Rebecca J. Brooker[1]
Randi A. Phelps[2]
Richard J. Davidson[3]
H. Hill Goldsmith[3]

[1]*Department of Psychology*
Montana State University
PO Box 173440, Bozeman,
MT 59717-3440
E-mail: rebecca.brooker@montana.edu

[2]*Department of Psychology*
Miami University
Oxford, OH

[3]*Department of Psychology*
University of Wisconsin
Madison, WI

Context Differences in Delta Beta Coupling are Associated With Neuroendocrine Reactivity in Infants

ABSTRACT: *Although evidence suggests that delta–beta coupling may provide a useful index of trait level cortico-subcortical cross talk in baseline contexts, there has been little work done to clarify the role of delta–beta coupling across contexts and in association with other physiological markers of emotion processing. We examined whether individual differences in coupling were visible across both positive and negative emotion-eliciting episodes during infancy (age 6 months). We also tested the convergence between measures of delta–beta coupling and neuroendocrine reactivity, which is also believed to index emotion processing. Patterns of coupling across emotion-eliciting episodes differed based on infants' levels of cortisol reactivity. Low cortisol-reactive infants largely did not show differences in coupling across emotion contexts while high cortisol-reactive infants showed greater coupling in non-fear contexts during baseline and fear episodes. Moreover, high cortisol-reactive infants showed greater coupling than low-reactive infants in non-positive episodes.* © *2015 Wiley Periodicals, Inc.* Dev Psychobiol 58:406–418, 2016.

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INTRODUCTION

Though levels of emotion-physiology specificity are debated, it is generally accepted that physiological processes support emotional responding and comprise aspects of the emotional experience (Levenson, 2003). Consistent with the developmental psychopathology perspective, characterizing physiological function in emotion-eliciting contexts during early life can advance our understanding of individual differences in typical and atypical emotional development and help to clarify pathways to early risk for disorder (Levenson, 2003; Sroufe & Rutter, 1984). Despite this poten tial advantage, assessments of physiology are often conductedduring baseline periods, which may be ideal for understanding trait-level individual differences (Tomarken, Davidson, Wheeler, & Kinney, 1992), but ignore the state-level physiology that occurs *in situ* during periods of emotion. An absence of this type of work has resulted in little understanding of the degree to which distinct physiological markers provide convergent or unique information about emotional processing. Such knowledge could lead to increased precision for identifying atypical development and early risk for disorders (Cohen & Cicchetti, 2006; Sroufe & Rutter, 1984). In the current study, we provide initial evidence for the convergence of neural and neuroendocrine function across emotion-eliciting contexts during infancy.

Delta–Beta Coupling and Emotion-Eliciting Contexts

Electroencephalography (EEG) is a noninvasive technique for measuring neural activity that can be used throughout the lifespan. Although the functional significance of EEG measures is not yet fully understood,

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associations between aspects of the EEG and measures of emotion are well documented. Power in the low-frequency delta band of the EEG is predominant in human infants (Bell, 1998; Stern, Ray, & Quigley, 2001). Heightened delta power is linked to increased activity in subcortical regions that support salience tagging as well as motivational, reward, and emotional processing (Knyazev, 2007; Leung & Yim, 1993; Robinson, 1999; Uhlhaas & Singer, 2006). Accordingly, direct stimulation to limbic circuitry results in increases in low-frequency power (Gray, 1982; Guyton, 1976). In contrast, power in the high-frequency beta band of the EEG is associated with awake states and periods of cognitive processing (Bell, 1998; Ray & Cole, 1985; Stern et al., 2001). Specifically, beta power is increased during periods of attention control and self-regulation (Engel, Fries, & Singer, 2001; Knyazev & Slobodskaya, 2003; Ray & Cole, 1985), presumably reflecting increased connections in intra-cortical circuits that support high-level cognitive functions (Engel et al., 2001; Ray & Cole, 1985).

