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Context-Specific Freezing and Associated Physiological Reactivity as a Dysregulated Fear Response

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The putative association between fear-related behaviors and peripheral sympathetic and neuroendocrine reactivity has not been replicated consistently. This inconsistency was addressed in a reexamination of the characterization of children with extreme fearful reactions by focusing on the match between distress behaviors and the eliciting context. Eighty 24-month-old children were observed in 4 mildly threatening contexts, and the relations among different measures of fear-related behaviors, reactive and basal cortisol levels, and baseline cardiac measures of heart rate, respiratory sinus arrhythmia, and preejection period (PEP) were examined. The hypothesis that only behaviors under the less threatening context would be associated with higher cortisol and sympathetic cardiac activity was confirmed; only task-specific freezing behavior predicted higher reactive and basal cortisol levels and resting PEP measured 1 week later. Implications for the conceptualization of dysregulated fear behaviors in the classification of extremely fearful children are discussed.

Although several investigators have examined the link between fear-related behaviors and temperaments (e.g., behavioral inhibition) and physiological reactivity, findings have been inconsistent (Buss et al., 2003; Kagan, Reznick, & Snidman, 1987; Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988; Schmidt et al., 1997; Schmidt, Fox, Schulkin, & Gold, 1999). Discrepancies in the literature are difficult to resolve given methodological differences between studies: unselected samples versus extreme groups, whether the behavior of interest is a temperament trait or a behavioral state, variations in the number and type of physiological measures, and limits on the number and type of contexts assessed. The current study extends this literature by reexamining this association, with particular focus on alternative operationalizations of fearful temperament. We argue that *dysregulation* of the fear behavioral response across contexts will provide a stronger link to physiology.

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Measuring Fearful Temperament

The study of individual differences in affective behavior in infants and young children is often synonymous with temperament research. Definitions of temperament vary (Goldsmith et al., 1987), but most reflect individual differences in the reactivity and regulation of expressed emotions (e.g., fear). Observation across multiple contexts, across multiple raters, and from multiple behavior and physiological systems is necessary to adequately characterize this variation (Goldsmith & Campos, 1986; Rothbart & Bates, 1998). Given the complexity of temperament, no single measure of behavior is believed sufficient to characterize these individual differences.

In the case of fearful temperament, multiple behavioral indicators have been considered: motivational aspects (e.g., withdrawal, avoidance, and inhibition), various communicative behaviors (e.g., facial expressions and distress vocalizations), reactive responses (e.g., freezing and startle), and social manifestations (e.g., shyness). Although components of the same underlying process, these indices may have unique associations with physiology and the developmental risk for anxiety. Complications in interpreting the literature linking fear with physiology arise when studies use different behavioral indicators to measure fearful temperament. The current study compared multiple measures of fearful temperament behavior, in different contexts, with physiological reactivity. Unlike the case in previous studies, we stressed the importance of the freezing response. The tendency to reduce activity in response to threat has been commonly observed in infants (Campos, Barrett, Lamb, Goldsmith, & Sternberg, 1983; Kagan, Snidman, & Arcus, 1992; Rothbart, 1988). We suggest that freezing might be a crucial component of fearful temperament that will have strong links to physiological reactivity, especially when considered in reference to the eliciting context (Davidson, Jackson, & Kalin, 2000).

Developmental Consequences of a Fearful Temperament

The bulk of the temperament literature has focused on the construct of behavioral inhibition. Behaviorally inhibited children are characteristically shy and withdrawn in novel social situations and show signs of increased anxiety in the face of novel challenges (Garcia-Coll, Kagan, & Reznick, 1984; Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984; Kagan, Reznick, Snidman, et al., 1988). Like other temperament dimensions, inhibition is relatively stable throughout childhood (e.g., Caspi & Silva, 1995; Kagan et al., 1987; Pfeifer, Goldsmith, Davidson, & Rickman, 2002). However, there are limits to this stability (Asendorpf, 1994; Davidson & Rickman, 1999).

Fearful temperament—specifically, behavioral inhibition—has been linked to risk for behavioral and psychiatric problems. Studies in both clinical and nonclinical samples have shown a moderate association between inhibition and risk for anxiety disorders in childhood (Biederman et al., 1993; Hirshfeld et al., 1992; Rosenbaum et al., 1991; C. E. Schwartz, Snidman, & Kagan, 1999) and between inhibition and depression (Caspi, Moffitt, Newman, & Silva, 1996) or anxiety in early adulthood (Kagan & Snidman, 1999).

Despite evidence for a link between inhibition and anxiety disorders, only one third of inhibited children develop clinically significant symptoms (C. E. Schwartz et al., 1999). Many researchers have suggested that physiological measures will be necessary to identify a more homogeneous group of children with an extreme fearful phenotype (Buss et al., 2003; Davidson, 2001; Kagan, Snidman, McManis, Woodward, & Hardway, 2002). Others have argued for the role of context in the expression of fear-related behaviors (Davidson et al., 2000) and, in particular, that the regulation of fear behavior across multiple contexts, including contexts deemed low in threat, will provide a better measure of extreme fear temperaments. Moreover, examination of behaviors in particular contexts may have implications for understanding the biological substrates of inhibition and anxiety. With the goal of understanding the roles of physiology and context in fear behaviors, we turn to these literatures.

Physiological Characteristics of Fear

Multiple neural structures and systems (e.g., the amygdala, prefrontal cortex, and neuroendocrine systems) are hypothesized to be part of the fear emotion circuitry (Davidson et al., 2000). Several empirical studies during the last 15 years have tested the putative association between fear-related behaviors and physiological reactivity. These studies have generally focused on two physiological systems: the cardiovascular system, including measures of heart rate (heart period), heart rate variability, and respiratory sinus arrhythmia (RSA), and the neuroendocrine hypothalamic-pituitary-adrenocortical (HPA) system, including measures of cortisol.

Cardiac Physiology and Fear

Autonomic reactivity, including both the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) branches, has been a central focus of research on stress sensitivity and the development of behavior problems related to the fear system (e.g., Boyce et al., 2001). Kagan has characterized the physiological response patterns of extremely inhibited children as reflecting an overall increase in SNS activity (Kagan et al., 1987), which should be reflected in peripheral measures such as heart rate (HR). Some research has supported this hypothesis. For instance, inhibited children had higher and less variable HR (Kagan et al., 1987; Kagan, Reznick, & Snidman, 1988; Kagan, Reznick, Snidman, et al., 1988; Reznick et al., 1986). A popular cardiac measure in this area is respiratory sinus arrhythmia (RSA), known to measure PNS control of the heart and thought to reflect vagal tone (Porges, 1992). Infants with high resting RSA were less fearful, approached a stranger more often, and were more active than infants with low RSA (Stifter & Fox, 1990). However, some investigators failed to replicate the predicted relation between HR variability or RSA and behavioral inhibition, especially when children were unselected (Asendorpf & Meier, 1993; Calkins & Fox, 1992).

