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Biological Sensitivity to Context: The Interactive Effects of Stress Reactivity and Family Adversity on Socioemotional Behavior and School Readiness

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This study examined the direct and interactive effects of stress reactivity and family adversity on socioemotional and cognitive development in three hundred and thirty-eight 5- to 6-year-old children. Neurobiological stress reactivity was measured as respiratory sinus arrhythmia and salivary cortisol responses to social, cognitive, sensory, and emotional challenges. Adaptation was assessed using child, parent, and teacher reports of externalizing symptoms, prosocial behaviors, school engagement, and academic competence. Results revealed significant interactions between reactivity and adversity. High stress reactivity was associated with more maladaptive outcomes in the context of high adversity but with better adaption in the context of low adversity. The findings corroborate a reconceptualization of stress reactivity as *biological sensitivity to context* by showing that high reactivity can both hinder and promote adaptive functioning.

A substantive body of work has established that environmental adversity can have a deleterious effect on children's functioning (Luthar, 2006; Obradović, Shaffer, & Masten, in press; Rutter, 1983; Sameroff, 2006). Exposure to adverse, stressful events, such as marital conflict, maternal depression, and financial stress, has been linked to socioemotional behavior problems and cognitive deficits (Boyce, 2007; Boyce et al., 2001; Burchinal, Roberts,

Hooper, & Zeisel, 2000; Cummings & Davies, 2002; Essex, Klein, Cho, & Kalin, 2002; Masten & Shaffer, 2006). Nevertheless, not all children are equally susceptible to adverse environmental influences. In accordance with the "stress diathesis" hypothesis, behaviorally or biologically reactive children have been traditionally identified as particularly vulnerable to stressful experiences, showing higher levels of behavioral and health problems than their low-reactive peers in the context of environmental risk and adversity (Belsky, Hsieh, & Crnic, 1998; Cummings, El-Sheikh, Kouros, & Keller, 2007; Deater-Deckard & Dodge, 1997; El-Sheikh, 2005; Ramos, Guerin, Gottfried, Bathurst, & Oliver, 2005). In recent years, however, a few researchers have challenged this traditional view, arguing that high reactivity, whether measured at the emotional, behavioral, or biological level, is not a unitary, pathogenic response to adversity that invariably leads to maladaptation. Belsky and colleagues (Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007), for example, have posited that temperamentally

reactive children may show higher susceptibility to environmental influences ''for better and for worse.'' That is, reactive children may be more vulnerable to contextual risk, but they may also show more adaptive responses to interventions (Blair, 2002; Klein Velderman, Bakermans-Kranenburg, Juffer, & van IJzendoorn, 2006). Similarly, Boyce and colleagues have proposed a new theory suggesting that stress reactivity is better conceptualized as a high biological sensitivity to context (Boyce, 2007; Boyce & Ellis, 2005). From this theoretical perspective, children with heightened biological sensitivity to context are predicted to be more vulnerable to negative and stressful contextual factors, but also to have greater capacity to benefit from positive environmental influences. Thus, high biological sensitivity may be maladaptive in the context of adversity but adaptive in the context of a nurturing and supportive environment (Boyce, 1996; Boyce et al., 1995; Boyce et al., 2006; Ellis, Essex, & Boyce, 2005). This work underlines the importance of understanding more fully the biological processes that interact with environmental influences to shape children's adaptation, as indexed by competence and psychopathology (Curtis & Cicchetti, 2003; Masten & Obradović, 2006). The current study was designed to examine how early exposure to family adversity interacts with children's stress reactivity to predict indices of socioemotional behavior, school engagement, and academic competence.

Human Stress Response

Every day, in all human societies, children are exposed to challenging or stressful events from multiple sources. These events range from normative, potentially positive events, such as forming a new peer group; to adverse events, such as witnessing marital conflict; and truly traumatic events that directly threaten children's well-being, such as abuse and neglect. Children respond to these stressors with a set of highly integrated, neurobiological stress responses. A ''fight-or-flight'' response activates the autonomic nervous system (ANS), which initiates, within seconds, an integrated, short-onset repertoire of biobehavioral changes associated with accelerations of heart and respiratory rates, sweat production, and other physiological changes. The ANS comprises both a sympathetic (SNS) branch, which initiates physiological arousal, and a parasympathetic (PNS) branch, which modulates SNS input to the heart and other target organs, regulating recovery and restoring autonomic homeostasis

(Berntson, Cacioppo, & Quigley, 1993). In a lagged response occurring within minutes, activation of the hypothalamic–pituitary–adrenocortical (HPA) axis via glucocorticoid secretion regulates glucose metabolism, blood pressure, and immunity and counterbalances the effects of the integrated ANS stress response (Sapolsky, Romero, & Munck, 2000). There are numerous ways of assessing children's stress reactivity. Because of our interest in whether stress reactivity moderates the effects of adversity on indices of psychopathology and competence, we examined two indicators of stress reactivity that are known to play a role in regulating stress-induced arousal and maintaining homeostasis: (a) respiratory sinus arrhythmia (RSA) and (b) salivary cortisol.

Respiratory Sinus Arrhythmia

RSA is a measure of parasympathetic stress response and refers to high-frequency heart rate variation controlled by efferent fibers of the vagus nerve during the respiratory cycle. Vagal regulation, in the form of increases and decreases in RSA, has been regarded as an index of children's capacity to regulate responses to positive and negative environmental demands (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Porges, 2001, 2003, 2007). High levels of basal RSA have been associated with social competence, empathy, and emotion regulation (Beauchaine, 2001; Eisenberg et al., 1995; Fabes, Eisenberg, & Eisenbud, 1993; Fox & Field, 1989). Low basal RSA, on the other hand, may indicate emotional lability and dysregulation and has been linked to behavior problems in at-risk or clinical samples (Beauchaine, 2001; Beauchaine et al., 2007; Field et al., 1996; Mezzacappa et al., 1997; Pine et al., 1996; Pine et al., 1998). However, it is important to note that recent studies of community, nonclinical samples have found a positive relation, or no significant relation, between resting RSA and externalizing symptoms (Calkins, Graziano, & Keane, 2007; Dietrich et al., 2007).

Further, researchers have examined changes in RSA in response to stressful laboratory challenges. High reactivity, as indexed by decreases in RSA from basal levels (i.e., greater vagal withdrawal), has been associated with more sustained attention, better emotion regulation, and increased engagement during challenging tasks (Calkins, 1997; Suess, Porges, & Plude, 1994). However, in community samples of kindergarten children, higher RSA reactivity to various challenge tasks has been linked to seemingly contradictory indices of adaptation,

such as high levels of sociability (Doussard-Roosevelt, Montogomery, & Porges, 2003) and internalizing symptoms (Boyce et al., 2001). Some of the inconsistencies across studies might be explained by the nature of the sample. For example, low RSA reactivity has been associated with externalizing symptoms in normative samples of young children (Boyce et al., 2001; Calkins et al., 2007), whereas high RSA reactivity in response to emotional stimuli has been observed in children with clinical levels of behavior problems (Crowell et al., 2005).

Only a few studies have examined the effect of RSA in the context of both high and low adversity. Several studies of community samples indicate that high basal RSA levels and high RSA reactivity in response to emotion-evoking stimuli (e.g., an adult argument) buffered children from the effects of marital conflict on behavior problems, academic achievement, and health (El-Sheikh, Harger, & Whitson, 2001; El-Sheikh & Whitson, 2006; Katz & Gottman, 1995, 1997). Only children with low baseline RSA and RSA reactivity were at risk for maladaptation in the context of high marital conflict. However, these findings varied across gender, type of marital conflict (e.g., verbal, physical), and children's outcomes. Similarly, high RSA reactivity to interpersonal stress emerged as a protective factor against the effects of hostile-withdrawn parenting on peer conflict but not on positive indices of peer interactions (Leary & Katz, 2004). In contrast, in a clinical sample, parental psychopathology had a negative effect on children's emotional and behavioral problems only in children with high baseline RSA (Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Studies examining other indices of ANS stress reactivity (i.e., heart rate, blood pressure, skin conductance) in community samples also indicate that children with high stress reactivity may be at risk for social, cognitive, behavioral, and health problems in the context of high adversity but may show better developmental outcomes in the context of low adversity (Boyce, 1996; Boyce et al., 1995; Boyce et al., 2006; Cummings et al., 2007; El-Sheikh, 2005).