Fast-wave oscillations, such as beta, are purportedly linked to an inhibitory influence of cortical activity on subcortical systems (Robinson, 1999). Consistent with this notion, the synchrony of oscillations between fast-wave and slow-wave frequencies has been suggested to reflect functional interactions between cortical and subcortical circuitry that underlie behavioral responses to one's environment (Knyazev & Slobodskaya, 2003; Knyazev, 2007). Specifically, positive associations (i.e., coupling) between delta and beta power are believed to reflect crosstalk between subcortical limbic systems and cortical systems of regulation (Knyazev & Slobodskaya, 2003; Knyazev, 2007), both of which have clear implications for emotion behaviors. Thus, delta–beta coupling may provide a real-time proxy for processes that are responsive to emotion elicitation.

In adults, greater coupling between delta and beta during a resting baseline is associated with greater trait inhibition (Putman, 2011; van Peer, Roelofs, & Spinhoven, 2008) and higher levels of anxiety (Knyazev, 2011; Miskovic et al., 2010). Recent research in developing populations has suggested that baseline delta–beta coupling is similarly enhanced for children who are at risk for anxiety problems based on parent symptoms (Miskovic et al., 2011a) and in preschoolers with atypical fear propensities that may denote an early risk for anxiety problems (Phelps, Brooker, & Buss, in press). The possibility that cortical-subcortical crosstalk is enhanced in more anxious individuals is consistent with the idea that certain forms of anxiety and anxiety risk include propensities for hyper-responsivity in the form of over-regulation or over-control (DSM-5, 2013; Eisenberg et al., 2001; Phelps, Brooker & Buss, in press). Consistent with this notion, the remediation of anxiety symptoms has been linked to reductions in delta–beta coupling (Miskovic et al., 2011b).

To date, research on delta–beta coupling has primarily focused on baseline measures. Examining coupling in emotion-eliciting episodes may provide unique information about neural correlates of emotion processing and may be more directly associated with risk for disorder than are baseline measures (Coan, Allen, & McKnight, 2006; Davidson, 1994). To our knowledge, only one study has examined changes in delta–beta coupling across neutral and emotion-eliciting contexts. This work revealed greater coupling in socially anxious relative to non-anxious adults during anticipation of a public speech (Miskovic et al., 2010). Because, in contrast to other examinations, group differences were not visible during a relaxed-state baseline, this work highlighted the need to consider the eliciting context when interpreting coupling measures. Namely, the authors speculated on the possibility that, to the extent that participants perceived the research procedures to be threatening, even baseline paradigms may have induced altered patterns of neural activity. In their work, which involved a relaxing baseline, differences in delta–beta coupling became maximally apparent as contextual demands highlighted emotional salience and increased the need for regulation. Studies of the extent to which contextual demands are linked to fluctuations in coupling in infants and children are absent from the extant literature. Thus, it is unclear whether individual differences in delta–beta coupling are already apparent across contexts early in life.

To our knowledge, examinations of coupling in positive emotion contexts have not yet been conducted with either children or adults. This is a critical oversight in emotion research given that both negative and positive emotions are important for healthy psychological functioning across the lifespan (Cole, Michel, & Teti, 1994). A number of psychophysiological measures of emotion processing are not specific to negative emotions in young children (e.g., Dennis & Hajcak, 2009), further warranting separate examinations of coupling in positive and negative emotion contexts. In contexts that elicit low or moderate levels of positive arousal, the need for regulation is low (Stifter & Moyer, 1991). It is possible, therefore, that positive contexts elicit low levels of delta–beta coupling as positive affect is elicited but regulatory demands remain low. In the current study, we assessed delta–beta coupling across both positive and negative emotion contexts. We expected to see high levels of delta–beta coupling during a negative-emotion episode and low levels of delta–beta coupling in a positive-emotion episode.