The physiological response patterns of fear-related behaviors have largely been characterized as reflecting either an overall increase in SNS activity (Kagan et al., 1987) or PNS withdrawal (Porges, 1992; Porges & Doussard-Roosevelt, 1997). A comprehensive profile of cardiovascular reactivity, including both SNS and PNS measures, would be most informative in identifying specific autonomic concomitants of fearful behavior. In a recent study, children with internalizing versus externalizing behavior problems were found to have unique autonomic reactivity profiles reflecting a balance between SNS and PNS activity (Boyce et al., 2001). Specifically, the children classified as internalizers showed the highest levels of PNS withdrawal, whereas children classified as externalizers showed an overall diminished reactivity of both the PNS and the SNS. We collected measures of PNS and SNS activity in the current study.

Neuroendocrine Reactivity and Fear

Cortisol has also been implicated in the fear response. Cortisol is the main glucocorticoid of the HPA axis. Emotional stressors, such as novelty and uncertainty, involving fearful responses result in cortisol increases (Gunnar, 1989; Kirschbaum & Hellhammer, 1989, 1994; Mason, 1975). Elevations in cortisol correlated with distress in 9-month-old infants during maternal separation (Gunnar, Larson, Hertsgaard, Harris, & Broderson, 1992) and with withdrawal and crying in novel situations (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Of particular relevance to the current study, elevations in cortisol were limited to one context (Gunnar et al., 1992). High levels of cortisol during a laboratory visit at 21 months were associated with concurrent and follow-up inhibition classifications (Kagan et al., 1987). Research with nonhuman primates has established an important link between the duration of freezing behavior and the activity of the HPA system (Kalin, 1993). Monkeys with high cortisol levels engaged in longer durations of freezing during threat. Thus, the activity of the HPA system may regulate behavior in the face of threat (Kalin, 1993). However, several studies have failed to find an association between fear behaviors and stress cortisol levels (e.g., Buss et al., 2003; Kagan, Reznick, Snidman, et al., 1988; Schmidt et al., 1997, 1999). The predicted association has also been observed with basal levels of cortisol. Inhibited children had higher nonstress morning basal cortisol levels compared with uninhibited children (Kagan et

al., 1987; Schmidt et al., 1997). In a recent study, afternoon basal cortisol levels predicted fear-related behaviors of internalizing, self-isolation, and social withdrawal reported by parents and teachers 1.5 years later (Smider et al., 2002).

Elevations in cortisol are not unique to fear-related behaviors; they have also been observed for bold and exuberant behaviors (de Haan, Gunnar, Tout, Hart, & Stansbury, 1998; Dettling, Gunnar, & Donzella, 1999; Donzella, Gunnar, Krueger, & Alwin, 2000; Tout, de Haan, Kipp Campbell, & Gunnar, 1998). Note, however, that all of these studies reported reactive measures of cortisol.

Cardiac and HPA Associations

Some studies have focused on the interconnections between these systems in animal models and human adults, suggesting that cortisol may directly affect changes in HR. Evidence in rats suggested a strong interaction between neuroendocrine activity and resultant cardiac reactivity (i.e., increases in HR). When the adrenal cortex was removed, increases in HR under stress conditions were abolished (Korte, Eisinga, Timmerman, Nyakas, & Bohus, 1992). In adults, HPA activity and SNS cardiac activity were associated under stress conditions, but cortisol was not associated with PNS activity (Sgoutas-Emch et al., 1994). However, it is important to note that these systems are activated at different times (i.e., HPA activity has a slower onset) and often under different circumstances (Bauer, Quas, & Boyce, 2002), and thus these responses may not always be associated.

Dysregulation of Behavior

The traditional approach to measuring fearful temperament involves a focus on the intensity of fear reactions. However, simply responding with intense fear may not represent a maladaptive response, especially when the situation is designed to elicit that reaction. Recently, temperament and emotion researchers, following the lead of animal researchers, have begun to emphasize the dysregulation (i.e., the inability to control or suppress behavior in response to task demands) of emotional expression across eliciting contexts as reflecting a maladaptive response. Recent observations of inhibited children in nonthreatening laboratory contexts are of theoretical importance because they highlight dysregulated behavior. Inhibited children showed less impulsivity, less exuberance, and more sadness than uninhibited children across several laboratory situations (Pfeifer et al., 2002).

Kalin and colleagues (Kalin, 1993; Kalin & Shelton, 1989; Kalin, Shelton, & Takahashi, 1991) have developed a nonhuman primate model for identification of dysregulated fear-related behavior by demonstrating that changes in the context of threatening situations resulted in dramatic changes in fear-related behaviors. Predictable regulation of fear-related behaviors was observed across three contexts: maternal separation, human intruder with no eye contact, and human intruder staring (Kalin & Shelton, 1989). Unique fear behaviors were consistently displayed in these three contexts. For example, monkeys were more likely to freeze during the intruder no-eye-contact condition. Despite these strong context effects, individual differences in intensity and regulation were observed (Kalin, Larson, Shelton, & Davidson, 1998). Of particular relevance to the current study was the identification of animals that were observed to freeze during the stare condition. This out-of-context freezing reflected the *dysregulation* of the animal's fear behavior. Moreover, the animals that displayed this dysregulated freezing had the highest basal cortisol levels (Kalin, Shelton, Rickman, & Davidson, 1998).

The Current Study

The aforementioned research with primates serves as a model for the hypothesis that dysregulation of fear-related behaviors across contexts may have negative physiological consequences. We suggest that an alternative to the traditional method of identifying maladaptive fear responses (i.e., focusing on intensity) would be to look at the regulation of behavior across contexts varying in threat incentives. To this end, we examined fear-related behaviors in four mildly threatening contexts. Several different fear-related behaviors were measured and compared on their level of association with cortisol and cardiac reactivity. In particular, we highlight the significance of the freezing response. Although we expected to find associations among the fear-related behavior measures and physiological reactivity, we hypothesized that freezing during the less threatening context would be most strongly associated with high cortisol levels and SNS cardiac reactivity. Because individual differences in fear-related behaviors should be accentuated under less threatening circumstances, we would classify children who react with intense fear behavior to these situations as dysregulated in their fear response.

Method

Participants

Participants were eighty 24-month-old children (39 girls and 41 boys) recruited from local public birth records. Participants and families were not selected for any behavior or characteristic. Most of the participants were Caucasian (96%) and were from intact middle-class families (Hollingshead index = 48.66).