In sum, the effect of stress reactivity on adaptation seems to be context dependent, but the directions of reactivity effects across different contexts remain unclear. Further studies are needed to elucidate the role of RSA reactivity in the development of competence and psychopathology, bearing in mind the limitations of previous research. First, many existing studies of RSA reactivity have important methodological flaws, such as a lack of appropriate controls for respiratory rate, attention, and motor activity (Beauchaine, 2001). Second, RSA reactivity has been examined mostly in relation to a single challenge, potentially misrepresenting the comprehensive reactivity to various types of challenges that children may encounter in their daily lives. Third, studies have often focused on a single source of adversity, such as marital conflict, yet children face various kinds of adversities in family environments. Fourth, studies of stress reactivity in general have been largely focused on measures of behavioral and health problems to the exclusion of more positive and prosocial indicators of adaptation.

Cortisol and Adaptive Functioning

Cortisol is the human glucocorticoid hormone secreted by the adrenal glands and passively diffuses into saliva. Salivary cortisol reflects the levels of unbound and biologically active cortisol circulating in the blood. As with RSA, researchers have linked individual differences in both basal and reactive cortisol expression primarily to indices of behavior problems (Gunnar & Vazquez, 2006). In general, in both clinical and community samples, elevated daily cortisol levels have been associated with internalizing symptoms (Goodyer, Herbert, & Althan, 1998; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001), whereas lower levels of daily cortisol have been associated with externalizing symptoms (Hardie, Moss, Vanyukov, Yao, & Kirillovac, 2002; King, Barkley, & Barrett, 1998; McBurnett, Lahey, Rathouz, & Loeber, 2000; McBurnett et al., 1991; Moss, Vanyukov, & Martin, 1995; Shirtcliff, Granger, Booth, & Johnson, 2005). However, in community samples of kindergarten children, basal cortisol secretion was positively related to internalizing symptoms, social wariness, and symptom severity (Essex et al., 2002; Smider et al., 2002).

A few studies have examined cortisol reactivity to specific laboratory challenges. Moderate elevation of cortisol in response to cognitive tasks has been associated with better executive functioning and self-regulation skills in both middle- and low-income samples (Blair, Granger, & Razza, 2005; Davis, Bruce, & Gunnar, 2002). In addition, high cortisol reactivity in response to parent–child conflict tasks has been associated with social problems and internalizing symptoms in clinical samples (Granger, Weisz, & Kauneckis, 1994; Granger, Weisz, McCracken, Ikeda, & Douglas, 1996). However, differences in sample characteristics and stimuli across these studies preclude any conclusions

regarding the relation between cortisol reactivity to different challenges and adaptive functioning.

Studies examining cortisol changes over a day in child care or preschool in community samples have linked elevated cortisol to impulsivity, poor effortful control, peer rejection, and aggression (Dettling, Gunnar, & Donzella, 1999; Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003). Similarly, elevated cortisol in response to starting a new school year has been associated with negative affectivity, and surgency and extraversion (Davis, Donzella, Krueger, & Gunnar, 1999). However, a more complex picture emerged from a study that measured children's cortisol response during the first weeks of school, when children forge new peer groups and connections, and again later in the school year, when peer groups have been fully formed (Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997). Children showing elevated cortisol expression during the first weeks of school and normal levels later in the year were rated as extraverted, socially competent, and outgoing, whereas children who showed high levels of cortisol later in the school year showed more solitary and negative behaviors and were seen as less competent and less outgoing.

Although these studies indicate that the relation between cortisol reactivity and adaptation may be context dependent and may vary with levels of contextual stress, there is a paucity of studies examining the interactive effects of cortisol and adversity on adaptation. We are aware of only one such study, which shows that cortisol reactivity is related to symptom severity only for children with low father involvement in infancy (Boyce et al., 2006). Moreover, studies examining cortisol response to various age-appropriate stressors and in the context of overall family adversity are rare. Thus, it is unknown whether cortisol reactivity may diminish or amplify the effects of adversity on children's adaptation. In order to better understand the regulatory function of cortisol, researchers need to examine whether cortisol reactivity interacts with contextual factors in predicting both positive and negative developmental outcomes.

Current Study

The main goal of this study was to investigate how the interplay between children's stress reactivity and overall family adversity influences adaptation. We focused our study on kindergarten children because understanding processes that subserve adaptation during the salient developmental transition to a school setting is very important, given the reverberatory effects that early behavior problems or academic failure can have on later developmental trajectories. Consequently, we examined adaptation across four domains of functioning known to contribute to early school success and later adjustment: (a) externalizing symptoms, (b) prosocial behaviors toward peers, (c) school engagement, and (d) academic competence.

Based on the broad literature on risk and adversity, we hypothesized a robust negative main effect of family adversity across all indices of adaptation. We also expected to find main effects of stress reactivity on adaptation, but given the simultaneous test of interactive effects, as well as the paucity of studies examining the effects of RSA and cortisol reactivity, especially for positive developmental outcomes, and some inconsistencies within such studies, we did not hypothesize the directions of these main effects. More importantly, in accordance with the theory of biological sensitivity to context (Boyce & Ellis, 2005; Ellis et al., 2005), we expected to find evidence that ANS and HPA reactivity moderate the effects of early family adversity on various domains of functioning. We hypothesized that in high-adversity family environments, elevated levels of stress reactivity would be associated with maladaptive outcomes, whereas low stress reactivity would act as a protective factor. In the context of low family adversity, on the other hand, we expected high levels of reactivity to be associated with better adaptation. It is important to note that although biological sensitivity to context should be examined in both positive and negative settings, our assessment focuses on six types of family adversities, and a lack of overall family adversity does not necessarily imply a supportive and nurturing environment. In addition to the hypothesized Adversity × Stress Reactivity interactions, we controlled for children's sex and tested whether main and interactive effects of adversity and reactivity vary across sex.

Method

Participants

The sample included 338 children (163 females, 175 males) who participated in a longitudinal study of social status, biological responses to adversity, and child mental and physical health. Participants were recruited in three waves from 29 kindergarten classrooms within six public schools in the San Francisco Bay Area (Oakland, Albany, and

Piedmont Unified school districts) during the falls of 2003, 2004, and 2005. Schools were selected to represent a variety of sociodemographic and ethnic characteristics of the metropolitan area. Families were recruited through a series of efforts that included home mailings, presentations at kindergarten parent welcome nights, and in-person recruitment during drop-off and pick-up. Every family with a child in the target classrooms was invited to participate. However, families who were not fluent in either English or Spanish were excluded to ensure adequate comprehension of study questionnaires. Schools were provided with $20 per child enrolled in the study.

The mean age of the children at the kindergarten entry was 5.32 years (SD = 0.32, range = 4.75–6.28). The sample was ethnically diverse, and children were identified as 19% African American, 11% Asian, 43% European or White, 4% Latino, 22% multiethnic, and 2% as ''other'' ethnicity. Family demographic information was not provided by 16 families. Primary caregivers were 87% biological mothers, 9% biological fathers, 2.5% adoptive mothers, 0.6% biological grandmothers, and 0.9% ''other'' relations. As the vast majority of primary caregivers were parents, these terms are used interchangeably here. Average annual household income ranged from < $10,000 to > $400,000 (M = $60,000–79,999; Mdn = $80,000–99,999). Highest level of educational attainment in the household ranged from less than a high school diploma to advanced degrees, with 75% of caregivers having at least a college degree.

Procedures

Data for this study were collected in the fall (Time 1) and spring (Time 2) during the kindergarten year. Parents' informed consent and children's assent were obtained prior to the start of data collection. Parent report of family adversity and children's functioning was collected through a series of home mailings, and families were compensated with $50 for each completed time point. Teacher report of children's functioning was collected through questionnaires dropped off and picked up at the child's school, and teachers were compensated $15 per child for each report returned. Self-report of children's functioning was collected through a series of structured, scripted interviews conducted in a separate, quiet room at elementary schools.