Delta–Beta Coupling and Cortisol Reactivity

Just as the dynamics of delta–beta coupling across emotion contexts have not been fully elucidated, it is unclear whether coupling is associated with other physiological processes that support emotion behaviors; this lack of clarity is particularly salient in childhood. If delta–beta coupling reflects a true aspect of emotional responding, it is reasonable to expect that coupling will fluctuate in predictable ways with other physiological correlates of emotion. There is evidence, in adults, for associations between delta–beta coupling and levels of cortisol, the glucocorticoid end product of the hypothalamic-pituitary-adrenocortical (HPA) axis, which supports responses to emotional stressors (Gunnar, Marvinney, Isensee, Fisch, & Palermo, 1989; Gunnar, Talge, & Herrera, 2009). Enhanced delta–beta coupling has been observed in young adults with greater baseline cortisol levels (Schutter & van Honk, 2005) and college students who received an oral administration of cortisol in the laboratory (van Peer et al., 2008). Administrations of testosterone, which has an antagonistic effect on the HPA axis, are associated with delta–beta decoupling, suggesting that coupling declines when HPA activity is inhibited (Schutter & van Honk, 2004). Developmental examinations of associations between delta–beta coupling and cortisol levels are largely absent from the literature. A demonstration of such associations early in life, as both neural and neuroendocrine systems are developing, would provide insight into the developmental timing of links between physiological systems that underlie emotion processing.

It is similarly unclear, in both children and adults, whether natural associations exist between neural and neuroendocrine responses to emotion-eliciting contexts. Cortisol reactivity, assessed as cortisol levels following an emotional experience relative to baseline cortisol levels, has been linked to expressions of negative emotion across development (Gunnar & Quevedo, 2007; Gunnar et al., 2009). A review of developmental research using reactive cortisol measures revealed the difficulty of evoking cortisol responses in very young children, possibly due to a dampened cortisol response during early development (Gunnar et al., 2009). The most successful contexts for eliciting a reactive cortisol response in the first year of life appear to be those that induce negative emotions via physical stressors or in contexts that involve uncontrollable social threats. An acute salience of social stressors is consistent with past work on emotional development (Bretherton & Ainsworth, 1974; Sroufe, 1977) and the role of cortisol in adaptive responding (Gunnar et al., 2009; Sapolsky, 1998). Cortisol has less frequently been examined relative to changes in positive emotion. There is evidence that greater positive emotion buffers against increases in cortisol levels during periods of stress (Buchanan, al'Absi, & Lovallo, 1999; Lai et al., 2005). Notably, contexts that elicit positive emotion also tend to be free of social stressors, making it unclear whether effects result from positive affect per se. The degree to which neural and neuroendocrine systems act in concert in positive emotion contexts during early in life remains unexamined. Thus, our second aim was to examine whether infants' cortisol reactivity was associated with delta–beta coupling. Specifically, we assessed whether a measure of laboratory cortisol, indicating reactive cortisol levels, was associated with delta–beta coupling as measured in different emotion-eliciting episodes. Given that cortisol levels are associated with greater reactivity to more stressful, less positive situations, we expected that greater cortisol would be associated with greater delta–beta coupling in non-positive episodes, such that both greater cortisol and greater coupling would be observed. In contrast, we expected no associations between cortisol and delta–beta coupling in positive episodes, where cortisol may be enhanced (indicating a neuroendocrine response to the laboratory visit) but delta–beta coupling would remain low.

In sum, the current study addressed two gaps in the literature. First, we examined whether changes in delta–beta coupling could be observed across emotion-eliciting episodes. Based on the limited work that has been conducted with adults, we expected greater delta–beta coupling in non-positive emotion episodes relative to positive emotion episodes. Second, we tested whether delta–beta coupling was associated with levels of neuroendocrine reactivity. Similar to the first hypothesis, we tested associations between cortisol reactivity and measures of delta–beta across emotion-eliciting contexts. Based on findings in adults, we expected that greater cortisol reactivity would be linked to greater delta–beta coupling during non-positive relative to positive contexts, providing evidence for convergence among systems. Notably, we conducted this work in infants in order to extend the developmental literature on delta–beta coupling and more fully characterizing physiological function in emotion-eliciting contexts during early life.

METHOD

Procedure

Participants were drawn from a longitudinal twin study of emotional development (Schmidt et al., 2013). When

infants ($N = 121$) reached approximately 6-months of age, they visited the laboratory where they participated in a series of behavioral episodes while EEG data were collected[1]. Before leaving, parents scheduled a time to return to repeat the procedure approximately 2 weeks later. Two visits were conducted with each participant in an attempt to maximize usable data from each infant; thus, available data were composited across both visits for each infant. Saliva samples were collected at the end of the first laboratory visit and for 3 consecutive days at home. Children's mean age at the time of each laboratory visit was 30.05 ($SD = 4.42$) and 30.74 ($SD = 2.45$) weeks.