Procedure

The children participated in two laboratory visits separated by 1 week. The first visit consisted of a series of modified Preschool Laboratory Temperament Assessment Battery (Lab-TAB) episodes (Goldsmith, Reilly, Lemery, Longley, & Prescott, 1994). We modified these episodes for use with children under age 3 (Buss & Goldsmith, 2000). Only data from four fear/threat-eliciting contexts were included in this study. The second visit involved the recording of cardiovascular reactivity, including electrocardiogram (ECG) and impedance cardiography (ZCG). We collected saliva samples before and after each visit, and parents collected saliva samples at home.

Stranger approach. We observed children during three stranger approaches, two during the first visit and one during the physiology visit. There were two general stranger approach contexts. In both contexts, a male stranger entered the room and paused at least 8 feet away from the child; following a 10-second pause, he slowly approached the child, knelt down, and looked at the child without speaking for up to 2 min. The experimenter terminated the episode if the child became too distressed (i.e., cried for 20 s without calming) or upon request from the mother. The two stranger approaches differed only in the restraint that was placed on the child's behavior. In the first approach (the stranger high chair context), the child finished a snack while sitting in a high chair. This episode was designed to limit the availability of coping behaviors such as escape. In the second approach (the stranger free play context), the child played with a

few toys on the floor, a context that allowed the child to escape. In both contexts, the mother was present, but the experimenter instructed her not to interact with her child. The stranger approach involving the high chair context was repeated during the physiology visit (the stranger physiology context). For the stranger high chair context, seven episodes were shortened by child distress and two were shortened by mothers. None of the episodes was shortened by child distress or mother termination for the stranger free play context. For the stranger physiology context, six episodes were shortened by child distress and one by mother termination. We scored the following behaviors on a 4-point intensity scale (0–3, with higher numbers denoting higher levels of the fear-related behavior) every 5 s during the episode: facial fear, bodily fear, vocal distress (crying), and escape behavior. These 5-s epochs were averaged across the entire episode. Correlations among the discrete behavior averages were moderate to high (*r*s ranged from .27 to .71), so they were averaged together to create a fear composite ($n = 73$, $M = 1.07$, $SD = 0.54$, $\alpha = .78$; $n = 78$, $M = 0.44$, $SD = 0.26$, $\alpha = .71$; and $n = 66$, $M = 1.20$, $SD = 0.44$, $\alpha = .47$[1] for the stranger high chair, stranger free play, and stranger physiology contexts, respectively). In addition, we scored the total duration of freezing behavior in seconds (range = 0–170 s) during each stranger episode. Total duration of freezing was calculated by summing discrete periods of inactivity across the entire episode ($n = 73$, $M = 55.88$, $SD = 56.87$; $n = 78$, $M = 48.5$, $SD = 49.53$; and $n = 66$, $M = 83.63$, $SD = 39.01$ for the stranger high chair, stranger free play, and stranger physiology contexts, respectively). Duration of freezing was defined as a marked decrease in activity (> 2 s) with little or no movement, with or without indication of muscular tension. However, it should be noted that freezing often involved the child appearing stuck in an unnatural or uncomfortable position. Coders were required to reach and maintain reliability, which was checked for at least 20% of cases with the master coder (Kristin A. Buss). Percentage agreement reliabilities exceeded 90%, and kappa values ranged from .73 to .88 for all the behaviors.

Risk room. We adapted this episode from research with behaviorally inhibited children. This episode included two phases. During Phase 1, the experimenter led the child into the room, where there were several items for him or her to climb on and/or play with (a set of stairs and a mattress, a balance beam, a tunnel, a scary mask, and a large black box with an opening that appeared like a mouth with sharp teeth). The experimenter instructed the child to play with the objects "however you like." The experimenter then left the room and allowed the child to play and explore the room for 5 min. The parent was present but did not interact with the child. During Phase 2, the experimenter returned and asked the child to try each of the tasks. We created an inhibition composite ($n = 80$, $M = 1.26$, $SD = 0.53$, $\alpha = .73$) from the following behaviors: latencies to play with each toy (i.e., longer latencies indicating inhibition), latency to talk, number of objects played with (i.e., fewer objects indicating inhibition), length of time spent playing (i.e., less time indicating inhibition), intensity of facial fear, tentativeness of play (i.e., intensity of hesitant or cautious play), and proximity/approach to the parent (i.e., distance from the parent). Intercorrelations among these behaviors ranged from .31 to .83. These last three behaviors (facial fear, tentativeness, and proximity to the parent) were all scored every 10 s on 4-point intensity scales and were averaged across the entire episode. All behaviors were transformed (using quartiles) to the same scale (0–3); some were reversed (e.g., number of objects) and averaged together to create the inhibition composite. Coders were required to reach and maintain reliability, which was checked on at least 20% of cases with the master coder. Percentage agreement was 92%, and kappa was .88 for the inhibition composite.

Physiology Measures

Salivary cortisol. We collected saliva samples from children at both laboratory visits, one upon arrival (previsit) and one following completion of the visit (postvisit). Parents collected saliva samples in the home during the week between the two laboratory visits. Parents collected samples from the children four times per day (8–9 a.m., 11–12 p.m., 3–4 p.m., and 8–9 p.m.) on three consecutive days. We collected saliva using cotton dental rolls and Salivette tubes (Sarstedt, Inc., Newton, NC). Children used a small amount (< 1/8 teaspoon) of a cinnamon and sugar mixture for saliva stimulation. Cortisol values can be overestimated by radioimmunoassay (RIA) if the sample is acidic (i.e., low pH). Because stimulants such as Kool-Aid have been found to lower pH and increase cortisol values (E. B. Schwartz, Granger, Susman, Gunnar, & Laird, 1998), we conducted a method check prior to data collection to determine the effects of different stimulants on saliva (see Smider et al., 2002, for full details). For this study, we compared the use of cinnamon and sugar crystals to no substance, and we calculated pH levels. The pH levels of the samples containing cinnamon and sugar (6.33) were not statistically different from the pH levels of the samples without cinnamon and sugar (6.64), $t(16) = 0.658$, and the two types of samples were highly correlated ($r = .89$, $p < .01$). Cortisol was assayed in duplicate using the Pantex Cortisol I-125 RIA Kit modified for saliva (Pantex division of Bio-Analysis, Inc., Santa Monica, CA). The coefficient of variation (CV %), which represents the error of the assay, of the duplicate samples was ≤ 15%, with a mean of 3.8%. Samples that did not meet this criterion were reassayed. The error between unique assays was 7.4%. On each day of salivary cortisol collection, parents provided information about sleep patterns, eating, illnesses, and medications taken. Samples were removed from the analyses if children were ill and/or taking antibiotics or if they had eaten less than 1 hour before the sample was taken. Individual differences in sleep schedules were not associated with cortisol values across the samples (all *r*s < .10).