In addition, children participated in the 20-min reactivity protocol, which included four age-appropriate tasks designed to elicit stress responses to social, cognitive, sensory, and emotional challenges (Alkon et al., 2003; Boyce et al., 1995). Since PNS can be activated by psychomotor activity, such as gesturing, speaking, focused attending, and nonchallenging social engagement (Bazhenova & Porges, 1997; Bernardi et al., 2000; Berntson, Cacioppo, & Fieldstone, 1996; Porges, 2001; Porges et al., 2007), RSA assessed during a challenge task reflects both stress reactivity in response to the challenge and reactivity in response to the psychomotor or engagement demands of the challenge task. The recent literature has emphasized the importance of maximizing the mobilization of the psychological response to the particular challenge and minimizing the peripheral triggers of cardiovascular activation (Bush, Alkon, Obradović, Stamperdahl, & Boyce, 2009; Kamarck & Lovallo, 2003). Thus, in order to best measure the psychophysiological response to the four challenges, each challenge, task in the reactivity protocol was preceded by a nonchallenging ''control task'' that paralleled the motor and engagement demands of that challenge, and levels of autonomic arousal during the control tasks were used as baseline values.

The autonomic reactivity protocol started with an experimenter reading a calming short story (2 min). Next, the child completed four sets of paired tasks, each consisting of a control condition and challenge condition. The *social challenge task* (2 min) was a structured interview about the child's family, friends, and likes/dislikes adapted from the Gesell School Readiness Screening Test (Carlson, 1985). This was preceded by the *social control task* in which children were asked to name common animals and colors from a picture book to capture arousal associated with speaking, gestures during social speech, and attention involved in social engagement (2 min). The *cognitive challenge task* (2 min) was a digit span recitation task derived from the Kaufman Assessment Battery for Children (Kaufman & Kaufman, 1983) in which children were asked to recall sequences of numbers up to six digits in length and received negative verbal feedback after making a mistake. This was preceded by the *cognitive control task* in which children were asked to repeat simple, one- or two-digit number sequences to capture arousal associated with listening, speaking numbers, and social engagement (1 min). The *sensory challenge task* (1 min) was a taste-identification task (Kagan & Snidman, 1991) during which children were asked to identify two drops of concentrated lemon juice placed on their tongues. This was preceded by the

sensory control task in which children were asked to identify two drops of water placed on their tongues to capture arousal associated with mouth opening and swallowing, anticipation, and guessing the content of the liquid (1 min). The *emotional challenge task* (2 min) consisted of watching a short emotion-evoking movie clip chosen to elicit fear (Eisenberg et al., 1988). This was preceded by the *emotion-control task* in which children were asked to watch an emotionally neutral movie clip to capture physiological responding associated with attending to visual stimuli (2 min). The autonomic reactivity protocol concluded with the reading of another calming story (2 min).

Measures

Stress reactivity. Children's stress reactivity was assessed using changes in both RSA and *salivary cortisol* during the stress reactivity protocol. After the child was familiarized with the equipment, four spot electrodes (two current, two impedance) were placed in the standard tetrapolar configuration on the child's neck and chest, and ECG electrodes were placed on the right clavicle and lower left rib. A 4 mA AC current at 100 kHz was passed through the two current electrodes. RSA was monitored continuously during the protocol. Data were acquired using the Biopac MP150 (Biopac Systems, Santa Barbara, CA) interfaced to a PC-based computer. Analog data were continuously monitored on the computer for signal and noise, and digitized data were stored for offline analysis. RSA was derived in accordance with recommendations of the Society for Psychophysiological Research committee on heart rate variability (Berntson et al., 1997). The sampling frequency was 1 kHz. Prior to analyses, each waveform was verified, IBIs were visually checked, and artifacts were identified using Berntson, Quigley, Jang, and Boysen's (1990) algorithm within the MindWare software program (http://www.mindwaretech.com). RSA was estimated as the natural logarithm of the variance of heart period within the high-frequency bandpass associated with respiration at this age (i.e., 0.15–0.80 Hz; Bar-Haim, Marshall, & Fox, 2000; Rudolph, Rudolph, Hostetter, Lister, & Siegel, 2003). Outlier data were checked and verified minute by minute if they were > 3 SD from the group mean. Mean RSA magnitude was calculated for each 1-min interval and averaged within each task (Cacioppo, Uchino, & Berntson, 1994).

RSA reactivity responses during each of the control tasks were used as baseline reference values to create four task RSA reactivity scores. To control for the influence of baseline levels of arousal, we created four standardized residual scores as measures of reactivity to social, cognitive, sensory, and emotional challenges by regressing RSA values during the challenge tasks on the respective control task RSA values. Residuals reflect the extent to which an individual's physiologic response deviated from the regression line derived from sample responses (Manuck, Kasprowicz, & Muldoon, 1990). The four standardized residual scores were then averaged to create one index of RSA stress reactivity. Elevated RSA reactivity was represented by negative residual scores, indicating a decrease in RSA and vagal withdrawal in response to a given challenge. Conversely, low reactivity was represented by positive residual scores, indicating an increase in RSA and vagal activation in response to the challenge.

Descriptive statistics for RSA responses to tasks are presented in Table 1. Average RSA differences between challenge and control tasks were −0.25 (SD = 0.66) for the social challenge, 0.60 (SD = 0.67) for the cognitive challenge, −0.32 (SD = 0.84) for the sensory challenge, and 0.10 (SD = 0.47) for the emotional challenge task. RSA reactivity levels were significantly different from zero during the social (t = −6.99, $p < .001$), cognitive (t = 16.40, $p < .001$), sensory (t = −7.05, $p < .001$), and

Table 1

Descriptive Statistics for Stress Reactivity and Adversity Variables

Variable	Scale	Min.	Max.	M	SD
RSA					
Social control		3.29	9.65	6.81	1.12
Social challenge		3.47	10.26	6.56	1.10
Cognitive control		2.62	9.69	6.12	1.20
Cognitive challenge		3.35	9.82	6.72	1.10
Sensory control		2.77	9.91	6.98	1.13
Sensory challenge		3.04	10.90	6.66	1.20
Emotional control		2.74	10.15	7.03	1.12
Emotional challenge		2.48	10.17	7.13	1.10
Cortisol					
Preprotocol		0.10	24.40	5.10	3.51
Postprotocol		0.10	30.50	4.70	3.93
Adversity					
Financial stress	1–5	1.00	5.00	2.42	0.93
Parenting overload	1–5	1.20	5.00	3.12	0.68
Marital conflict	1–5	1.00	3.10	1.74	0.38
Family expressiveness	1–9	1.20	7.20	4.03	0.99
Anger expression	0–10	0.25	6.13	2.42	0.84
Maternal depression	1–4	1.00	3.00	1.37	0.30
Harsh/restrictive parenting	1–7	1.76	6.56	3.66	0.75

emotional ($t = 4.04$, $p < .001$) tasks. Averaged across the four challenge tasks, 47.1% of the sample showed a decrease in RSA from the levels elicited by the control tasks, suggesting that about half of the sample showed vagal withdrawal in response to challenges. Thus, the reactivity protocol effectively captured variability in child RSA responses to challenges.

