slope toward this peak was positive, time $\beta = .284$, $t(554) = 13.96$, $p < .001$, and displayed a nonlinear acceleration, time² $\beta = .005$, $t(554) = 8.78$, $p < .001$.

We then conducted conditional unadjusted and adjusted models for each of our parenting domains: warmth, induction, and physical punishment as they influenced both the slope and peak parameters of poststress cortisol curves (see Tab. 4 for a summary of all models). In our unadjusted maternal warmth model (AIC = 2,208.7), there was a main effect of maternal warmth on peak values, $p < .001$, indicating that greater maternal warmth predicted lower cortisol peaks. However, maternal warmth did not impact the linear or quadratic slopes. Given that peak values are a function of starting value (baseline), acceleration (slopes), and tempo (timing of activation), the impact on peaks but not slope suggest that maternal warmth may decrease the timing of activation (how long it takes to reach peak) but not the intensity (slope) of such activation. In our unadjusted maternal induction model (AIC = 2,234.1), there was a main effect of induction on peak

Table 4. Unadjusted and Adjusted Modeling of Age 5 Parenting Behaviors Predicting Age 7 HPA-Axis Reactivity

Model	Predictor Variable	β	Std. Error	t-Value
Unadjusted models				
Warmth	Warmth	-.374	.080	-4.65**
	Warmth effect on linear slope	-.004	.010	-.37
	Warmth effect on quadratic slope	1.24×10^{-4}	2.08×10^{-4}	.59
Induction	Induction	-.283	.086	-3.30**
	Induction effect on linear slope	-.037	.010	-3.80**
	Induction effect on quadratic slope	-5.70×10^{-4}	2.08×10^{-4}	-2.75**
Physical punishment	M. punishment	1.55	.625	2.49*
	Punishment effect on linear slope	.163	.069	2.35*
	Punishment effect on quadratic slope	.002	.001	1.62
Adjusted model				
Parenting model	Warmth	-.426	.080	-5.31**
	Induction	-.321	.095	-3.37**
	Punishment	.533	.683	.78
	Warmth effect on linear slope	-.009	.010	-.99
	Induction effect on linear slope	-.036	.011	-3.27**
	Punishment effect on linear slope	.038	.076	.50
	Warmth effect on quadratic slope	3.70×10^{-5}	2.09×10^{-4}	.18
	Induction effect on quadratic slope	-.001	2.31×10^{-4}	-2.34*
	Punishment effect on quadratic slope	3.47×10^{-4}	.002	.22

* $p < .05$.
 ** $p < .01$.

values, $p < .01$, indicating that greater induction predicted lower peaks. There was also a significant impact of induction on both linear and quadratic slopes, $p < .001$ and $p < .01$, respectively. This suggests that induction reduces the intensity (acceleration) of the activation. In our unadjusted physical punishment model (AIC = 2,231.3), there was a main effect of physical punishment on peak values, $p = .05$, indicating that more reported physical punishment predicted higher peaks. In addition, physical punishment impacted the linear slope, $p < .05$, indicating that more reported physical punishment increased the intensity of activation.

We then conducted an adjusted model of all three parenting domains as predictors of peak cortisol and activation slopes (AIC = 2,229.7). See Table 4 for model fit and parameter estimates for parenting predicting HPA-axis reactivity. Consistent with the unadjusted models, there were main effects for both maternal warmth, and induction on peak cortisol, $p < .001$ and $p < .01$, respectively. There was also an interaction between induction and both the linear and quadratic slope of time to peak such that high reported induction decreased the intensity of the activation, $p < .01$ and $p < .02$. However, the effect of physical punishment on peak and acceleration slope was no longer significant after controlling for maternal warmth and induction. See Figure 2 for adjusted peak reactivity by warmth, induction, and physical punishment.

HPA-Axis as Mediator Between Parenting and Preadolescent Internalizing Symptoms

Given that only maternal warmth met the basic mediation conditions, physical punishment, and induction were not subjected to mediation analyses. The first regression equation (estimate path c) demonstrated a significant effect of maternal warmth on age 11 internalizing symptoms, $\beta = -.462$, $p < .001$. The second regression equation (estimate path a) demonstrated that the predictor (age 5 maternal warmth) had an effect on the proposed mediator (AUCi), $\beta = -.361$, $p < .01$. The third regression equation (estimate path b) demonstrated an effect of the proposed mediator (AUCi) on age 11 internalizing symptoms, when controlling for age 5 internalizing symptoms, $\beta = .325$, $p = .05$. Finally, in a fourth step to estimate path c', the effect of maternal warmth on age 11 internalizing symptoms controlling for the proposed mediator (AUCi), was still significant. This suggests a partial mediation effect of AUCi, $\beta = -.390$, $p < .01$. This was confirmed by bootstrapping the indirect effect and significance (10,000 bootstrap resamples) using normal distribution with a 95% confidence interval (2-tailed), value = $-.25$,