We averaged the three home samples at each time point to obtain mean values at each collection time. Children needed to have at least two samples at each time to be included in the final sample ($n = 59$). The means and standard deviations for the cortisol measures are presented in Table 1. We present the raw values in the table. The cortisol distributions were skewed and log10 transformed. There was variability in the time of visit and thus in cortisol collection. Because the magnitude of a stress response may vary with the circadian rhythm (Dallman et al., 1992), the effects of time of day on cortisol levels were examined. Slightly more than half of the sessions started in the mid to late morning (55% and 58% for Visit 1 and Visit 2, respectively), fewer were in the early afternoon (27% and 14%), and the rest were in the late afternoon (18% and 28%). We found evidence for significant time-of-day effects for both samples from Visit 1: $F(2, 61) = 3.69$, $p < .05$ and $F(2, 54) = 2.62$, $p < .09$ for the previsit and postvisit samples, respectively. In both cases, the late afternoon sample was significantly lower than the morning sample. We found the same effect for the postvisit sample from Visit 2, $F(2, 47) = 3.09$, $p = .05$, but not for the previsit sample. Postvisit samples collected in the late afternoon were lower than postvisit samples collected in the morning. Therefore, we regressed out time-of-day effects from the Visit 1 samples and the Visit 2 postvisit sample prior to the analyses. In order to measure reactivity to the visits, we calculated cortisol reactivity scores (Δ cortisol) by subtracting the previsit values from the postvisit values ($n = 58$, $M = .03$, $SD = .67$; $n = 50$, $M = -.02$, $SD = .60$ for Visit 1 and Visit 2, respectively). Time of day was not associated with Visit 1 or Visit 2 Δ cortisol.

We calculated four basal home averages across the 3 days of sampling: morning cortisol, noon cortisol, afternoon cortisol, and evening cortisol (see Table 1). There appeared to be a slight flattening of the slope for the afternoon sample. This has been reported elsewhere (e.g., Dettling et al., 1999; Watamura, Sebanc, & Gunnar, 2002) and may be associated with

[1] The alpha for the stranger physiology fear composite was lower than those for the other two episodes. Correlations among discrete behaviors were slightly lower than in the other two episodes (*r*s = .16–.58), and some correlations were not significant (e.g., crying and facial fear). Given that the overall pattern of correlations was the same as for the other stranger approach contexts, we created the same composite for this episode.

Table 1
Descriptive Statistics for Cortisol and Cardiac Measures

Physiological measure	*M*	*SD*
Cortisol		
Morning home[a]	.51	.67
Noon home[a]	.30	.33
Afternoon home[a]	.38	.58
Evening home[a]	.31	.73
Visit 1, previsit[b]	.35	.50
Visit 1, postvisit[b]	.37	.81
Visit 2, previsit[c]	.34	.47
Visit 2, postvisit[c]	.28	.52
Cardiac		
Baseline HR[d]	119.03	8.53
Stranger HR[e]	122.98	9.27
Baseline RSA[d]	5.06	.95
Stranger RSA[e]	4.71	1.05
Baseline PEP[d]	.061	.01
Stranger PEP[e]	.060	.01

Note. Cortisol values are in μg/dl. Heart rate (HR) values are in beats/minute. Respiratory sinus arrhythmia (RSA) values are in $ln(ms^2)$. Preejection period (PEP) values are in milliseconds.
[a] $n = 59$. [b] $n = 58$. [c] $n = 50$. [d] $n = 46$. [e] $n = 48$.

napping (i.e., afternoon cortisol values were higher on days when children napped). We calculated additional measures of basal reactivity including a cortisol slope measure, obtained by subtracting morning cortisol from evening cortisol ($n = 47$, $M = -.18$, $SD = .65$), and total cortisol production, which was a cortisol volume measure calculated from the area under each child's average cortisol curve[2] ($n = 47$, $M = 1.11$, $SD = 1.32$).

Cardiovascular recording. We collected and scored measures of cardiac output using the CIC-1000™ Impedance Cardiograph, software version 7.2 (SORBA Medical Systems, 1997). These measures were collected during rest (i.e., baseline) and during a stranger approach. For baseline cardiac measures, children watched a quiet videotape, "Baby Mugs," for 5 min. We videotaped children and coded the presence (0–1) of neutral, positive, and negative affect during the videotape presentation. Children showed predominantly neutral affect ($M = .99$, $SD = .04$; $M = .28$, $SD = .31$; and $M = .18$, $SD = .29$ for neutral, positive, and negative affect, respectively). None of the baseline recording episodes was terminated because of distress (i.e., crying).

Impedance cardiography (ZCG) gives estimates of volumetric changes, changes in stroke volume and cardiac output, and systolic time intervals, such as preejection time and left-ventricular ejection time (Kubicek, Karnegis, Patterson, Witsoe, & Mattson, 1966). In order to accurately calculate the impedance measures, we entered height, weight, and electrode separation (i.e., distance between the middle electrodes) measurements for each participant in the SORBA system. Note that we did not find significant gender differences in these measures ($ts < 1.30$). The SORBA software derived and displayed an averaged waveform for each 30-s consecutive interval. A minimum of four artifact-free waveforms was required to make the average. There were two waveforms created by ZCG: the ECG and dZ/dt waveforms. Using standard criteria (Kubicek et al., 1966), we calculated preejection period (PEP) as the time between the onset of the Q-wave of the ECG (i.e., the onset of ventricular contraction) and the B-point of the dZ/dt wave (i.e., the opening of the semilunar valve). Therefore, a shorter PEP accompanied greater heart muscle contractility and reflected sympathetic innervation. The data were inspected offline for accuracy in detecting the B-point. We summed across the 30-s averages to obtain a PEP baseline score and a PEP stranger score (see Table 1).

We extracted the continuous, raw ECG signal from the SORBA output system and calculated RSA and HR offline. This signal was then band-pass filtered at 30 Hz and 100 Hz, amplified 20K, and sampled at 500-ms intervals. We computed measures of HR and HR variability (specifically, RSA) using a moving polynomial filter to detrend the data prior to analysis, according to the procedures described by Porges and Bohrer (1990). We transformed the raw ECG signal into a file containing interbeat intervals (IBIs) with a program that used an adjustable threshold to detect R-waves. These IBI files were then entered into MXedit software (Delta-Biometrics, Inc., Bethesda, MD; Porges, 1985) to identify and edit artifacts.[3] We estimate that less than 3% of data points were edited and that the majority of the 5-min baseline signal ($n = 46$, $M = 276.80$ s, $SD = 15.20$) and the 2½-minute stranger signals ($n = 48$, $M = 142.66$ s, $SD = 28.90$) were preserved. The heart period time series was sampled in successive 500-ms intervals using a 21-point moving cubic polynomial to detrend data and a 25-point band-pass filter applied to pass variance between 0.24 and 1.04 Hz. RSA scores reflect the natural logarithm of the variance of this frequency band, $ln(ms^2)$. MXedit output calculates cardiac indices sequentially every 30 s and sums across them. The baseline and stranger HR and RSA values are presented in Table 1.