At the beginning and end of the reactivity protocol, salivary cortisol was collected by instructing the child to chew on a cotton roll for 20–30 s. Wet cotton rolls were deposited in salivette tubes and stored at −7 °C until assayed. The cortisol samples were assayed using a commercial immunoassay with chemiluminescence detection (Cortisol Luminescence Immunoassay; IBL-Hamburg, Hamburg, Germany). The detection limit of the assay was 0.41 nmol/L. The mean interassay and intra-assay variations were 8.5% and 6.1%, respectively. Cortisol values above 55 nmol/L (< 1% of samples) were considered unreliable data and were discarded. Ten children in this sample were taking medications, such as human growth hormone and exogenous glucocorticoids, known to alter salivary cortisol levels (Masharani et al., 2005). These children were excluded from analyses of cortisol reactivity. Given that cortisol levels in saliva reach their peak approximately 15–20 min following stressor onset, cortisol values collected at the beginning of the session were considered baseline reference values, as they indexed children's cortisol expression in the familiar context of the kindergarten classroom. The average session lasted 27 min ($SD = 3$ min, range = 19–38 min). Thus, cortisol values collected at the end of the session were considered a measure of reactivity to a novel, mildly stressful context (e.g., strange experimenter, electrodes, challenge tasks), at the midpoint of the reactivity protocol. Descriptive statistics for pre- and postprotocol values are presented in Table 1. We created standardized residual scores by regressing postprotocol cortisol values on preprotocol, baseline values. Because cortisol collection occurred both in the morning and in the afternoon, we examined whether cortisol reactivity varied across the day, due to the known circadian activity of the HPA system. The time of collection was related to absolute preprotocol values ($r = .18$, $p < .01$) but not to postprotocol values and cortisol reactivity as indexed by standardized residual scores, which is consistent with the current literature (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Length of the session also did not significantly relate to cortisol reactivity. Mean cortisol levels pre- and postprotocol were

5.10 nmol/L ($SD = 3.51$) and 4.70 nmol/L ($SD = 3.93$), respectively. Mean difference between pre- and postprotocol values was −0.40 nmol/L ($SD = 3.81$). Positive cortisol reactivity values indicate a stress response, and 37.5% of the sample showed such a response to the challenge protocol. The average difference between pre- and postprotocol values for the responder group was 2.58 nmol/L ($SD = 3.86$).

Adversity. Children's exposure to adversity was assessed by six indices of potential sources of family stress. All measures of adversity were based on parent report, and reliability statistics are reported for the current sample. *Financial Stress* was assessed with four items derived from Essex et al. (2002) that measured parents' thoughts about money problems, difficulty paying bills, and limited opportunities due to lack of finances ($\alpha = .81$) *Parenting Overload* was assessed with five items derived from Essex et al. that measured feelings of being overwhelmed with parenting duties, juggling conflicting obligations, and lacking time to rest or pursue desired activities ($\alpha = .79$). *Marital Conflict* was assessed using the 10-item O'Leary–Porter Overt Hostility Scale ($\alpha = .72$) that measured how often parents openly argue, display physical and verbal hostility, and criticize each other in the presence of their children (Johnson & O'Leary, 1987; Porter & O'Leary, 1980). Exposure to *Negative/Anger Expressiveness* in the family was assessed using both the Family Expressiveness Questionnaire (FAQ; Halberstadt, 1986) and the Anger Expression Inventory (AEI; Spielberger, 1988). The FAQ consists of a 10-item negative dominant subscale ($\alpha = .83$), measuring the frequency of overt anger, contempt, and hostility among family members, and a 10-item negative subdominant subscale ($\alpha = .75$), measuring the frequency of passive sulking, crying, and disappointment among family members. The two FAQ subscales were averaged ($r = .55$, $p < .001$) to yield one measure of negative family expressiveness. The total AEI score was calculated using three 8-item subscales that assessed parents' tendency to express overtly towards other people ($\alpha = .69$), hold angry feelings inside ($\alpha = .68$), and control the experience and expression of anger ($\alpha = .74$). The overall scores based on FAQ and AEI were standardized and averaged ($r = .47$, $p < .001$) into one indicator of exposure to negative/anger expressiveness. *Maternal Depression* was assessed with a 20-item Center for Epidemiological Studies Depression Scale (CES–D; $\alpha = .81$; Radloff, 1977). *Harsh and Restrictive Parenting* was assessed using a questionnaire version of Child-Rearing Practice Report

(CRPR; Block, 1965). The 18-item scale (α = .83) was based on previous studies (Deković, Janssens, & Gerris, 1991; Rickel & Biasatti, 1982). Descriptive statistics for adversity measures are shown in Table 1. As we were interested in capturing children's overall exposure to family adversity, the six indices of adversity were standardized and composited into one adversity index. Although a few adversity measures included positive items, which provided some assessment of positive family environment (e.g., financial security, parental affection, parent who is happy, has time to relax, shows patience, and controls anger), it is important to note that, low levels of adversity on this measure do not necessarily indicate a supportive family environment.

Adaptive functioning. Adaptive functioning was assessed during the fall and spring of the kindergarten year using parent-, teacher-, and child-reported measures. For the purposes of this study, we focused on four developmentally salient domains—externalizing symptoms, prosocial behaviors, school engagement, and academic competence—which are described in more detail next. Parent and teacher perceptions of the child's behaviors and skills across these domains were assessed using The MacArthur Health and Behavior Questionnaire (HBQ; Armstrong, Goldstein, & the MacArthur Working Group on Outcome Assessment, 2003). Parent and teacher forms of the HBQ contain parallel scales, described in detail next. Children's perceptions of their own behavior and skills across four domains were assessed using the Berkeley Puppet Interview, which is designed to elicit children's responses to statements that parallel the parent and teacher HBQ scales (BPI; Ablow, Measelle, & the MacArthur Working Group on Outcome Assessment, 2003). The BPI combines both structured and clinical interviewing techniques and allows young children to respond either verbally or nonverbally to opposing statements issued by two puppets about their behavior. Interviewers were extensively trained to probe for more information, to deal with unclear, inaudible, indecisive, or alternate responses, and to create a comfortable, engaging, and interactive environment. Videotaped responses were coded using a 7-point scale based on the statement the child chose to endorse and the degree of endorsement. Interclass correlation coefficients (ICC) were calculated based on 35% of double-coded fall BPIs (ICC range = .91–.98) and 29% of double-coded spring BPIs (ICC range = .92–.99), indicating excellent interrater reliability. Individual items were averaged into

subscales and scales according to the BPI manual. The validity and reliability of HBQ and BPI measures have been tested with children ages 5–6, and all three measures show good measurement properties (for details see Ablow et al., 2003; Armstrong et al., 2003).

All three measures contained the same scales. *Externalizing Symptoms* were assessed using an Oppositional Defiant scale (e.g., argues, angry, resentful, misbehaves), Conduct Problem scale (e.g., lies, vandalizes, threatens, cheats), and an Overt Hostility scale (e.g., teases, fights, kicks, taunts). Preliminary analyses revealed that indices of stress reactivity were related differently to a fourth Relational Aggression scale in comparison to the other three scales of externalizing symptoms. We believe that this finding deserves further investigation outside the scope of this study; we thus omitted the Relational Aggression scale from our measure of Externalizing Symptoms. *Prosocial Behaviors* were assessed using a scale measuring the child's predisposition to help others, share, invite bystanders to play, and be considerate, collaborative, fair and empathic. *School Engagement* was assessed using a scale measuring the extent to which the child was excited, eager, happy, frustrated, bored, or irritable about school. *Academic Competence* was assessed with a scale indexing the child's achievement, innate ability, and difficulties in literacy and math, as well as the child's overall academic performance in the classroom in comparison to other classmates. The sample's descriptive and reliability statistics for scales and domains across three informants and two time periods are presented in Table 2.