The study includes infants with a family history of right-handedness who provided at least 30 s of usable EEG data per episode at 6 months of age ($N = 77$). Episodes ranged from 2 to 5 min in total length. Reflecting the recruitment area, the majority (97.4%) of infants were Caucasian; 2.6% of participants were African-American. Approximately half (45.5%) were male.

Measures

Baseline. Infants were seated in a highchair for the duration of each session during the laboratory visit. For the resting baseline, infants were kept alert and quiet by being shown several neutral objects for five 1 min trials.

Emotion-Eliciting Episodes. Emotion episodes were drawn from the Prelocomotor Version of the standardized Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith & Rothbart, 1999). Episodes included an interaction designed to elicit fear (non-positive episode) and an interaction designed to elicit positive affect (positive episode). Emotion-eliciting episodes were video-recorded for offline coding of infants' emotion behaviors. Off-line coding of emotion behaviors allowed us to verify whether fluctuations in physiological indices co-occurred with changes in observed emotion during the emotion episodes.

Non-Positive Emotion Episode. Following the conclusion of the baseline period, a male stranger entered the room where the mother and child were located. The stranger turned to look at the child and then paused for 10 s. After this, the stranger moved approximately half of the distance toward the child, where he paused for 5 s and then said, "Hello (*child name*), I'm going to come a little closer to you." The stranger then approached to within 1 foot of the child and stood next to him/her for 2 min. Following the conclusion of this two-minute period, the stranger turned away from the child and left the room. Recording was discontinued if the child displayed 30 s of intense crying.

Positive Emotion Episode. Following the Stranger Approach, an experimenter entered the room and instructed the parent and child in a game of peekaboo. This generally involved the parent moving behind a wooden screen while the experimenter asked, "Where's Mommy?" On the experimenter's cue, the parent emerged from behind the screen, smiling and saying "Peekaboo." This sequence was repeated six times and lasted approximately 2 min.

EEG Recording. Following consenting procedures, a lycra electrode cap (Electro-Cap International; Eaton, OH) was positioned on the infant's head using known anatomical landmarks. EEG data were recorded from 18 sites based on the 10/20 system (Electrode Position Nomeclature Committee, 1994). Sites included: Fp1, Fp2, F3, F4, F7, F8, T3, T4, T5, T6, C3, C4, P3, P4, O1, O2, Pz, and Fz. Data were referenced to Cz during acquisition and rereferenced offline to a whole-head average, with a minimum of 12 sites evenly distributed across the head (Bertrand, Perrin, & Pernier, 1985; Hagemann, Naumann, & Thayer, 2001). Electrode impedances were kept below 20 kΩ and impedances at homologous sites were kept within 5 kΩ of each other. EEG data were sampled at 500 Hz and amplified[2] with a gain of 20,000. A bandpass filter (1–200 Hz) and 60 Hz notch filter were applied during data collection.

Cortisol Collection. Saliva samples were collected at the end of the laboratory visit (approximately 1 hr after the session began) by the primary experimenter using a suction catheter. Because participants completed laboratory episodes in a standard order, all infant cortisol samples were collected roughly 20 min after the electrode application. Laboratory samples were time-matched to samples acquired on 3 consecutive days

[1] Infants also participated in an arm-restraint episode. However, because that context produced high numbers of EEG artifacts, those data were not included in this paper. Electrocardiogram and respiration data were also recorded, but were not included because they do not test study hypotheses.

[2] EEG was amplified with either a Grass Model 12 Neurodata System amplifier or James Long amplifier; amplifiers were changed approximately halfway through the study as a result of laboratory remodeling.

when the infant was at home[3]. All samples were stored at −80°C until they were thawed and centrifuged at 5000 rpm for 10 min to remove impurities. Cortisol was assayed in duplicate with a salivary enzyme immunoassay kit (Salimetrics, Inc; State College, PA). Two nonblind internal controls were used for each trial. Results were considered acceptable if the Coefficient of Variation of duplicate samples was less than 20%. Repeat assays were performed on any samples that did not meet this requirement.