In order to measure cardiac reactivity, we calculated change in HR, RSA, and PEP by subtracting baseline measures from stranger measures ($n = 43$, $M = 4.04$, $SD = 6.38$; $n = 43$, $M = -.38$, $SD = .55$; and $n = 29$, $M = -.001$, $SD = .01$ for HR, RSA, and PEP, respectively). The baseline and stranger cardiac measures were highly correlated ($r = .75$, $p < .01$; $r = .80$, $p < .01$; and $r = .86$, $p < .01$ for HR, RSA, and PEP, respectively).

Data Reduction and Preliminary Data Analyses

Recall that we created fear behavior composites (inhibition, fear, and freezing) for each episode. The correlations among these fear behavior composites are presented in Table 2. Forming composites across multiple tasks is typical in the literature. The pattern of correlations supported the formation of two higher order composites across the three stranger approaches: a freezing composite ($n = 66$, $M = 63$, $SD = 48.47$) and a stranger fear composite ($n = 66$, $M = .87$, $SD = .34$). With the exception of one marginal correlation between risk room inhibition and stranger free play freezing, there was evidence for the independence of the different fear measures across the risk room and stranger contexts. In addition, the correlations between fear and freezing in the stranger approaches did not support the formation of an overall stranger fear behavior composite. These three fear composites were used in the analyses.

These fear behavior composites, cortisol, HR, RSA, and PEP were analyzed for main effects and interactions with gender. No effects for gender were found. Therefore, we pooled boys and girls for all analyses.

Finally, we addressed the issue of the amount of missing data between the two visits as well as missing cardiac data. Specifically, we observed 66 children during the stranger physiology episode. We compared three groups of children: children who did not return for the physiology visit ($n = 14$), children who provided both cardiac and stranger behavioral data for the physiology visit ($n = 46$), and children who completed the stranger physiology episode but only had partial or unusable cardiac data ($n = 20$). We compared these groups on distress behaviors during the risk room, stranger high chair, stranger free play, and stranger physiology episodes and on cortisol. No effects for group were found. The majority of the lost cardiac data was due to signal loss for the entire sampling duration because of equipment or electrode failure. For baseline, 50 children had ECG signals that were subjected to editing (i.e., 16 children had data that were

[2] Details of the cortisol area/volume calculation may be obtained from Kristin A. Buss.

[3] An undergraduate research assistant edited the ECG for RSA calculations. Although not entirely blind to the procedures (i.e., each episode had a unique code and file size), he was unaware of the hypotheses and of the children's behavior.

Table 2
Correlations Among Fear Measures for Three Stranger Approaches and Risk Room Episode

Measure	1	2	3	4	5	6	7
1. Freeze, stranger high chair	—						
2. Freeze, stranger free play	.43**	—					
3. Freeze, stranger physiology	.30*	.24†	—				
4. Fear, stranger high chair	−.12	.09	−.15	—			
5. Fear, stranger free play	.12	.32**	−.05	.40**	—		
6. Fear, stranger physiology	.13	.10	.21†	.29*	.28*	—	
7. Inhibition, risk room	.18	.20†	−.02	−.03	.11	.02	—

Note. Listwise $n = 66$.
† $p < .10$. * $p < .05$. ** $p < .01$.

lost because of equipment failure). Editing was difficult for 4 children possibly because of movement artifacts; therefore, we excluded these data. We correlated the amount of artifact-free cardiac data with distress behaviors, during baseline and stranger ECG recordings, and found no evidence for an association. Therefore, we were confident that attrition and missing data were not systematically related to distress behaviors.

Results

Results are presented first for bivariate relations between each fear composite and the physiology measures. Where applicable, individual behaviors used to create composites were examined more closely to (a) facilitate the investigation of the underlying constituents of the observed aggregate effects and (b) test hypotheses about behavior–context interactions. The second section of the results presents analyses of multiple physiological variables predicting variance in the fear behaviors.

Bivariate Relations Between Fear Behaviors and Physiology

Zero-order correlations between fear behavior composites and all physiological variables are shown in Table 3. Modest associations were found between the fear behavior composites and physiological reactivity (see the first three columns). Risk room inhibition, which most closely resembles behavioral inhibition, was marginally associated with basal cortisol and stranger PEP, but in the opposite directions from those predicted. There were no significant associations between the stranger fear composite and physiology. The freezing behavior composite was associated with noon cortisol, lower stranger HR, and baseline PEP. As expected, children who were high in freezing behavior had the highest basal cortisol levels (marginally significant) and the fastest PEP at baseline. These children also had lower task HR during the stranger approach. We found no association between change in HR, RSA, or PEP and any of the fear behavior composites (all rs $< .20$). We inspected the scatter plots of significant and marginally significant associations and found no striking effects of outliers.

Because the freezing measure represented a composite across the three stranger approaches, we deconstructed the composite to examine whether freezing behaviors in the three contexts differed in their associations with the physiology measures. First, we determined whether the behaviors differed across contexts. Less overall expression of distress was expected in the stranger approach in the free play context than in the high chair context. Using repeated measures multivariate analysis of variance, we compared facial fear, bodily fear, and freezing behavior across the three stranger contexts. There was a significant main effect of context, $F(6, 226) = 18.19$, $p < .01$. Univariate repeated measures tests for each behavior revealed that the amount of facial and bodily fear expressed varied across the three stranger approaches: $F(2, 114) = 31.61$, $p < .01$ and $F(2, 114) = 79.24$, $p < .01$ for facial fear and bodily fear, respectively. Planned comparisons with Bonferroni correction revealed that for facial and bodily fear, distress levels were lower in the stranger free play context than in the stranger

Table 3
Correlations Between Fear Behaviors and Physiology

Physiological measure	Fear behavior composites: Inhibition, risk room	Fear, stranger approach	Freeze, stranger approach	Freeze, stranger free play
Cortisol				
Morning[a]	−.13	−.06	−.21	−.14
Noon[a]	.02	−.01	.22†	.33**
Afternoon[b]	−.24*	.00	−.04	.05
Evening[c]	−.16	−.04	.01	.19
Total[b]	−.14	−.02	.08	.24†
Slope[c]	−.22	−.05	.12	.26†
Visit 1, previsit	−.11	.00	−.05	.00
Visit 1, postvisit	−.07	−.16	−.09	−.16
Visit 1, Δ cortisol[d]	.08	−.20	−.11	−.23†
Visit 2, previsit	−.21	−.18	.12	.32*
Visit 2, postvisit	−.10	.05	.08	.31*
Visit 2, Δ cortisol[e]	−.09	.12	−.16	−.16
Cardiac				
Baseline HR[f]	.03	−.04	−.21	−.24
Stranger HR[g]	−.02	.05	−.29*	−.03
Baseline RSA[f]	−.12	−.14	.11	.15
Stranger RSA[g]	−.14	.02	.20	.19
Baseline PEP[f]	.08	−.17	−.30*	−.32*
Stranger PEP[g]	.30†	.01	−.03	−.18