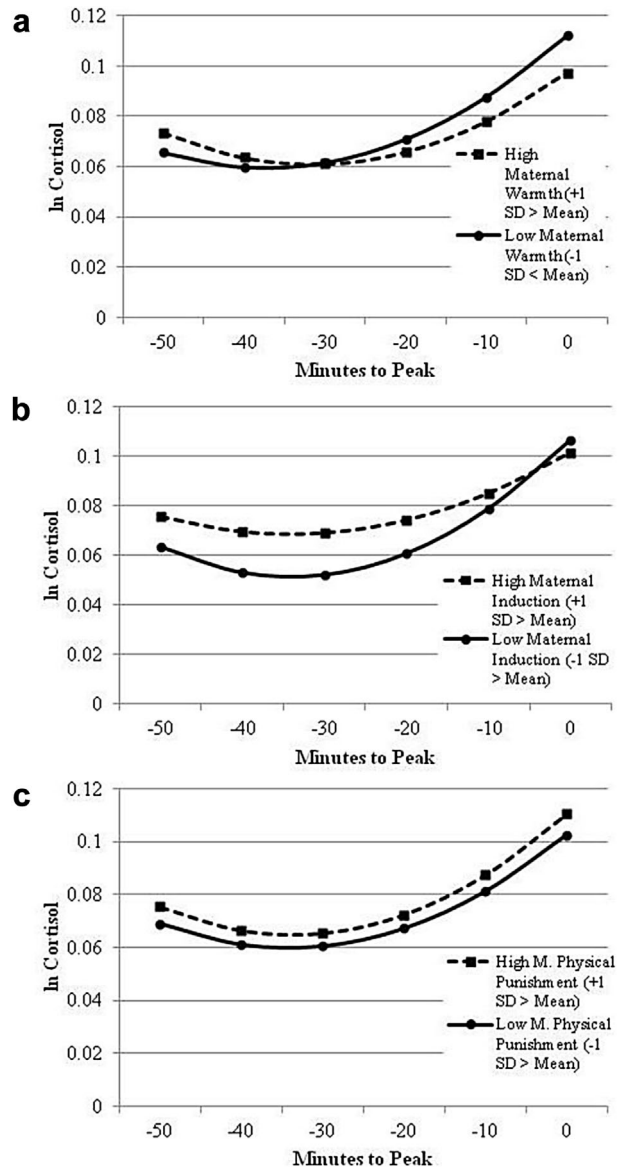


FIGURE 2 Slope of salivary cortisol reactivity peak by high and low reported (a) maternal warmth and responsiveness, (b) maternal induction, and (c) physical punishment.

$SE = .16$, 95% CIs $[-.68, -.03]$, $t = -4.29$, $p < .0001$. See Figure 3. We further tested this association by conducting the reverse mediation, with age 7 HPA-axis reactivity as a mediator between age 5 internalizing behaviors and age 11 maternal warmth. However, age 5 internalizing behaviors were only a predictor of change in maternal warmth from age 5 to 11 at trend level, $\beta = .244$, $p = .07$, and age 5 internalizing behaviors were not a significant predictor of HPA-axis response at age 7, $\beta = .197$, $p = .14$.

We conducted post hoc analyses which demonstrated that youth with mothers reporting above the mean on

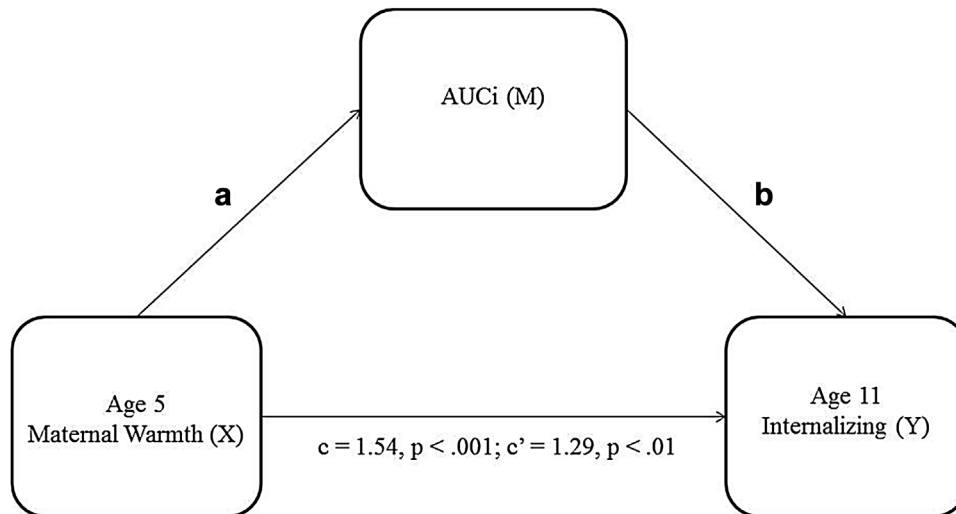


FIGURE 3 HPA-axis reactivity (AUCi) as a partial mediator between age 5 maternal warmth and internalizing symptoms during the transition into adolescence (5–6 years later).

warmth and youth with greater cortisol reactivity to stress (AUCi) were less likely to score in the clinically significant range of internalizing symptoms at age 11, $\chi^2 = 3.37$, $p = .067$ and $\chi^2 = 6.48$, $p = .01$, respectively. Thus, there appear to be some specificity to our findings with regard to clinically significant internalizing symptoms. However, given the small number of youth who exceeded the clinically significant cut-off for internalizing symptoms in this sample ($n = 6$), we would approach these findings with caution and strongly encourage replication with a clinical sample.

DISCUSSION

In this longitudinal study, we examined parenting and HPA-axis predictors of changes in internalizing symptoms from age 5 to 11. Among the examined parenting behaviors, warmth was the strongest predictor of internalizing symptoms during preadolescence. Specifically, greater warmth at age 5 was a protective factor in the emergence of internalizing symptoms at age 11. In addition to maternal warmth, HPA-axis reactivity was a significant predictor of changes in internalizing symptoms from age 5 to 11. While maternal warmth was the only parenting domain predictive of changes in internalizing symptoms, all three parenting domains at age 5 were predictive of HPA-axis reactivity at age 7. Finally, warmth may impact the trajectory of internalizing symptoms in part through its influence on the child's HPA-axis reactivity. To our knowledge, this is the first prospective study to longitudinally characterize the interplay between parenting and the child's HPA-

axis as predictors of internalizing symptoms during the transition to adolescence.