Using HBQ and BPI data, we conducted eight principal component analyses (PCA) to obtain multi-informant indices of externalizing symptoms, prosocial behaviors, school engagement, and academic competence, separately for the fall and spring assessments. We used a procedure described by Kraemer et al. (2003) for integrating data from multiple informants by triangulating three potentially orthogonal sources of information (i.e., parent, teacher, child). Following this approach, we used parent, teacher, and child report to extract three components for each PCA that conceptually correspond to the following dimensions: (a) trait (i.e., individual differences in adaptation), (b) perspective (i.e., informant characteristics that influence the assessment of the trait), and (c) context (i.e., environmental features that influences expression of the trait). For the purposes of this study, we used only factor scores based on the ''trait'' component, which weighs each source of information in

Table 2
Adaptive Functioning Measures

Domains/scales	Scale	No. items	Fall				Spring			
			M	*SD*	α	λ	*M*	*SD*	α	λ
Externalizing behavior problems										
P–HBQ			0.29	0.21	.78	.71	0.30	0.23	.80	.75
Oppositional defiant	0–2	9	0.44	0.31	.77		0.46	0.33	.80	
Conduct problems	0–2	12	0.13	0.16	.70		0.14	0.18	.77	
Overt hostility	0–2	4	0.27	0.27	.56		0.28	0.30	.66	
T–HBQ			0.19	0.30	.89	.71	0.26	0.36	.91	.76
Oppositional defiant	0–2	9	0.21	0.33	.88		0.29	0.41	.92	
Conduct problems	0–2	11	0.10	0.22	.73		0.14	0.26	.88	
Overt hostility	0–2	4	0.25	0.41	.82		0.33	0.47	.85	
C–BPI			2.40	0.55	.81	.67	2.36	0.51	.79	.73
Oppositional defiant	1–7	6	2.46	0.64	.49		2.41	0.63	.53	
Conduct problems	1–7	9	2.39	0.60	.65		2.35	0.55	.65	
Overt hostility	1–7	7	2.32	0.65	.72		2.30	0.64	.69	
Prosocial behaviors										
P–HBQ	0–2	20	1.31	0.31	.86	.68	1.29	0.32	.88	.70
T–HBQ	0–2	20	1.22	0.48	.95	.64	1.25	0.51	.95	.80
C–BPI	1–7	7	5.34	0.83	.64	.74	5.42	0.79	.67	.59
School engagement										
P–HBQ	1–4	8	3.73	0.35	.83	.65	3.65	0.42	.87	.75
T–HBQ	0–2	8	1.80	0.28	.81	.68	1.78	0.30	.84	.65
C–BPI	1–7	8	5.24	1.03	.80	.76	5.27	0.93	.77	.71
Academic competence										
P–HBQ	1–7	8	5.01	1.03	.92	.85	5.19	1.03	.93	.89
T–HBQ	1–5	5	3.34	0.80	.95	.75	3.58	0.92	.97	.86
C–BPI	1–7	12	5.13	0.80	.71	.53	5.16	0.75	.73	.52

Note. λ = factor loading; C–BPI = child report on Berkeley Puppet Interview; P–HBQ = parent report on Health and Behavioral Questionnaire; T–HBQ = teacher report on Health and Behavior Questionnaire.

the same direction, as an overall core measure of each adaptive domain. Thus, each adaptive domain (e.g., externalizing) is measured by standardized factor scores that represent the common variance across three reports of the domain and are largely free of informant and contextual effects. Table 2 shows PCA loadings for the first (trait) component of each adaptation domain in fall and spring.

Data Preparation and Analytic Procedures

Missing data for the six adversity components, the BPI and HBQ scales, pre- and postprotocol cortisol values, and RSA values to the four challenge and four control tasks were handled using the recommended maximum-likelihood estimation procedure for missing data, specifically, the expectation-maximization (EM) algorithm (Schafer & Graham, 2002). Percentages of missing data were as follows: fall teacher report (0.0%), fall child report (0.9%–1.5%), fall parent report (13.3%–13.9%),

spring teacher report (3.0%), spring child report (3.6%–4.4%), spring parent report (16.9%–17.5%), adversity components (11.8%–13.0%), RSA values (3.6%–4.7%), and cortisol values (7.7%–9.8%). Extreme values on standardized final composites were truncated to −3.50 or 3.50 standardized values. All analyses were conducted using the SPSS 15.0 program (SPSS, Chicago). All hierarchical multiple regression analyses were conducted controlling for the child's sex and age at kindergarten entry.

Results

Bivariate Correlations

Bivariate correlations among key variables included in this study are presented in Table 3. Age at kindergarten entry was not related to indices of stress reactivity, adversity, or adaptation, and thus was not included in further analyses. Boys in

Table 3

Bivariate Correlations Among Age, Gender, Stress Reactivity, and Adaptive Functioning

	1	2	3	4	5	6	7	8	9	10	11	12
	Age	Sex	RSA	CORT	ADV	EXT–F	PRO–F	SE–F	ACA–F	EXT–S	PRO–S	SE–S
1 Age	—											
2 Sex	−.16**	—										
3 RSA	.02	.00	—									
4 CORT	−.06	.00	−.02	—								
5 ADV	.02	−.15**	.05	−.02	—							
6 EXT–F	.05	−.27***	.12*	.14*	.29***	—						
7 PRO–F	.00	.35***	.06	−.03	−.09	−.50***	—					
8 SE–F	.00	.21***	−.04	−.13*	−.25***	−.52***	.45***	—				
9 ACA–F	.00	.14**	−.07	−.09	−.02	−.17**	.38***	.35***	—			
19 EXT–S	.07	−.31***	.08	.10	.31***	.75***	−.46***	−.46***	−.14**	—		
11 PRO–S	−.01	.39***	.07	−.06	−.19***	−.48***	.71***	.43***	.24***	−.61***	—	
12 SE–S	.03	.19***	.00	−.14*	−.26***	−.47***	.44***	.59***	.22***	−.55***	.54***	—
13 ACA–S	.04	.11*	−.05	−.09	.01	.13*	.31***	.28***	.74***	−.16**	.28***	.30***

Note. ACA = academic competence; CORT = Cortisol reactivity; EXT = externalizing symptoms; F = fall; PRO = prosocial behavior; RSA = respiratory sinus arrhythmia reactivity; S = spring; SE = school engagement.
*p < .05. **p < .01. ***p < .001.

this sample were older than girls, experienced higher levels of adversity, and showed poorer adaptation across all domains. RSA reactivity was significantly related only to fall externalizing, with low reactive children exhibiting higher levels of symptoms. Cortisol reactivity was significantly related to fall externalizing symptoms and to both fall and spring school engagement, with high-reactive children exhibiting higher levels of symptoms and lower levels of engagement. Adversity exposure was related to higher externalizing symptoms and lower school engagement across both time points, as well as to lower prosocial behaviors in spring. Lastly, all domains of adaptation showed significant within-time correlations with other domains, as well as significant longitudinal stability from fall to spring.

Regression Analyses

The main goal of the regression analyses was to examine the interactive effects of adversity and stress reactivity on indices of adaptive functioning. While we also tested the main effects of family adversity and stress reactivity on adaptation, these direct effects can only be interpreted in the context of the significant interactive effects that qualify them. For each measure of stress reactivity (i.e., RSA and cortisol), two sets of regression analyses were conducted to predict (a) concurrent fall adaptation and (b) longitudinal change in adaptation by

predicting spring adaptation while controlling for fall adaptation. Interactive effects were tested following the procedure outlined by Baron and Kenny (1986). Significant interactions were further investigated using the simple slopes technique proposed by Aiken and West (1991) by comparing the effect of high (i.e., 1 *SD* above the mean) and low (i.e., 1 *SD* below the mean) adversity in children with high and low stress reactivity. High RSA reactivity (i.e., vagal withdrawal) was defined as 1 *SD* below the mean, while low RSA reactivity was defined as 1 *SD* above the mean. In contrast, high cortisol reactivity was defined as 1 *SD* above the mean, and low cortisol reactivity was defined as 1 *SD* below the mean. Finally, we examined whether these main and interactive effects varied for boys and girls.

RSA reactivity. Results of regression analyses examining the effects of RSA reactivity and adversity on adaptation are presented in Table 4. Compared to boys, girls showed better adaptive functioning across all four domains in fall, as well as a decrease in externalizing symptoms and increase in prosocial behaviors across the 1st year of kindergarten. Lower RSA reactivity, as indexed by positive RSA residual values, significantly predicted higher levels of externalizing in fall. Higher adversity exposure predicted higher levels of externalizing and lower school engagement in fall. In addition, adversity exposure predicted an increase in externalizing symptoms as well as a decrease in

Table 4

Standardized Regression Coefficients for Models Testing the Effects of RSA Reactivity and Adversity on Adaptation

	Externalizing		Prosocial behavior		School engagement		Academic competence	
	Fall	Spring	Fall	Spring	Fall	Spring	Fall	Spring
Fall adaptation		.69***		.64***		.53***		.73***
Sex	−.22***	−.11**	.33***	.15***	.17**	.06	.13*	.00
RSA	.11*	−.01	.08	.04	−.03	.02	−.04	.02
ADV	.25***	.10**	−.05	−.11**	−.23***	−.12**	−.02	.00
RSA × ADV	−.11*	−.01	.11*	.03	.12*	.05	.07	.13***
ADV × Sex	−.07	−.06	.01	.02	.09	.03	−.10	.01
RSA × Sex	.03	−.05	.02	.05	.02	.00	.10	.08*
RSA × ADV × Sex	.02	−.03	.03	.01	−.02	−.08	−.03	.01
Total R^2	.17***	.60***	.14***	.54***	.12***	.37***	.05*	.57***

Note. ACA = academic competence; ADV = adversity; EXT = externalizing behavior problems; PRO = prosocial behavior; RSA = respiratory sinus arrhythmia reactivity; SE = school engagement.
*$p < .05$. **$p < .01$. ***$p < .001$.

prosocial behavior and school engagement from fall to spring. However, these main effects were qualified by significant interactions.