Data Reduction

EEG Power. On average, infants provided sufficient amounts of usable data (M across episodes = 74.77 s; $SD = 33.90$). As might be expected, more negative affect during the stranger approach was associated with less available data ($r = .30$, $p = .04$). More available data was associated with less beta power at frontal electrode sites during the baseline ($r = -.24$, $p = .04$) and stranger approach ($r = -.37$, $p < .01$) episodes. More available data was associated with less delta power at central electrodes during peekaboo ($r = -.30$, $p = .02$). The number of seconds of available edited data was not associated with any other measures of beta our delta power during baseline (all rs $< .20$, ps $> .05$), stranger approach (all rs $< .20$, ps $> .05$), or peekaboo (all rs $< .20$, ps $> .05$) episodes.

EEG data were visually scored and edited within episode to remove artifact resulting from eye movement, muscle activity, and gross muscle movement. A Fast Hartley Transform (FHT; Bracewell, 1984) was applied to all artifact-free chunks of data that were at least 1.024 s in duration. Power in the 0 to 3 Hz and 8 to 12 Hz bands were computed and composited within episode; these values have been shown approximate the adult delta and beta bands, respectively, in samples of young children (Bell, 1998; Marshall & Fox, 2006). Focusing on previously-established delta and beta bands allows for the direct comparison of our results with other studies of delta and/or beta power in young children. Consistent with the procedures of Miskovic et al. (2010), delta and beta power were derived for F3, F4, C3, C4, and P3, P4 and composited to examine coupling in frontal (F3/4), central (C3/4), and parietal (P3/4) electrodes during each of the laboratory episodes. Because a correlation cannot be estimated from two within-person measures, coupling was examined at the group-level as the correlation between delta power and beta power. This approach is similar to previous work (Miskovic et al., 2010, 2011a).

Cortisol. Raw cortisol values were regressed on time since waking. The three home cortisol samples were averaged to derive a stable measure of baseline cortisol. The distributions of both the home and lab cortisol measures were skewed and so were $\log_{10}$ transformed. Home cortisol levels were subtracted from lab cortisol levels to obtain a measure of infants' cortisol reactivity to the laboratory assessment (Δ_{cortisol}). Samples were excluded from analyses if the infant was ill (e.g., ear infection), if the infant was taking antibiotics at the time of the sample, and/or if the sample was taken within 1 hr of eating. Sixty-two infants with usable EEG data also provided usable laboratory cortisol samples. However, note that because of our use of a change score to control for home cortisol, our measure necessitated that infants provide usable samples both at home and in the laboratory. A total of 43 infants who had usable EEG and laboratory cortisol also provided usable home samples.

Effects of sample time on cortisol samples were also examined. Sample time was uncorrelated with both home ($r = -.22$, $p > .10$) and laboratory cortisol ($r = .01$, $p > .10$). Of importance given our use of the home sample as a baseline measure, time of the home sample was highly correlated with time of the matched laboratory sample ($r = .74$, $p < .01$).

Groups of individuals who showed high and low levels of reactivity to the laboratory visit were created using a mean split based on values of the Δ_{cortisol} variable. A mean split was selected over other previously-used methods (e.g., median split, ± 1 SD) in order to maximize the likelihood of equal numbers of infants in both groups, though it should be noted that the pattern of results is preserved when other methods are used to split the sample. Twenty-seven infants were classified as low reactive; 16 infants were classified as high reactive. Cortisol reactivity was significantly different between the two groups ($t(41) = -5.29$, $p < .01$), with high-reactive infants showing greater increases in cortisol to the laboratory visit relative to low-reactive infants (Low: $M = -.08$, $SD = .15$, High: $M = .14$, $SD = .09$; $d = -1.81$).