Maternal warmth served as a protective factor that mitigated increases in internalizing symptoms that occur from age 5 to 11. This is consistent with previous findings that maternal warmth is associated with positive adolescent outcomes. For example, concurrent maternal warmth has been linked to fewer depression and anxiety symptoms (e.g., Schwartz et al., 2012), better academic performance (Spera, 2005), better social adjustment (Domitrovich & Bierman, 2001), and better effortful control (Eisenberg et al., 2005). Given that parents can serve as interpersonal regulatory agents to their developing children (see Kovacs & Lopez-Duran, 2012), it is possible that when parents respond to distress in their children with warmth they are facilitating acquisition of regulatory resources by selectively reinforcing the use of social agents as a source of regulation. Over time, the children whose parents modeled or selectively reinforced adaptive regulatory responses to stress may develop adaptive coping strategies to be used in the transition to adolescence and beyond. Physical punishment, however, was not associated with internalizing symptoms, which is contrary to previous findings (e.g., Bender et al., 2007; Gershoff, 2002; Leve et al., 2005). This finding may be attributed to 68% of our mothers reporting no physical punishment at all while parents who endorsed the use of physical punishment reported very infrequent use. Therefore, our data can only be interpreted as the failure to find an association between *minimal physical punishment* and changes in internalizing symptoms from age 5 to 11.

Furthermore, greater HPA-axis reactivity at age 7 was associated with increases in internalizing symptoms at age 11. This is consistent with other studies linking HPA-axis reactivity with internalizing symptoms and disorders in late childhood (Hankin, Badanes, Abela, & Watamura, 2010; Luby et al., 2003; Smider et al., 2002), adolescence (Guerry & Hastings, 2011; Lopez-Duran et al., 2009b; Rao et al., 2010), and studies linking atypical HPA-axis functioning throughout the day to internalizing symptoms in middle childhood (Turner-Cobb et al., 2008). It is possible that HPA-axis reactivity impacts the trajectory of internalizing symptoms by facilitating increased affective responses to environmental stressors. For example, adaptive stress reactivity (including heart rate, cortisol, and blood pressure) has been associated with better anger regulation during acute stress (Cook, Chaplin, Sinha, Tebes, & Mayes, 2012). In addition, elevated cortisol increases physiological arousal to *neutral* stimuli (Abercrombie, Kalin, & Davidson, 2005). Thus, given that arousal is a key component of emotional experiences (Schachter & Singer, 1962), it is possible that greater HPA-axis reactivity to stress may facilitate or extend the experience of negative emotional states in response to nonstressful situations. It is also possible that HPA-axis hyperreactivity to stressors may prolong negative emotional states through impairments in cognitive regulation strategies (e.g., attention disengagement). For example, failure to rapidly disengage from dysphoric and threat-related stimuli is associated with greater cortisol reactivity as well as decreases in mood (Ellenbogen, Schwartzman, Stewart, & Walker, 2006). Over time, atypical reactivity of the HPA-axis in response to acute stressors may facilitate long-term impairments in cognitive regulation strategies while maintaining negative affectivity. It is possible that increased negative affective responses and failure to disengage from negative stimuli as facilitated by exposure to stress hormones are particularly germane to the transition into adolescence, and thus may be a source of vulnerability for the development of internalizing disorders.

When examining the association between parenting behaviors and HPA-axis reactivity, all three domains of parenting (warmth, induction, and physical punishment) were associated with HPA-axis reactivity in unadjusted models. More maternal warmth at age 5 was associated with lower cortisol peaks 1–2 years later, which is consistent with cross-sectional examinations of the link between parental warmth and HPA-axis functioning (Engert, Efanov, Dedovic, Dagher, & Pruessner, 2011; Marsman et al., 2012). Also, maternal induction (i.e., reasoning and reminding) was associated with a less reactive HPA-axis affecting both peak levels and the

acceleration slope. To date, there have been no studies looking specifically at inductive parenting and HPA-axis reactivity. However, studies have noted a longitudinal relationship between “effective” or “positive” parenting and a less reactive HPA-axis (e.g., Bugental, 2004; Hagan et al., 2011). These broader parenting constructs have included parenting behaviors such as affection, responsiveness as well as inductive discipline using reasoning and reminding about the consequences of a child’s behavior (see Sandler, Schoenfelder, Wolchik, & MacKinnon, 2011, for review). Finally, while physical punishment was not associated with changes in internalizing symptoms across our observations, we did find that reports of physical punishment at age 5 were associated with a steeper cortisol acceleration slope and higher peak cortisol levels even though our sample reported infrequent use of physical punishment. These findings are consistent with literature suggesting that the use of physical punishment in childrearing exerts a negative influence on the HPA-axis across many stages of development (Bugental et al., 2003; Roisman et al., 2009). However, physical punishment did not impact HPA-axis once we controlled for the impact of other parenting behaviors.

Most surprisingly, however, our data suggest that different parenting behaviors may impact different aspects of the HPA-axis. Specifically, atypically high cortisol in response to stress can reflect dysregulation within various aspects of the HPA-axis response, such as adrenal hypersensitivity to ACTH or deficits with the system’s negative feedback mechanism (Miller & O’Callaghan, 2002). However, a closer examination of the pattern of results may help us speculate about specific mechanisms at play. For example, peak levels are a function of *baselines* (controlled for in all models), *acceleration* (speed and intensity) of adrenal activation (reflected by activation slope), and *duration* of such acceleration, primarily controlled by glucocorticoid receptors (GRs). Maternal warmth was associated with final peak but not with activation slopes, suggesting that warmth may not impact the acceleration of activation (slopes) but instead decreases the *duration* of the activation, maybe through sensitization of GRs. This is also consistent with animal studies, where maternal care behaviors (e.g., licking and grooming) are associated with the development of more GRs in the brain (Caldji, Hellstrom, Zhang, Diorio, & Meaney, 2011). On the other hand, when controlling for the effect of warmth, induction was associated with acceleration slopes and peaks. This suggests that low induction may result in a faster or more intense activation of the axis and possibly low feedback sensitivity. Thus, low induction may impact reactivity by increasing CRH or ACTH sensitivity resulting in

greater adrenal output, or alternatively by limiting cognitive processes that help regulate the activation of the HPA-axis (e.g., Gaab, Rohleder, Nater, & Ehlert, 2005).