The interaction effect between RSA reactivity and adversity significantly predicted fall adaptation across three of four domains: externalizing symptoms, prosocial behaviors, and school engagement. Examination of simple slopes revealed that the effect of adversity exposure on adaptation varied across different levels of RSA reactivity. A stronger relation between adversity and externalizing symptoms was found for children with high RSA reactivity ($b = .35$, $p < .001$) than for those with low RSA reactivity ($b = .15$, $p < .05$). In the context of high adversity, both low- and high-reactivity groups showed elevated externalizing symptom levels, whereas in the context of low adversity exposure, high-reactive children had lower levels of symptoms than low reactive children (see Figure 1). Similarly, adversity exposure was significantly related to prosocial behavior in high-reactive children ($b = −.16$, $p < .05$) but not in low-reactive children ($b = .05$, $p > .05$). However, contrary to findings for externalizing symptoms, high-reactive children showed lower levels of prosocial behaviors than low-reactive children in the context of high adversity but did not differ in the context of low adversity (see Figure 2). Finally, adversity exposure was significantly related to fall school engagement in high-reactive children ($b = −.33$, $p < .001$) but not in low-reactive children ($b = −.12$, $p > .05$). In comparison to low-reactive peers, high-reactive children showed lower levels of fall school engagement in the context of high adversity but higher levels of

school engagement in the context of low adversity (see Figure 3). High reactivity thus operated as a risk factor under conditions of high adversity but as a promotive, engagement-enhancing factor in circumstances of low adversity.

Although the interaction between RSA reactivity and adversity exposure did not predict fall levels of academic competence, it did predict change in academic competence from fall to spring. Analyses of simple slopes revealed that exposure to adversity was related to an increase in academic competence in low-reactive children ($b = .12$, $p < .05$) but to a
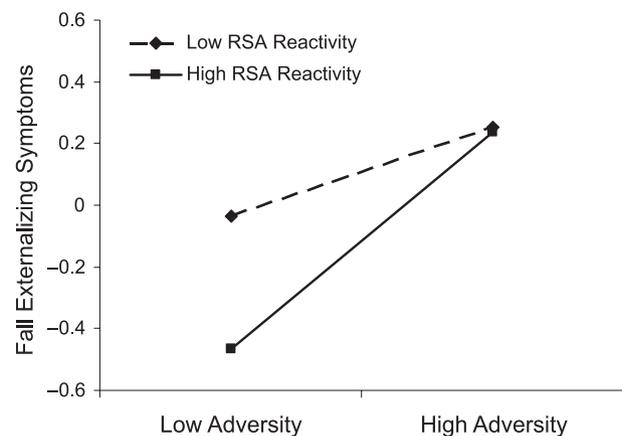

Figure 1. Fall externalizing symptoms as a function of adversity exposure and RSA reactivity.
Note. Low and high adversity values are graphed at 1 *SD* below and above the mean, respectively, whereas low and high RSA reactivity are graphed at 1 *SD* above and below the residual score mean, respectively. RSA = respiratory sinus arrhythmia.
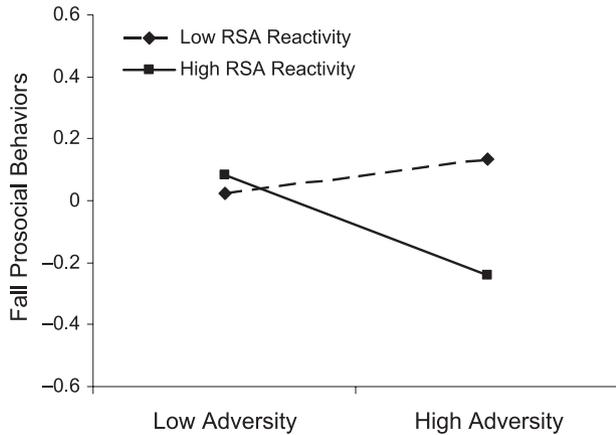
Figure 2. Fall prosocial behaviors as a function of adversity exposure and RSA reactivity.
Note. Low and high adversity values are graphed at 1 *SD* below and above the mean, respectively, whereas low and high RSA reactivity are graphed at 1 *SD* above and below the residual score mean, respectively. RSA = respiratory sinus arrhythmia.

decrease in academic competence in high-reactive children ($b = -.12$, $p < .05$). In the context of high adversity, low-reactive children showed higher levels of improvement in academic competence than high-reactive children (see Figure 4). However, in the context of low adversity, the reverse was true, with high-reactive children showing higher levels of improvement in academic competence than their low-reactive peers.
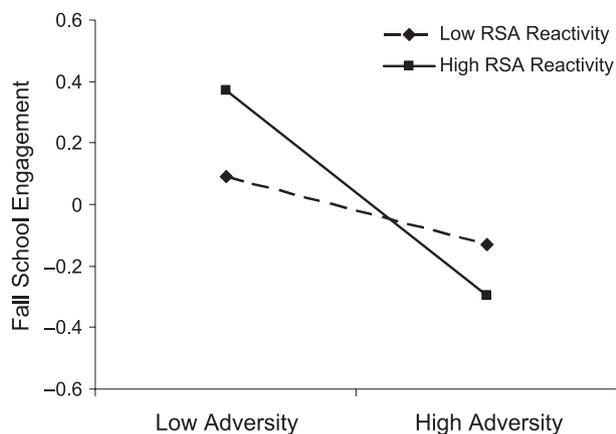
The interaction effects of reactivity and adversity on different domains of adaptation did not vary across children's sex, as the three-way interaction effects between sex, reactivity, and adversity were not significant (see Table 4). However, a significant two-way interaction revealed that the main effect of RSA reactivity on 6-month change in academic competence varied for boys and girls. The test of simple slopes indicated a crossover effect such that high reactivity was related to academic improvement in boys ($b = -.06$, $p > .05$), whereas low reactivity was related to academic improvement in girls ($b = .10$, $p > .05$); however, neither slope reached statistical significance.

Cortisol reactivity. Results of regression analyses examining the effects of RSA reactivity and adversity on adaptation are presented in Table 5. High cortisol reactivity significantly predicted higher levels of externalizing and lower levels of school engagement and academic competence in fall. Adversity effects were the same as in regression analyses focused on RSA reactivity. However, these main effects are qualified by the significant interactions.

The interaction between cortisol reactivity and adversity significantly predicted fall prosocial behavior. According to tests of simple slopes, adversity exposure was significantly related to fall prosocial behaviors for high-reactive children ($b = -.16$, $p < .05$) but not in low-reactive children ($b = .06$, $p > .05$). In comparison to low-reactive peers, high-reactive children showed lower levels
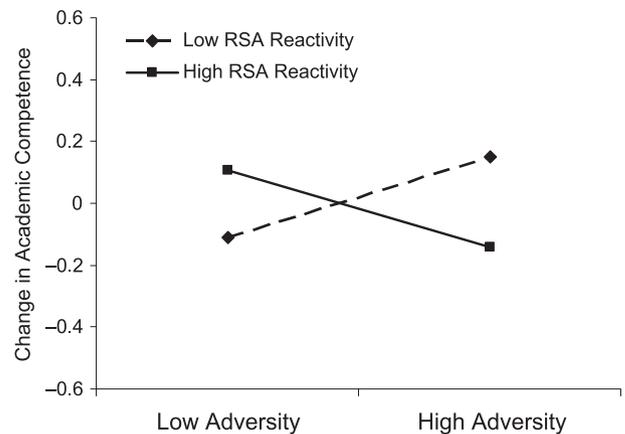

Figure 3. Fall school engagement as a function of adversity exposure and RSA reactivity.
Note. Low and high adversity values are graphed at 1 *SD* below and above the mean, respectively, whereas low and high RSA reactivity are graphed at 1 *SD* above and below the residual score mean, respectively. RSA = respiratory sinus arrhythmia.