Observed Behavior. Trained undergraduate coders scored emotion behaviors offline using video recordings

[3] A previsit saliva sample was not collected because infants and parents were already being asked to do a considerable amount at the laboratory visit. In particular, the sensor placement for the electrocardiogram and the capping for EEG were somewhat intrusive for the infants. The method of saliva collection also periodically resulted in periods of distress for the infants; we wanted to avoid an early-visit stressor to make sure that all infants began the visit in as neutral a state as possible. Moreover, the potential stress of traveling to and entering the novel lab with novel persons present would render the interpretation of a baseline measure ambiguous.

of emotion-eliciting episodes (*Stranger Approach*, *Peekaboo*). Emotion behaviors were not coded for the baseline episode and behavioral coding was not synced to flags in the EEG. Coders were required to achieve a minimum reliability of $\kappa = .70$ with a master coder before coding independently.

Consistent with Lab-TAB procedures, the following fear behaviors were coded during the *Stranger Approach* episode: intensity of facial fear and sadness (derived from Izard, Dougherty, & Hembree, 1983), intensity of bodily fear and sadness, intensity of distress vocalizations, intensity of escape behaviors, and latency to first fear behavior. The mean reliability across behaviors was considered to be good (mean $\kappa = .77$). With the exception of latency scores, codes for fear behaviors were assigned every 5–10 s (Goldsmith & Rothbart, 1999) and then averaged across the episode. Latency scores were reversed to reflect speed to fear responses. All scores were then standardized (z-scores) within behavior and mean composited to create a single composite measure of fearfulness across the episode.

Also consistent with Lab-TAB procedures, we coded the following behaviors indicating positive affect during the *Peekaboo* episode: intensity of smiling (derived from Izard et al., 1983), presence of laughter, positive vocalizations, and positive motor activity, and latency to first positive affect behavior. The mean reliability across behaviors was considered to be good (mean $\kappa = .82$). With the exception of latency scores, codes for positive behaviors were assigned every trial of the peekaboo game and then averaged across the episode. Latency scores were reversed to reflect speed to fear responses. Scores for all behaviors were then standardized (z-scores) and composited to create a single composite of fearfulness across the episode.

Missing Data. The Little's Missing Completely at Random (MCAR) analysis of patterns of missing data suggested that data were missing completely at random (Little's MCAR $\chi 2(102) = 111.79$, $p > .10$). Little's MCAR analysis is equivalent to a multivariate extension to the t-test that simultaneously evaluates mean differences on all variables in the data set (Little, 1988). A nonsignificant result suggests the probability of missing data is unrelated to any of the variables in the data set (Enders, 2010). Given that data were missing completely at random, we used a complete-cases analysis strategy, in which all available data were used for each analysis, to handle missing data. Sample sizes for different analyses vary as a result of the difficulty in collecting psychophysiological data from infants. In particular, the available data differed across contexts as a result of attrition across the visit.

RESULTS

Plan for Analysis

Following preliminary analyses to characterize group differences in cortisol reactivity and observed behavior, we examined delta–beta coupling across contexts using Pearson correlation coefficients (r). We used Fisher's r-to-z transform to test for group differences in coupling across contexts between infants showing low versus high levels of cortisol reactivity. Infants do not yet show the degree of specialization of neural activity seen in adults (Casey, Galvan, & Hare, 2005; Durston et al., 2002), for whom regulatory processes are largely localized to frontal and prefrontal areas. Thus, no differences were expected across frontal, central, and parietal sites for this infant sample. Therefore, our data analytic and interpretation strategy focused on patterns of findings that were simi lar across all electrodes. Imposing this requirement also aided in guarding against Type I error. This approach allows for a unique opportunity for internally replicating patterns of group differences in our relatively small sample.

Preliminary Analyses

Data were visually inspected for outliers. Consistent with the recommendations of Tabachnick & Fidell (2013) for dealing with univariate outliers, extreme values on all variables were windsorized to within 3 *SD* of the full-sample mean (total $n = 10$ across all variables). Descriptive statistics are shown for psychophysiological variables in Table 1. A one-sample t-test suggested that, for the group as a whole, differences in cortisol levels between the lab and home measures were not significant ($t(42) = -.04$, $p > .10$). However, examining the high and low cortisol reactivity groups separately revealed distinct patterns of change in cortisol levels between the home and laboratory assessment. In the low-reactive group, infants largely showed lower cortisol levels in the lab than in the home (M *(change)* $= -.08$, $SD = .15$, $t(26) = -2.88$, $p < .01$) while infants in the high-reactive group showed higher cortisol levels in the lab than in the home (M *(change)* $= .14$, $SD = .09$, $t(15) = 5.87$, $p < .01$).