Finally, maternal warmth was associated with preadolescent internalizing symptoms partially through its impact on HPA-axis reactivity. Consistent with this finding, more structured parenting (e.g., organization and consistency) in middle childhood predicted lower stress reactivity to acute stress and better behavioral adjustment at age 13–16 for children of parents with Bipolar Disorder (Ellenbogen & Hodgins, 2009). Our study extends these findings to parental warmth and internalizing symptoms and demonstrates that this influence can be seen as early as 11. Additionally, this finding now extends to children who may not be at familial risk for the development of a mood disorder. This finding is important to our understanding of the transition to adolescence because parental warmth in childhood may contribute to improved negative feedback sensitivity and thus mitigate the impact of adolescent stress. Furthermore, the *partial* mediation suggests that regulation of HPA-axis functioning do not fully explain the impact of parenting on the development of internalizing symptoms. It is likely that there are additional mediators in the relationship between parenting behaviors and adolescent symptoms which may be independent of HPA-axis functioning, such as life satisfaction (Suldo & Huebner, 2004) and self-worth (Garber, Robinson, & Valentiner, 1997).

Limitations

Our findings should be considered in the context of some limitations. Despite the longitudinal and prospective nature of this study, our findings are correlational and therefore causal relationships between parenting and preadolescent internalizing symptoms cannot be inferred from these data. For example, there is also a wealth of evidence to support the bidirectional relationship between parenting and child behaviors (e.g., Clark, Kochanska, & Ready, 2000; Sameroff, 2009). To address this in our data, we conducted the mediation in reverse; however, the parameters for mediation in this direction were not met. It is also important to note that the prevalence of clinically significant symptoms of internalizing in our sample was relatively low. In addition, the variations in maternal warmth were associated with 1–2 symptom differences on the internalizing symptom scale. Thus, our findings highlight the impact of maternal warmth on internalizing symptoms within a fairly normative range, and thus may be less informative to the development of specific psychopathology. Also, only maternal reports of parent-

ing behaviors were included and thus our findings only reflect the potential role of mothers in the development of internalizing symptoms. Most preclinical animal models have also been limited to the effects of maternal care on the regulation of the HPA-axis (e.g., Suchecki et al., 1993); however, there is growing evidence that paternal parenting behaviors play a distinct role in adolescent depressive disorder development (Sheeber, Davis, Leve, Hops, & Tildesley, 2007). Therefore, efforts to replicate these findings may consider the differential roles mothers and fathers play in the development of the HPA-axis and internalizing symptoms. Additionally, parenting was self-reported by mothers and therefore socially desirable parenting behaviors, such as warmth, may be over-reported while other parenting behaviors, such as use of physical punishment may be under-reported due to social-monitoring. These findings would be strengthened if compared with more objective behavioral measures of parenting in a laboratory setting. In this study, we did not collect data on the pubertal status of our sample and therefore cannot comment on how our findings are related to pubertal development. A large proportion of our “preadolescent” sample has likely surpassed Tanner Stage III by our age 11 assessment (see Table 3 of Euling et al., 2008). Therefore, our sample likely reflected a transitional cohort of children ranging from pre to early adolescence. Considering the associations between internalizing symptoms and pubertal development (Marceau, Neiderhiser, Lichtenstein, & Reiss, 2012) as well as pubertal status and HPA-axis functioning (Hankin et al., 2010), future studies of this nature would benefit from the assessment of pubertal development in youth ages 9 and older. Finally, the stress tasks used to facilitate HPA-axis reactivity in this study were designed to elicit fear and frustration in a limited social setting (no explicit peer evaluation). Previous research suggests that HPA-axis activation is best facilitated by social threat (Kirschbaum, Pirke, & Hellhammer, 1993; Kudielka, Hellhammer, & Kirschbaum, 2007). Therefore, it would be useful for future investigations to replicate these findings using a more common HPA-axis stress protocol that is more explicitly social, such as the TSST-C (Buske-Kirschbaum et al., 1997), or that is known to elicit similar responses in adult populations, such as the TSST-M (Yim, Quas, Cahill, & Hayakawa, 2010).

CONCLUSIONS

In conclusion, our study provides evidence for the link between parenting and specific aspects of the HPA-axis. Specifically, this study suggests that maternal warmth

during middle childhood may be a protective factor in the development of internalizing symptoms during the transition to adolescence through its role in modulating HPA-axis reactivity. Current theories on a biobehavioral model for the development of internalizing disorders explain that un-supportive, neglectful and cold family environments can be a risk factor that inhibits adaptive emotion processing and social competence (Repetti, Taylor, & Seeman, 2002). There is evidence, however, that early environmental stress and its effects can be reversed by the presence of supportive maternal nurturing behavior (Caldji, Diorio, & Meaney, 2000; Kuhn & Schanberg, 1998). Furthermore, the introduction of nurturing caretaker behaviors to an individual who is already at-risk or experiencing internalizing symptoms can stabilize observed dysregulation in HPA-axis functioning (Southwick, Vythilingam, & Charney, 2005). This suggests that nurturing behavior has a dynamic relationship to HPA-axis development and that the positive developmental consequences of nurturing behavior extend beyond infancy. Our findings highlight the role of maternal warmth and the child's HPA-axis functioning in the protection against internalizing symptoms and thus may contribute to the identification of intervention targets for preventing internalizing disorders. For example, interventions which aim to increase inductive discipline, allow for more parent-child opportunities for warmth, and reduce the use of harsh punishment as a parenting strategy may have long-term biological and behavioral benefits.