Figure 4. Change in academic competence as a function of adversity exposure and RSA reactivity.
Note. Low and high adversity values are graphed at 1 *SD* below and above the mean, respectively, whereas low and high RSA reactivity are graphed at 1 *SD* above and below the residual score mean, respectively. RSA = respiratory sinus arrhythmia.

Table 5

Standardized Regression Coefficients for Models Testing the Effects of Cortisol Reactivity and Adversity on Adaptation

	Externalizing		Prosocial behavior		School engagement		Academic competence	
	Fall	Spring	Fall	Spring	Fall	Spring	Fall	Spring
Fall adaptation		.70***		.65***		.54***		.75***
Sex	−.23***	−.11**	.32***	.15***	.17**	.05	.13*	.01
CORT	.14**	.00	−.02	−.05	−.13*	−.08	−.11*	−.02
ADV	.25***	.09*	−.05	−.09*	−.24***	−.10*	.00	.03
CORT × ADV	.05	.04	−.13*	−.05	−.04	−.06	−.08	.03
ADV × Sex	−.04	−.07*	.02	.03	.11*	.01	−.08	.00
CORT × Sex	−.12*	.04	.02	−.02	.07	.03	.07	.00
CORT × ADV × Sex	−.03	−.01	.09	−.03	.02	−.04	−.06	.00
Total R^2	.18***	.59***	.14***	.56***	.14***	.39***	.06**	.57***

Note. ACA = academic competence; ADV = adversity; CORT = cortisol reactivity; EXT = externalizing behavior problems; PRO = prosocial behavior; SE = school engagement.
*$p < .05$. **$p < .01$. ***$p < .001$.

of fall prosocial behavior in the context of high adversity but in the context of low adversity showed slightly higher levels of prosocial behavior (see Figure 5).

The interaction effect of cortisol reactivity and adversity did not vary across children's sex, as indicated by nonsignificant three-way interactions (see Table 5). However, significant two-way interactions between sex and adversity emerged for fall school engagement and change in externalizing from fall to spring. Tests of simple slopes indicate that adversity exposure was significantly related to lower school engagement in boys ($b = .34$, $p < .001$) but not in girls ($b = −.13$, $p > .05$). Likewise, adver-

sity exposure was related to an increase in externalizing symptoms in boys ($b = .16$, $p < .01$) but not in girls ($b = .01$, $p > .05$). In addition, the main effect of cortisol reactivity on fall externalizing differed for boys and girls, as indicated by a significant two-way interaction between reactivity and sex in Table 5. Cortisol reactivity was related to higher levels of externalizing in boys ($b = .31$, $p < .001$) but not in girls ($b = .03$, $p > .05$).

Discussion

The main goal of this study was to examine how stress reactivity and family adversity influence socioemotional behavior and school readiness skills in children attending kindergarten. Several significant main effects emerged. Consistent with the broad literature on the effects of risk and adversity (e.g., Luthar, 2006), family adversity was associated with concurrent externalizing symptoms and lower school engagement, and with increases in externalizing symptoms and decreases in prosocial behaviors and school engagement over the kindergarten year. However, the effect of adversity on fall school engagement and change in externalizing symptoms was significant only for boys. Adversity did not have a main effect on early kindergarten indices of academic competence. This discrepancy with previous research could be due to the fact that the majority of children in this study come from highly educated families, which may support academic success despite adversity exposure.
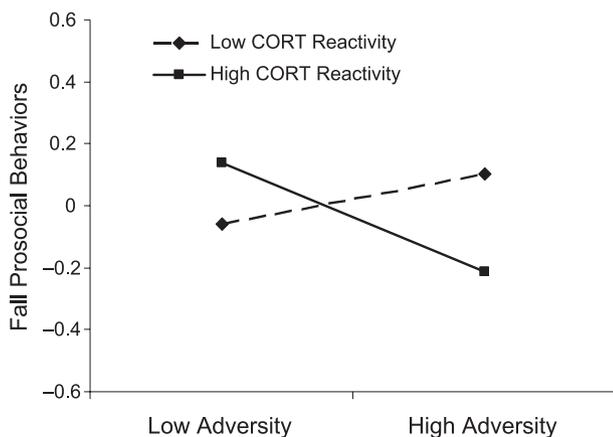

Figure 5. Prosocial behaviors as a function of adversity exposure and cortisol reactivity.
Note. Low and high adversity and cortisol reactivity values are graphed at 1 *SD* below and above the mean, respectively.

Low RSA reactivity was associated with higher levels of externalizing symptoms in the fall, corroborating previous findings of low stress reactivity and underarousal in children with externalizing symptoms (Boyce et al., 2001; Calkins et al., 2007; Mezzacappa et al., 1997; Pine et al., 1996; Pine et al., 1998) and the idea that low RSA reactivity is an index of emotional dysregulation and lability (Beauchaine, 2001). On the other hand, high cortisol reactivity was associated with high levels of externalizing behaviors and low levels of school engagement and academic competence. These findings are consistent with studies that have linked elevated cortisol in response to classroom challenges to poor self-regulation, impulsivity, peer rejection, and more solitary, negative behaviors (Dettling et al., 1999; Dettling et al., 2000; Gunnar et al., 1997; Gunnar et al., 2003), and the notion that hyperresponsivity of the HPA axis signals a risk for maladaptation (Gunnar & Vazquez, 2006). However, these main effects and the absence of others in these models should be interpreted with caution, as they were conditioned by significant interactions. For example, the effect of RSA reactivity on change in academic competence significantly varied across sex, with high reactivity being promotive for boys but a risk factor for girls. In addition, high cortisol reactivity was related to higher levels of fall externalizing symptoms only in boys. These findings suggest that children's sex may be an important factor to consider when examining the effects of stress reactivity on adaptation.

The study's most novel and salient findings emerged when adversity and stress reactivity were considered together, as components of interactions between environmental exposures and measures of biological sensitivity. Stress reactivity moderated the negative effect of family adversity across various domains of adaptation. Overall, the findings are consistent with the stress diathesis hypothesis that high-reactive children show worse adaptive functioning in the context of high adversity. Indeed, such children generally evinced the lowest levels of adaptive functioning of the entire study sample.

However, equally reactive children in settings of low adversity showed the highest levels of adaptation, levels even higher than those of their less reactive counterparts. Specifically, in the context of low family adversity, children who showed high RSA reactivity in response to challenges had the lowest levels of externalizing symptoms and the highest levels of prosocial behaviors and school engagement. Although adaptation showed significant stability from fall to spring, high-reactive children showed improvement in academic competence in the context of low adversity and a decline in competence in the context of high adversity, whereas the inverse was true for low reactive children. Similarly, children who showed high cortisol reactivity to the challenge protocol had the highest levels of prosocial behaviors in the context of low adversity. Further, children exhibiting low RSA reactivity in response to challenges were fully or partially buffered against the harmful effects of adversity on externalizing symptoms, prosocial behavior, and school engagement. Likewise, among children who showed low cortisol reactivity, levels of prosocial behaviors did not significantly change across different levels of adversity.

These findings support the biological sensitivity to context theory advanced by Boyce and colleagues (Boyce 2007; Boyce & Ellis, 2005) and the concept of differential susceptibility to environmental influences proposed by Belsky and colleagues (Belsky, 2005; Belsky et al., 2007). This study illustrates that high reactivity is not merely a pathogenic, risk-amplifying response to adversity but can also promote adaptive functioning. Corroborating Boyce and colleagues' theoretical perspective, children exhibiting high levels of biological sensitivity to context, as indexed by high autonomic and adrenocortical reactivity, were more susceptible to environmental influences in the context of both low and high family adversity. Thus, biologically sensitive children showed the highest levels of symptoms in the context of high family adversity but the highest levels of competence in the context of low family adversity. However, a lack of family adversity does not necessarily imply the presence of a nurturing family environment. Thus, future studies will need to further examine the role of heightened biological sensitivity to context across both stressful, health-undermining and supportive, health-enhancing contexts.