Note. HR = heart rate; RSA = respiratory sinus arrhythmia; PEP = preejection period.
[a] $n = 57$. [b] $n = 54$. [c] $n = 47$. [d] $n = 52$. [e] $n = 39$. [f] $n = 46$. [g] $n = 48$.
† $p < .10$. * $p < .05$. ** $p \leq .01$.

high chair ($p < .05$) and stranger physiology ($p < .05$) contexts. Finally, freezing behavior was lowest in the stranger free play context, $F(2, 114) = 9.76$, $p < .01$. Follow-up tests revealed a significant difference between the stranger high chair and stranger physiology contexts ($p < .05$), with the latter having the longest duration of freezing. Freezing in the stranger physiology context was also higher than freezing in the stranger free play context ($p < .05$). There was not a significant interaction.

Second, in order to test our specific hypotheses about behavior in context, we correlated freezing behavior in each context with the physiology measures. None of the correlations with freezing in the stranger high chair and stranger physiology contexts were significant ($rs < .19$). Despite the overall reduction in fear behaviors in the stranger free play context, longer durations of freezing behavior in this less threatening context were still associated with higher basal cortisol, higher previsit and postvisit laboratory cortisol (Visit 2 only), and faster baseline PEP (see Table 3, fourth column); these effects were more pronounced compared with the freezing composite (see Table 3, third column). We present scatter plots of the association of freezing in the free play context with noon cortisol (see Figure 1) and with baseline PEP (see Figure 2). In sum, only freezing in the stranger free play context was consistently associated with cortisol and PEP in the expected direction.

For exploratory analyses, we adopted an extreme group approach similar to that used in the behavioral inhibition literature (Kagan et al., 1987), and we were able to identify a group of children who appeared to show a dysregulated fear response. Examination of data at the individual level revealed that 12 children were extreme in the duration of freezing (> 1 *SD*, >100 s, $M = 128.69$ s, $SD = 25.27$) in the stranger free play context. We compared this group with children who were average in their freezing response ($n = 19$, $M = 45.40$ s, $SD = 12.86$). The high freezing group had significantly higher noon cortisol levels ($M = .52$ μg/dl, $SD = .42$), higher total cortisol production ($M = 1.71$, $SD = 1.36$), and faster baseline PEP ($M = .05$, $SD = .01$) than the comparison group: $F(1, 26) = 6.93$, $p < .02$; $F(1, 26) = 4.29$, $p < .05$; and $F(1, 26) = 9.45$, $p < .01$ for noon cortisol, total cortisol, and PEP, respectively. These children were also extreme in freezing behavior in the stranger high chair ($M = 90.06$ s, $SD = 52.44$) and stranger physiology ($M = 101.08$ s, $SD = 38.37$) contexts. However, it was only their behavior in the stranger free play context that was associated with neuroendocrine and SNS activity. Moreover, of the 12 children who were classified as extreme freezers in the stranger free play context, only 2 had noon cortisol below the overall mean, 4 had cortisol area values below the overall mean, and only 1 had a baseline PEP value equal to or less than the overall mean.

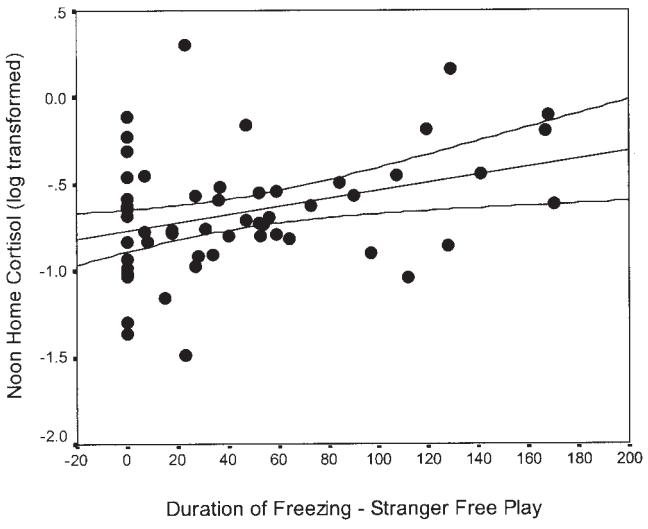

Figure 1. Scatter plot of duration of freezing and noon home cortisol for the stranger free play context. Lines represent the best fitting regression line and the 95% confidence interval.

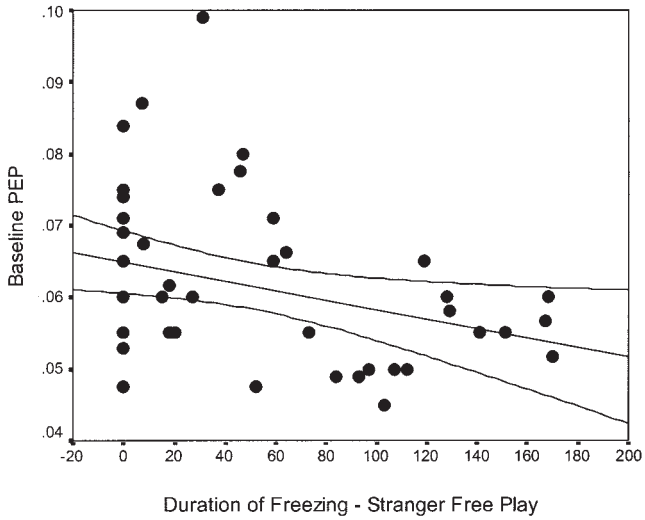

Figure 2. Scatter plot of duration of freezing and baseline PEP (preejection period) for the stranger free play context. Lines represent the best fitting regression line and the 95% confidence interval.

A final set of analyses suggested that children who showed extreme freezing behavior in the stranger free play context were not likely to be categorized as extreme on the other fear behavioral composites. Stranger free play freezing was not significantly correlated with the stranger fear composite, $r(78) = .17$, *ns*, or the risk room inhibition composite, $r(78) = .20$, $p < .10$. Moreover, of the 12 children who were classified as extreme freezers in the stranger free play context, only 4 had risk room inhibition scores higher than the mean, and only 3 had stranger fear composite scores higher than the mean. Thus, the majority of children who were high in freezing behavior were not extreme on other behaviors.