NOTES

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REFERENCES

- Abercrombie, H. C., Kalin, N. H., & Davidson, R. J. (2005). Acute cortisol elevations cause heightened arousal ratings of objectively nonarousing stimuli. *Emotion, 5*(3), 354–359. doi: 10.1037/1528-3542.5.3.354
- Achenbach, T. M. (1991). *Child behavior checklist/4–18*. Burlington: University of Vermont.
- Achenbach, T. (1992). *Child behavior checklist for ages 2–3*. Burlington, VT: Department of Psychiatry, University of Vermont.
- Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173–1182. doi: 10.1037/0022-3514.51.6.1173
- Bayer, J. K., Sanson, A. V., & Hemphill, S. A. (2006). Parent influences on early childhood internalizing difficulties. *Journal of Applied Developmental Psychology, 27*(6), 542–559. doi: 10.1016/j.appdev.2006.08.002
- Bender, H. L., Allen, J. P., McElhaney, K. B., Antonishak, J., Moore, C. M., Kelly, H. O., & Davis, S. M. (2007). Use of harsh physical discipline and developmental outcomes in adolescence. *Development and Psychopathology, 19*(1), 227–242. doi: 10.1017/S0954579407070125
- Berkien, M., Louwerse, A., Verhulst, F., & van der Ende, J. (2012). Children's perceptions of dissimilarity in parenting styles are associated with internalizing and externalizing behavior. *European Child & Adolescent Psychiatry, 21*(2), 79–85. doi: 10.1007/s00787-011-0234-9
- Bugental, D. B. (2004). Thriving in the face of early adversity. *Journal of Social Issues, 60*(1), 219–235. doi: 10.1111/j.0022-4537.2004.00108.x
- Bugental, D. B., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior, 43*(1), 237–244. doi: 10.1016/S0018-506X(02)00008-9
- Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine, 59*(4), 419–426.
- Caldji, C., Diorio, J., & Meaney, M. J. (2000). Variations in maternal care in infancy regulate the development of stress reactivity. *Biological Psychiatry, 48*(12), 1164–1174. doi: 10.1016/S0006-3223(00)01084-2
- Caldji, C., Hellstrom, I. C., Zhang, T. Y., Diorio, J., & Meaney, M. J. (2011). Environmental regulation of the neural epigenome. *FEBS Letters, 585*(13), 2049–2058. doi: 10.1016/j.febslet.2011.03.032
- Calkins, S. D. (1997). Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. *Developmental Psychobiology, 31*(2), 125–135. doi: 10.1002/(SICI)1098-2302(199709)31:2.<125:AID-DEV5>3.0.CO;2-M
- Calkins, S. D., Graziano, P. A., Berdan, L. E., Keane, S. P., & Degnan, K. A. (2008). Predicting cardiac vagal regulation in early childhood from maternal-child relationship quality during toddlerhood. *Developmental Psychobiology, 50*(8), 751–766. doi: 10.1002/dev.20344
- Clark, L. A., Kochanska, G., & Ready, R. (2000). Mothers' personality and its interaction with child temperament as predictors of parenting behavior. *Journal of Personality and Social Psychology, 79*(2), 274.
- Clements, A. D. (2012). Salivary cortisol measurement in developmental research: Where do we go from here? *Developmental Psychobiology, 55*(3), 205–220. doi: 10.1002/dev.21025
- Conger, R. D., & Conger, K. J. (2002). Resilience in midwestern families: Selected findings from the first