Together, these findings provide strong support for reframing stress reactivity as a biological sensitivity to context. First, the findings are relatively robust, especially for RSA reactivity, which emerged as a significant moderator of adversity effects across all four domains of adaptation. The evidence was parallel, though less robust, for cortisol reactivity, as the interaction effect was significant only for one domain. Second, each observed interaction was consistent with the biological sensitivity to context theory; high reactivity was promotive in the context of low adversity but a risk factor in the context of high adversity. Third, the interaction effect was found for both positive and negative indices of adaptation. Past studies have rarely

examined how interactive processes apply to multiple domains of functioning and have often focused primarily on measures of psychopathology. Fourth, these highly consistent interaction effects were found in a normative, community sample of typically developing children. If stress reactivity moderated the effects of adversity on development in a community sample of children, who on average do not face extreme disadvantage or show clinical levels of problem behavior, the interactive effects are likely to be larger at the extreme ends of such distributions.

Finally, the reported interaction effects complement recent work examining individual differences in markers of behavioral and genetic susceptibility to environmental influences. The current findings are consistent with research on behavioral reactivity indicating that children with high levels of negative affectivity are particularly susceptible to both negative and positive experiences (Belsky, 2005; Belsky et al., 2007; Klein Velderman et al., 2006). In addition, the current findings parallel recent research showing that genetic polymorphisms can moderate the effects on adaptive functioning of family adversity, unresolved trauma, or abusive parenting (Bakermans-Kranenburg & van IJzendoorn, 2007; Caspi et al., 2002; Taylor et al., 2006; van IJzendoorn & Bakermans-Kranenburg, 2006) and resonate with the observations of Rutter and colleagues on the previously overlooked protective effects of ''risk-engendering'' genetic variants (Rutter, Moffitt, & Caspi, 2006). Future studies should examine whether these behavioral, physiological, and genetic markers of susceptibility to contextual factors represent the same phenomena expressed at different levels of assessment or whether they represent different types of susceptibility that may have a cumulative effect on development.

For the purposes of future studies, it is important to note that the nature of the reported interaction effects may depend on several dimensions depicted in the conceptual model of Figure 6. Single studies may be capable of capturing only a portion of the underlying, paradigmatic interaction shown in Figure 6, as the ''observational window'' shifts from study to study depending on sampling characteristics, the outcomes of the interest, the measure of reactivity, and the type of challenge. For example, the observational window may shift to the right in studies of highly disadvantaged child populations or upward in studies of children with severe behavior problems. Similarly, the slopes of the two component lines may be determined by the

researchers' ability to capture the full range of stress reactivity responses and thus distinguish between high- and low-reactive children. It is critical, therefore, that developmental researchers examine and compare the interactive effects of stress reactivity and context across a range of outcomes and environmental influences and that the full scope of existing literature be taken into account in the interpretation of such effects.

The relations between stress reactivity and context are also likely to be dynamic and evolving over time and the present study provides only a snapshot into a potentially complex cycle of bidirectional and reverberatory influences. Boyce and colleagues (Boyce, 2006, 2007; Boyce et al., 2005) have argued, for example, that stress reactivity may not only moderate effects of early experience but may itself *be influenced by* the quality and character of early experience. While our analyses addressed stress reactivity as a moderator of adversity, given the developmental stage of the participants and the nature of the study data, we are mindful that adversity exposure could also play a role in shaping individuals' stress reactivity. From the perspective of evolutionary biology (Ellis, Jackson, & Boyce, 2006), children growing up in supportive and nurturing families might be expected to develop high levels of biological sensitivity to context in order to take greater advantage of the positive and stimulating features of their environments. As a result of both high sensitivity and protective environments, these children would show high levels of competence and low rates of mental and health problems. On the other hand, children
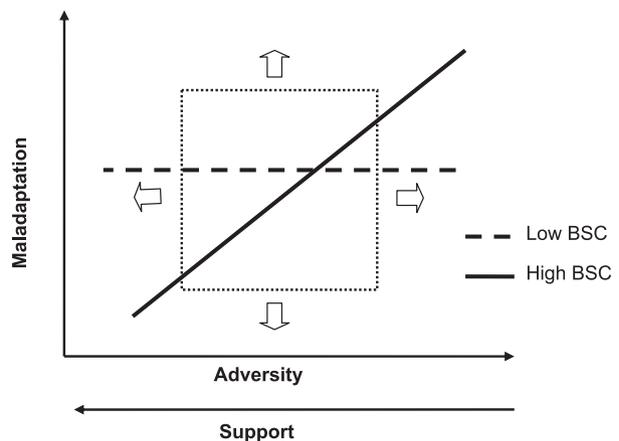

Figure 6. Conceptual model of the interactive effects between biological sensitivity to context (BSC) and environmental influences.

exposed to high levels of early risk and adversity may also develop high biological sensitivity to context, as a means of sustaining vigilance for environmental threats and hazards. Although such vigilance may be adaptive in the short run, over a longer period of time it could augment children's vulnerability to the deleterious effects of adversity. It is not surprising, then, that these children tend to have higher rates of mental and health problems. On the other hand, children who develop low biological sensitivity to context seem to be less affected by both positive and negative environmental influences. These children may consequently demonstrate resilience—adaptation that is better than expected given their adversity exposures—or vulnerability—low levels of adaptation despite growing up in environments abundant with resources and support (Luthar, 2006; Masten & Obradović, 2006). Future studies are needed to examine the processes by which early experiences shape children's biological stress responses.

Strengths, Limitations, and Future Directions

This study had several unique strengths as well as notable limitations that highlight important directions for future improvements. First, the study was based on a large, ethnically diverse sample of kindergarten children; however, a majority of children came from families with relatively high socioeconomic status, as indicated by income and parents' education. Second, the study was unique in that it employed a carefully controlled measure of autonomic reactivity that took into account the possible effects of motor activity and engagement demands elicited by challenge tasks. However, only one third of the sample had elevated cortisol in response to stress reactivity protocol, which may have contributed to less robust findings for cortisol reactivity. Future studies should attempt to raise the responder rate, as a lack of response to the current protocol does not necessarily indicate a low reactivity to more intense, real-life stressors. Third, we included the various sources of family adversity as an index of early experience. However, future researchers should explicitly assess positive environmental influences in children's lives by including measures of social support and resources. Another strength of the current study was the use of multiple informants. While it may not always be feasible to use multiple informants when assessing family adversity, given the young age of the participants and the nature of the construct, future studies should, where possible, triangulate the child,

parent, and teacher reports of adaptation, thus minimizing informant variance. Fourth, the study was unique in its effort to compare how both indices of autonomic and adrenocortical reactivity interact with early family adversity to predict adaptation. In addition to examining how indices of different types of stress reactivity affect children's behaviors and skills, future research should examine how different stress response systems interact to produce unique stress reactivity profiles, which may be related to different adaptive patterns across adversity exposure. As discussed earlier, researchers might use person-focused analytic approaches to examine whether the same children express analogous behavioral, physiological, and genetic sensitivity to contextual factors. Finally, future research should examine transactional relations between stress reactivity and adversity across longer periods of development, as adaptation in this study showed high stability over the 6-month period.

Conclusion

Given the pervasive and long-lasting effects that adversity can have on adaptation (Luthar, 2006; Obradović et al., in press; Sameroff, 2006), it is important to elucidate the processes that buffer or exacerbate these effects. This study represents a rare attempt to examine complex interactions between biological and environmental factors. The findings indicate that children's biological sensitivity to social context played an important role in moderating the effects of early experiences of family adversity on positive and negative indices of adaptive functioning. We believe that studies such as the one reported here can advance future prevention and intervention efforts, to address more holistically the social disparities in children's competence and psychopathology.

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