In sum, while most children found the stranger free play context to be the least stressful—as indexed by distress behaviors—we identified a small group of children who showed extreme freezing durations across all three contexts. These children were found to have the highest cortisol levels and the fastest baseline PEP scores.

Multivariate Analyses of Freezing With Cortisol and PEP

The pattern of correlations in Table 3 suggested the possibility for multivariate effects of cortisol and PEP on duration of freezing behavior. We used stranger free play freezing as the dependent measure in the regression analyses because of its theoretical and empirical centrality, as described above. Only children with both

Table 4
Regression Analyses of the Effects of Basal Cortisol and Preejection Period (PEP) on Duration of Freezing in the Stranger Approach Episodes

Variable	Model 1			Model 2			Model 3		
	B	*SE B*	β	*B*	*SE B*	β	*B*	*SE B*	β
A. Dependent variable = duration of freezing in stranger free play context									
1. Cortisol[a]	0.56	0.22	.40**	0.56	0.21	.41**	1.29	1.71	.94
2. PEP				−0.37	0.17	−.33*	−0.64	0.67	−.57
3. Cortisol × PEP							−0.69	1.61	−.58
R^2		.16			.27			.27	
ΔR^2		.16**			.11*			.00	
B. Dependent variable = duration of freezing in stranger free play context[b]									
1. Noon cortisol	0.54	0.23	.38*	0.59	0.21	.41**	0.59	0.22	.42
Visit 2 postvisit cortisol	0.52	0.23	.38*	0.46	0.21	.34*	0.35	0.35	.26
2. PEP				−0.37	0.15	−.36*	−0.37	0.15	−.35
3. Cortisol × PEP							−0.12	0.29	−.10
R^2		.40			.53			.53	
ΔR^2		.40**			.12*			.00	

Note. $N = 34$.
[a] Three-day average of noon cortisol measured at home. [b] $N = 29$.
* $p < .05$. ** $p \leq .01$.

cortisol and PEP values were analyzed ($n = 34$). Each regression analysis consisted of three nested models. Model 1 accounted for the variability in freezing that was due to cortisol.[4] Model 2 accounted for the variability in freezing that was due to baseline PEP. Model 3 examined whether cortisol and PEP had an interactive effect.

Results of the regressions are presented in Table 4. The regression analysis for freezing in the stranger free play context (see Table 4, Regression A) yielded a significant ΔR^2 for Model 1, $F(1, 32) = 6.23$, $p \leq .01$, and for Model 2, $F(1, 31) = 4.50$, $p < .05$. The interaction in Model 3 failed to account for a significant amount of new variance. Together, cortisol and PEP accounted for 27% of the variance in freezing behavior in the stranger free play context. We conducted a second regression entering both basal cortisol (noon home cortisol) and reactive cortisol (Visit 2 postvisit) in the first step (see Table 4, Regression B). In this regression, ΔR^2 for Model 1, $F(1, 26) = 8.80$, $p < .01$, and ΔR^2 for Model 2, $F(1, 25) = 6.56$, $p < .05$, were significant. Together, reactive cortisol, basal cortisol, and PEP accounted for 53% of the variance in freezing behavior in the stranger free play context. Note, however, that noon home cortisol and reactive cortisol were correlated ($r = .39$, $p < .05$), which likely inflated R^2 in Model 1.

Discussion

We predicted that freezing within the less threatening contexts would be more strongly associated with physiological reactivity than would more traditional fear behavior measures displayed in the context-appropriate situations. The results largely supported this prediction. We found that freezing during the stranger free play episode was associated with higher basal cortisol and resting PEP. In fact, only freezing behavior predicted baseline PEP 1 week later. This is an impressive finding when we consider that most of the reported fear–physiology associations are found only when fear and physiology are measured concurrently. Although freezing behavior in general across the three stranger approaches was associated with higher cortisol and PEP, the relationship between freezing and physiology was higher when we examined only freezing in the stranger free play context.

With this nonselected sample, we failed to replicate the findings reported in the literature that traditional measures of fear behavior, such as inhibition, facial fear, crying, and escape behavior, are associated with higher cortisol and cardiac activity. The inhibition composite was associated with lower basal cortisol and slower task PEP and was not associated with reactive cortisol, HR, RSA, or resting PEP. However, it should be noted that the current measure of inhibition was restricted to one episode. Although our findings from an unselected sample were inconsistent with findings from some studies of extreme groups of behaviorally inhibited children (e.g., Kagan et al., 1987; Kagan, Reznick, Snidman, et al., 1988; Reznick et al., 1986), failure to replicate the inhibition–physiology association has been reported elsewhere. In a longitudinal study of behaviorally inhibited children, Schmidt and colleagues (1997) reported the predicted association with basal cortisol and inhibition (i.e., higher morning cortisol associated with greater inhibition) but

[4] For the freezing composite, the cortisol measure entered in Model 1 was noon cortisol. For the first regression with stranger free play freezing, the effect of cortisol was measured by entering noon cortisol, cortisol area, and cortisol slope. However, noon cortisol and cortisol area were highly correlated ($r = .76$, $p < .01$), which resulted in collinearity and low tolerance, so cortisol area was removed from the final analysis. In addition, cortisol slope was removed from the final analysis because of a nonsignificant β ($< .15$). We only used noon cortisol in Model 1 in the final regression analysis of stranger free play freezing (Regression B in Table 4).

found the opposite pattern with laboratory cortisol (i.e., higher laboratory cortisol levels associated with less inhibition). Recently, there have been reports of bold and surgent children showing elevations in cortisol (de Haan et al., 1998; Dettling et al., 1999; Donzella et al., 2000; Tout et al., 1998). Children rated by their teachers as high in surgency (e.g., approach and positive affect) and low in self-regulation tended to demonstrate stress-related cortisol increases during a laboratory challenge (Donzella et al., 2000). Donzella et al. suggested that surgent children were more likely to have elevated cortisol because they were taking more risks. Therefore, acute increases in cortisol are found for both fearful and bold children, which is consistent with our knowledge that cortisol increases to prepare the body to respond to challenge. We hesitate to interpret our finding of an association of high cortisol with low inhibition as reflecting surgent behavior because low inhibition is not necessarily the same as surgency, and we failed to find an association between laboratory cortisol reactivity and inhibition.