- decade of a prospective, longitudinal study. *Journal of Marriage and Family*, 64(2), 361–373. doi: 10.1111/j.1741-3737.2002.00361.x
- Cook, E. C., Chaplin, T. M., Sinha, R., Tebes, J. K., & Mayes, L. C. (2012). The stress response and adolescents' adjustment: The impact of child maltreatment. *Journal of Youth and Adolescence*, 41, 1067–1077. doi: 10.1007/s10964-012-9746-y
- Domitrovich, C. E., & Bierman, K. L. (2001). Parenting practices and child social adjustment: Multiple pathways of influence. *Merrill-Palmer Quarterly: Journal of Developmental Psychology*, 47(2), 235–263. doi: 10.1353/mpq.2001.0010
- Eisenberg, N., Zhou, Q., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control, and externalizing problems: A three-wave longitudinal study. *Child Development*, 76(5), 1055–1071. doi: 10.1111/j.1467-8624.2005.00897.x
- Ellenbogen, M. A., & Hodgins, S. (2009). Structure provided by parents in middle childhood predicts cortisol reactivity in adolescence among the offspring of parents with bipolar disorder and controls. *Psychoneuroendocrinology*, 34(5), 773–785. doi: 10.1016/j.psyneuen.2008.12.011
- Ellenbogen, M. A., Schwartzman, A. E., Stewart, J., & Walker, C. D. (2006). Automatic and effortful emotional information processing regulates different aspects of the stress response. *Psychoneuroendocrinology*, 31(3), 373–387. doi: 10.1016/j.psyneuen.2005.09.001
- Elzinga, B. M., Roelofs, K., Tollenaar, M. S., Bakvis, P., van Pelt, J., & Spinhoven, P. (2008). Diminished cortisol responses to psychosocial stress associated with lifetime adverse events: A study among healthy young subjects. *Psychoneuroendocrinology*, 33(2), 227–237. doi: 10.1016/j.psyneuen.2007.11.004
- Engert, V., Efanov, S. I., Dedovic, K., Dagher, A., & Pruessner, J. C. (2011). Increased cortisol awakening response and afternoon/evening cortisol output in healthy young adults with low early life parental care. *Psychopharmacology*, 214(1), 261–268.
- Essex, M. J., Klein, M. H., Cho, E., & Kalin, N. H. (2002). Maternal stress beginning in infancy may sensitize children to later stress exposure: Effects on cortisol and behavior. *Biological Psychiatry*, 52(8), 776–784. doi: 10.1016/S0006-3223(02)01553-6
- Euling, S. Y., Herman-Giddens, M. E., Lee, P. A., Selevan, S. G., Juul, A., Sørensen, T. I. A. ... Swan, S. H. (2008). Examination of US puberty-timing data from 1940 to 1994 for secular trends: Panel findings. *Pediatrics*, 121 (Suppl 3), S172–S191. doi: 10.1542/peds.2007-1813D
- Ford, T., Goodman, R., & Meltzer, H. (2003). The British child and adolescent mental health survey 1999: The prevalence of DSM-IV disorders. *Journal of the American Academy of Child, 42(10)*, 1203–1211. doi: 10.1097/00004583-200310000-00011
- Gaab, J., Rohleder, N., Nater, U. M., & Ehlert, U. (2005). Psychological determinants of the cortisol stress response: The role of anticipatory cognitive appraisal. *Psychoneuroendocrinology*, 30(6), 599–610. doi: 10.1016/j.psyneuen.2005.02.001
- Garber, J., Robinson, N. S., & Valentiner, D. (1997). The relation between parenting and adolescent depression: Self-worth as a mediator. *Journal of Adolescent Research*, 12(1), 12–33. doi: 10.1177/0743554897121003
- Gershoff, E. T. (2002). Corporal punishment by parents and associated child behaviors and experiences: A meta-analytic and theoretical review. *Psychological Bulletin*, 128(4), 539. doi: 10.1037/0033-2909.128.4.539
- Grusec, J. E., & Goodnow, J. J. (1994). Impact of parental discipline methods on the child's internalization of values: A reconceptualization of current points of view. *Developmental Psychology*, 30(1), 4–19. doi: 10.1037/0012-1649.30.1.4
- Guerry, J. D., & Hastings, P. D. (2011). In search of HPA axis dysregulation in child and adolescent depression. *Clinical Child and Family Psychology Review*, 14(2), 135–160. doi: 10.1007/s10567-011-0084-5
- Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, 27(1–2), 199–220. doi: 10.1016/S0306-4530(01)00045-2
- Hagan, M. J., Roubinov, D. S., Gress-Smith, J., Luecken, L. J., Sandler, I. N., & Wolchik, S. (2011). Positive parenting during childhood moderates the impact of recent negative events on cortisol activity in parentally bereaved youth. *Psychopharmacology*, 214(1), 231–238. doi: 10.1007/s00213-010-1889-5
- Hankin, B. L., Badanes, L. S., Abela, J. R. Z., & Watamura, S. E. (2010). Hypothalamic-pituitary-adrenal axis dysregulation in dysphoric children and adolescents: Cortisol reactivity to psychosocial stress from preschool through middle adolescence. *Biological Psychiatry*, 68(5), 484–490.
- Hessl, D., Dawson, G., Frey, K., Panagiotides, H., Self, H., Yamada, E., & Osterling, J. (1998). A longitudinal study of children of depressed mothers: Psychobiological findings related to stress. *Advancing research on developmental plasticity: Integrating the behavioral sciences and the neurosciences of mental health*, 256–265.
- Howell, K. H., Graham-Bermann, S. A., Czyn, E., & Lilly, M. (2010). Assessing resilience in preschool children exposed to intimate partner violence. *Violence and Victims*, 25(2), 150–164. doi: 10.1891/0886-6708.25.2.150
- Hruschka, D. J., Kohrt, B. A., & Worthman, C. M. (2005). Estimating between- and within-individual variation in cortisol levels using multilevel models. *Psychoneuroendocrinology*, 30(7), 698–714. doi: 10.1016/j.psyneuen.2005.03.002
- Hughes, K., & Coplan, R. J. (2010). Exploring processes linking shyness and academic achievement in childhood. *School Psychology Quarterly*, 25(4), 213–222. doi: 10.1037/a0022070
- Jöreskog, K. G. (1979). Statistical estimation of structural models in longitudinal developmental investigations. In J. R. Nesselroade & P. B. Baltes (Eds.), *Longitudinal research in the study of behavior and development*. (pp. 303–352). New York: Academic Press.

- Kerr, D. C., Lopez, N. L., Olson, S. L., & Sameroff, A. J. (2004). Parental discipline and externalizing behavior problems in early childhood: The roles of moral regulation and child gender. *Journal of Abnormal Child Psychology*, 32(4), 369–383. doi: 10.1023/B:JACP.0000030291.72775.96
- Kertes, D. A., Donzella, B., Talge, N. M., Garvin, M. C., Van Ryzin, M. J., & Gunnar, M. R. (2009). Inhibited temperament and parent emotional availability differentially predict young children's cortisol responses to novel social and nonsocial events. *Developmental Psychobiology*, 51(7), 521–532. doi: 10.1002/dev.20390
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test"—A tool for investigating psychobiology stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76–81.
- Kovacs, M., & Lopez-Duran, N. (2010). Prodromal symptoms and atypical affectivity as predictors of major depression in juveniles: Implications for prevention. *Journal of Child Psychology and Psychiatry*, 51(4), 472–496. doi: 10.1111/j.1469-7610.2010.02230.x
- Kovacs, M., & Lopez-Duran, N. L. (2012). Contextual emotion regulation therapy: A developmentally based intervention for pediatric depression. *Child and Adolescent Psychiatric Clinics of North America*, 21(2), 327–343. doi: 10.1016/j.chc.2012.01.002
- Kudielka, B. M., Hellhammer, D. H., & Kirschbaum, C. (2007). Ten years of research with the Trier Social Stress Test—revisited. In E. Harmon-Jones & P. Winkielman (Eds.), *Social Neuroscience: Integrating Biological and Psychological Explanations of Social Behavior* (pp. 56–83). New York, NY: Guilford Press.
- Kudielka, B. M., Gierens, A., Hellhammer, D. H., Wüst, S., & Schlotz, W. (2012). Salivary cortisol in ambulatory assessment—Some dos, some don'ts, and some open questions. *Psychosomatic Medicine*, 74(4), 418–431. doi: 10.1097/PSY.0b013e31825434c7
- Kuhn, C. M., & Schanberg, S. M. (1998). Responses to maternal separation: Mechanisms and mediators. *International Journal of Developmental Neuroscience*, 16(3–4), 261–270. doi: 10.1016/S0736-5748(98)00034-3
- Ladd, C. O., Huot, R. L., Thirivikraman, K. V., Nemeroff, C. B., Meaney, M. J., & Plotsky, P. M. (1999). Long-term behavioral and neuroendocrine adaptations to adverse early experience. *Progress in Brain Research*, 122, 81–103. doi: 10.1016/S0079-6123(08)62132-9
- Laskey, B. J., & Cartwright-Hatton, S. (2009). Parental discipline behaviours and beliefs about their child: Associations with child internalizing and mediation relationships. *Child: Care, Health and Development*, 35(5), 717–727. doi: 10.1111/j.1365-2214.2009.00977.x
- Leckman-Westin, E., Cohen, P. R., & Stueve, A. (2009). Maternal depression and mother-child interaction patterns: Association with toddler problems and continuity of effects to late childhood. *Journal of Child Psychology*, 50(9), 1176–1184. doi: 10.1111/j.1469-7610.2009.02083.x
- Leve, L. D., Kim, H. K., & Pears, K. C. (2005). Childhood temperament and family environment as predictors of internalizing and externalizing trajectories from age 5 to age 17. *Journal of Abnormal Child Psychology*, 33(5), 505–520. doi: 10.1007/s10802-005-6734-7
- Lopez-Duran, N. L., Hajal, N. J., Olson, S. L., Felt, B. T., & Vazquez, D. M. (2009a). Individual differences in cortisol responses to fear and frustration during middle childhood. *Journal of Experimental Child Psychology*, 103(3), 285–295. doi: 10.1016/j.jecp.2009.03.008
- Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009b). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, 34(9), 1272–1283. doi: 10.1016/j.psycheneu.2009.03.016
- Luby, J. L., Heffelfinger, A., Mrakotsky, C., Brown, K., Hessler, M., & Spitznagel, E. (2003). Alterations in stress cortisol reactivity in depressed preschoolers relative to psychiatric and no-disorder comparison groups. *Archives of General Psychiatry*, 60(12), 1248–1255. doi: 10.1001/archpsyc.60.12.1248
- Marceau, K., Neiderhiser, J., Lichtenstein, P., & Reiss, D. (2012). Genetic and environmental influences on the association between pubertal maturation and internalizing symptoms. *Journal of Youth and Adolescence*, 41(9), 1111–1126. doi: 10.1007/s10964-012-9762-y
- Marsman, R., Nederhof, E., Rosmalen, J. G. M., Oldehinkel, A. J., Ormel, J., & Buitelaar, J. K. (2012). Family environment is associated with HPA-axis activity in adolescents. The TRAILS study. *Biological Psychology*, 89(2), 460–466. doi: 10.1016/j.biopsycho.2011.12.013
- Matthews, J. N., Altman, D. G., Campbell, M. J., & Royston, P. (1990). Analysis of serial measurements in medical research. *BMJ*, 300(6719), 230–235. doi: 10.1136/bmj.300.6719.230
- Miller, D. B., & O'Callaghan, J. P. (2002). Neuroendocrine aspects of the response to stress. *Metabolism*, 51(6 Part B), 5–10. doi: 10.1053/meta.2002.33184
- Molinari, L., & Gasser, T. (2004). The human growth curve: Distance, velocity and acceleration. In R. C. Hauspie N. Cameron & L. Molinari (Eds.), *Methods in human growth research* (pp. 27–54). Cambridge, UK: Cambridge University Press.
- Muris, P., Meesters, C., & van den Berg, S. (2003). Internalizing and externalizing problems as correlates of self-reported attachment style and perceived parental rearing in normal adolescents. *Journal of Child and Family Studies*, 12(2), 171–183. doi: 10.1023/A:1022858715598
- Murray, L., Halligan, S. L., Goodyer, I., & Herbert, J. (2010). Disturbances in early parenting of depressed mothers and cortisol secretion in offspring: A preliminary study. *Journal of Affective Disorders*, 122(3), 218–223. doi: 10.1016/j.jad.2009.06.034
- Olson, S. L., Sameroff, A. J., Kerr, D. C. R., Lopez, N. L., & Wellman, H. M. (2005). Developmental foundations of externalizing problems in young children: The role of effortful control. *Development and Psychopathology*, 17(1), 25–45. doi: 10.1017/S0954579405050029
- Pope, A. W., & Bierman, K. L. (1999). Predicting adolescent peer problems and antisocial activities: The relative roles