Of particular interest for the broader issue of dysregulation of affect across contexts is that only behavior during the lowest threat context was associated with physiological reactivity in the predicted direction. Recall that for most children, distress levels were lowest in the stranger free play context. We suggest that the reduction in these fear behaviors resulted from the availability of more coping resources in this context (e.g., proximity to the mother). Although the stranger free play situation remained somewhat threatening, freezing is perhaps not an adaptive response when other behaviors may have been more effective in reducing distress (e.g., escape). Thus, although most children regulated or altered their responses from the more stressful to the less stressful contexts (i.e., they displayed fewer fear behaviors), we identified a small group of children who maintained a high level of freezing duration across all the stranger contexts. Therefore, we suggest that a longer duration of freezing in this context is somewhat analogous to freezing in the stare condition in the human intruder paradigm (Kalin & Shelton, 1989) and may be a contextually inappropriate, dysregulated response.

Although our sample of dysregulated children was small, it was consistent in proportion (15%) with samples found in research on behaviorally inhibited children (e.g., Kagan et al., 1984). The finding that these children were also extreme in their stress physiology supports our prediction that dysregulated fear behavior would be linked to stress physiology. Moreover, of the children who were characterized as high freezers in the stranger free play context, less than one third would have been classified as highly fearful if the more traditional measure of greater intensity of fear behaviors had been used. Finally, correlations between high fear and inhibition in the other contexts and physiology failed to reach significance, which replicates other studies' failure to find this association using an extreme group approach (Schmidt et al., 1999). Thus, the inability to *regulate* fear behavior to the demands of the situation was associated with physiological indices of stress. The results of the current study support our hypothesis that dysregulated fear reactions may put children at risk for extreme physiological reactivity. Thus, these findings highlight the importance of observing consistency of behavior across multiple contexts.

Implications

Lack of convergence in the literature. Why has there been a lack of consistency in reporting cortisol and fear associations? In this study, we found that inhibition during the normative context of the risk room was associated with lower cortisol, and freezing during a mildly stressful stranger approach was associated with higher cortisol. In a previous study of 6-month-old infants, fear behaviors were not associated with cortisol levels (Buss et al., 2003). In this study, intensity of freezing, a composite fear measure, and task-specific behavioral inhibition were not significantly correlated. Problems arise when these rather distinct behaviors are discussed as if they were part of the same construct (see Kagan, 1988, for an elaboration of this issue). Failing to distinguish components of the fear family of behaviors can thus lead to failure to discern physiology–behavior links. Another problem is that investigators often use the context to define the behaviors exhibited in the context. For example, in a threatening situation, any distress reaction by the child is labeled fear or inhibition without systematically differentiating discrete behaviors. As the current study demonstrates, each type of fear reaction (e.g., inhibition, the fear behavior composite, and freezing) may have different associations with physiology.

Intensity of fear versus dysregulation of fear. The behavioral inhibition and fearful temperament literature has largely focused on fear in a limited variety of contexts (novel or threatening), situations in which most children are likely to display some fearful behaviors (Garcia-Coll et al., 1984; Goldsmith & Campos, 1990; Kagan et al., 1984; Kagan, Reznick, & Snidman, 1988; Rothbart, 1986; Rubin, Burgess, & Hastings, 2002; Rubin, Hastings, Stewart, Henderson, & Chen, 1997). Although studies with this feature have produced important results, the design has limitations. First, research using this approach has demonstrated that fearful temperament is related to internalizing problems, but only in approximately one third of children (C. E. Schwartz et al., 1999). A second limitation is that inhibited children tend to avoid situations that they perceive as threatening; thus they may not always appear "fearful." A third limitation is that this line of research may not capture maladaptive fear responses. If we define dysregulation as a lack of appropriate fit between eliciting stimuli and emotional responses, studies measuring only eliciting situations in which fear is an adaptive response are not optimal for measuring maladaptive response styles. Therefore, we suggest that behavior outside the novel or threatening context might be most relevant for identifying maladaptive behavior patterns.

Developmental implications of dysregulated freezing/physiology profile. We identified a small subgroup of children for whom the mild stress of a stranger approaching was associated with extreme levels of freezing behavior and neuroendocrine and SNS activity. Given these findings, we suggest that children who engage in longer durations of freezing behavior, especially in contexts that are *objectively* less stressful (i.e., the stranger free play context), have a lower threshold for physiological reactivity. We believe examination of both context and physiology is necessary to identify children most at risk for anxiety-related behavior problems. A substantial number of children and adolescents develop symptoms of anxiety (21%: Albano, Chorpita, & Barlow, 1996) and depression (15%–40%: Hammen & Rudolph, 1996). Research and theory on the etiology and prevention of these problems has focused,

in part, on individual differences in affective behaviors (e.g., fear) with limited success. There is some evidence that physiological measures strengthen this prediction (Buss et al., 2003; Davidson, 2001; Kagan et al., 2002), with biology serving as a diathesis toward dysregulated fear behavior (Schmidt & Fox, 1999). For instance, recent theories of the etiology of anxiety disorders argue for the role of glucocorticoids (Rosen & Schulkin, 1998; Schulkin, McEwen, & Gold, 1994). Increases in cortisol during repeated fearful experiences may increase the excitability of the relevant neural circuitry, and this in turn may play a role in the establishment of pathological levels of anxiety. This interpretation is consistent with data from a sample of clinically referred children, in which anxious behaviors observed during a social-conflict play-group situation were positively correlated with increases in cortisol (Granger, Stansbury, & Henker, 1994). In a 6-month follow-up, children with increasing symptoms of severe anxiety had larger increases in cortisol in response to psychosocial challenges compared with children with reduced symptoms of anxiety (Granger, Weisz, McCracken, Ikeda, & Douglas, 1996).

Although context effects have prominence in animal models of fear (e.g., Kalin et al., 1991), the role of context in human fear reactivity is less prominent. Whereas many studies observe children in a variety of contexts, most studies aggregate behavior across those contexts to form fear composites (e.g., behavioral inhibition). Although this method is important for understanding individual variation in temperament (Rothbart & Bates, 1998), aggregating across contexts may obscure meaningful individual differences. As our results suggest, examination of behavior across contexts may yield more information about maladaptive behavior patterns (i.e., dysregulation) than may examination of intensity alone. Note that a defining feature of affective disorders according to criteria in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* is emotion dysregulation, the observance of emotional distress outside of the eliciting context (American Psychiatric Association, 2000). Finally, we argue that context effects on freezing behavior have implications for understanding the neural substrates of fear and the development of anxiety (Davidson et al., 2000). Our results suggest linkages between physiology and context effects in understanding the development of anxiety.

Limitations

The moderate sample size in the present study allowed for the identification of only 12 children with dysregulated freezing behavior, and it is clearly necessary to study larger samples of children who show these more extreme reactions. Our significant findings were achieved in the face of modest statistical power. Although the challenges of collecting complete physiological and behavioral data on large samples of very young children are considerable, such efforts are justified by our results. Future studies need to expand not only sample size but also the variety of contexts by providing contexts varying in the type of threatening incentive value (e.g., social, object, and risk avoidance), in the level of threat, and in the availability of coping resources.

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