- of aggression and dysregulation. *Developmental Psychology*, 35(2), 335–346.
- Power, T. G. (1993). Parenting dimensions inventory (PDI): A research manual. Unpublished manuscript, University of Houston.
- Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments & Computers*, 36(4), 717–731. doi: 10.3758/BF03206553
- Ramsay, J. O., & Li, X. (1998). Curve registration. *Journal of the Royal Statistical Society: Series B (Statistical Methodology)*, 60(2), 351–363. doi: 10.1111/1467-9868.00129
- Rao, U., Hammen, C. L., & Poland, R. E. (2010). Longitudinal course of adolescent depression: Neuroendocrine and psychosocial predictors. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(2), 141–151. doi: 10.1016/j.jaac.2009.09.008
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, 128(2), 330–366.
- Roelofs, J., Meesters, C., ter Huurne, M., Bamelis, L., & Muris, P. (2006). On the links between attachment style, parental rearing behaviors, and internalizing and externalizing problems in non-clinical children. *Journal of Child and Family Studies*, 15(3), 319–332. doi: 10.1007/s10826-006-9025-1
- Roisman, G. I., Susman, E., Barnett-Walker, K., Booth-LaForce, C., Owen, M. T., Belsky, J. ... Network, T. N. E. C. C. R. (2009). Early family and child-care antecedents of awakening cortisol levels in adolescence. *Child Development*, 80(3), 907–920. doi: 10.1111/j.1467-8624.2009.01305.x
- Sameroff, A. (2009). Transactional models in early social relations. *Human Development*, 18(1–2), 65–79.
- Sanchez, M. M. (2006). The impact of early adverse care on HPA axis development: Nonhuman primate models. *Hormones and Behavior*, 50(4), 623–631. doi: 10.1016/j.yhbeh.2006.06.012
- Sandler, I. N., Schoenfelder, E. N., Wolchik, S. A., & MacKinnon, D. P. (2011). Long-term impact of prevention programs to promote effective parenting: Lasting effects but uncertain processes. *Annual Review of Psychology*, 62, 299–329. doi: 10.1146/annurev.psych.121208.131619
- Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review*, 69(5), 379–399. doi: 10.1037/h0046234
- Schwartz, O. S., Dudgeon, P., Sheeber, L. B., Yap, M. B. H., Simmons, J. G., & Allen, N. B. (2012). Parental behaviors during family interactions predict changes in depression and anxiety symptoms during adolescence. *Journal of Abnormal Child Psychology*, 40(1), 59–71. doi: 10.1007/s10802-011-9542-2
- Shea, A., Walsh, C., MacMillan, H., & Steiner, M. (2005). Child maltreatment and HPA axis dysregulation: Relationship to major depressive disorder and post traumatic stress disorder in females. *Psychoneuroendocrinology*, 30(2), 162–178. doi: 10.1016/j.psyneuen.2004.07.001
- Sheeber, L. B., Davis, B., Leve, C., Hops, H., & Tildesley, E. (2007). Adolescents' relationships with their mothers and fathers: Associations with depressive disorder and sub-diagnostic symptomatology. *Journal of Abnormal Psychology*, 116(1), 144–154. doi: 10.1037/0021-843X.116.1.144
- Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, 7(4), 422–445. doi: 10.1037/1082-989X.7.4.422
- Silk, J. S., Vanderbilt-Adriance, E., Shaw, D. S., Forbes, E. E., Whalen, D. J., Ryan, N. D., & Dahl, R. E. (2007). Resilience among children and adolescents at risk for depression: Mediation and moderation across social and neurobiological contexts. *Development and Psychopathology*, 19(3), 841–865. doi: 10.1017/S0954579407000417
- Smider, N. A., Essex, M. J., Kalin, N. H., Buss, K. A., Klein, M. H., Davidson, R. J., & Goldsmith, H. H. (2002). Salivary cortisol as a predictor of socioemotional adjustment during kindergarten: A prospective study. *Child Development*, 73(1), 75–92. doi: 10.1111/1467-8624.00393
- Southwick, S. M., Vythilingam, M., & Charney, D. S. (2005). The psychobiology of depression and resilience to stress: Implications for prevention and treatment. *Annual Review of Clinical Psychology*, 1(1), 255–291. doi: 10.1146/annurev.clinpsy.1.102803.143948
- Spera, C. (2005). A review of the relationship among parenting practices, parenting styles, and adolescent school achievement. *Educational Psychology Review*, 17(2), 125–146. doi: 10.1007/s10648-005-3950-1
- Sturge-Apple, M. L., Davies, P. T., Martin, M. J., Cicchetti, D., & Hentges, R. G. (2012). An examination of the impact of harsh parenting contexts on children's adaptation within an evolutionary framework. *Developmental Psychology*, 48(3), 791–805. doi: 10.1037/a0026908
- Suchecki, D., Rosenfeld, P., & Levine, S. (1993). Maternal regulation of the hypothalamic-pituitary-adrenal axis in the infant rat: The roles of feeding and stroking. *Developmental Brain Research*, 75(2), 185–192. doi: 16/0165-3806(93)90022-3
- Suldo, S. M., & Huebner, E. S. (2004). The role of life satisfaction in the relationship between authoritative parenting dimensions and adolescent problem behavior. *Social Indicators Research*, 66(1–2), 165–195.
- Taylor, Z. E., Spinrad, T. L., Van Schyndel, S. K., Eisenberg, N., Huynh, J., Sulik, M. J., & Granger, D. A. (2012). Sociodemographic risk, parenting, and effortful control: Relations to salivary alpha-amylase and cortisol in early childhood. *Developmental Psychobiology*, 2–12. doi: 10.1002/dev.21079
- Turner-Cobb, J. M., Rixon, L., & Jessop, D. S. (2008). A prospective study of diurnal cortisol responses to the social experience of school transition in four-year-old children: Anticipation, exposure, and adaptation. *Developmental Psychobiology*, 50(4), 377–389. doi: 10.1002/dev.20298
- Walker, C. D. (2010). Maternal touch and feed as critical regulators of behavioral and stress responses in the offspring. *Developmental Psychobiology*, 52(7), 638–650. doi: 10.1002/dev.20492

Williams, J., & MacKinnon, D. P. (2008). Resampling and distribution of the product methods for testing indirect effects in complex models. *Structural Equation Modeling: A Multidisciplinary Journal*, 15(1), 23–51. doi: 10.1080/10705510701758166

Yim, I. S., Quas, J. A., Cahill, L., & Hayakawa, C. M. (2010). Children's and adults' salivary cortisol responses to an identical psychosocial laboratory stressor. *Psychoneuroendocrinology*, 35(2), 241–248. doi: 10.1016/j.psyneuen.2009.06.014