Levels of negative affect during the fear-eliciting episode were similar for both high cortisol reactive and low cortisol reactive groups ($t(26) = .66$, $p > .10$). Similarly, after correcting for unequal group variances, group differences in positive affect were similarly nonsignificant ($t(8.50) = -1.81$, $p > .10$). Thus, emotion episodes appeared to have elicited similar levels of negative and positive emotion in both high- and low-reactive infants.

Table 1. Descriptive Statistics for Psychophysiological Variables

		M	SD
Cortisol (lab-home)	43	−.0011	.1692
Baseline Beta Power			
Frontal	75	.1531	.3770
Central	75	.3614	.4311
Parietal	75	.3175	.4270
Baseline Delta Power			
Frontal	75	2.7952	.4514
Central	75	3.0076	.3806
Parietal	75	3.1312	.3864
Stranger Beta Power			
Frontal	62	.3750	.4554
Central	62	.3625	.4005
Parietal	62	.3576	.3629
Stranger Delta Power			
Frontal	62	2.8362	.4367
Central	62	3.0087	.4076
Parietal	62	3.2128	.3403
Peekaboo Beta Power			
Frontal	56	.2830	.4303
Central	56	.3846	.4127
Parietal	56	.3570	.3159
Peekaboo Delta Power			
Frontal	56	2.6771	.3802
Central	56	2.9173	.4010
Parietal	56	3.0641	.3185

Delta–Beta Coupling Across Contexts

First, we examined delta–beta coupling for all infants who provided usable EEG data. Delta–beta coupling was significant at all electrode sites across all three contexts (mean $r = .48$, all $ps < .05$). As suggested by Figure 1, delta–beta coupling at homologous electrodes was not significantly different across the baseline, fear and positive episodes. The exception to this was that coupling during the positive episode was significantly less than coupling during the baseline episode ($z = 2.39$, $p < .01$). However, this finding was significant only at the parietal electrode site. Overall, then, infants showed significant coupling in baseline, positive emotion and negative-emotion eliciting but levels of coupling were not significantly different in different contexts.

Delta–Beta Coupling and Cortisol Reactivity

As shown in Figure 2, delta–beta coupling in low-reactive infants was largely nonsignificant across episodes and electrode sites. Significant coupling was observed only at frontal electrodes during the baseline and positive emotion episodes. In addition, low reactive children largely showed nonsignificant differences in coupling across the baseline, fear, and positive contexts. The exception to this was significantly less coupling at central electrodes during the fear episode relative to the positive episode ($z = -1.66$, $p < .05$) and marginally less coupling relative to the baseline episode ($z = 1.43$, $p < .10$). Overall, then, low-reactive infants showed nonsignfiicant levels of coupling across episodes and appeared not to modulate levels of coupling according to the eliciting context.

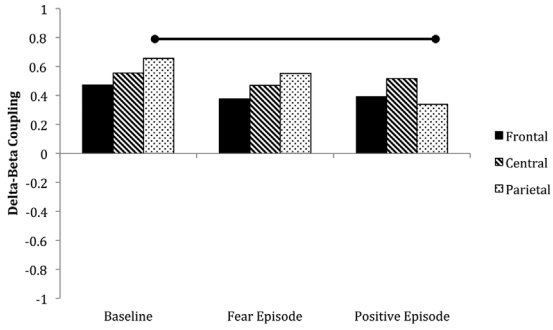

FIGURE 1 Delta–beta correlations across baseline, fear, and positive contexts.
Note: $N = 77$. Coupling was significant at all electrode sites in all contexts. The solid bar indicates a significant difference ($p < .05$) in coupling between episodes.

In contrast, delta–beta coupling was significant for high-reactive infants at nearly all electrode sites in the baseline and fear episodes. Additionally, high-reactive infants showed a pattern, across all electrode sites, for greater coupling during both the baseline (frontal: $z = 2.60$, $p < .01$, central: $z = 1.79$, $p < .05$, parietal: $z = 3.06$, $p < .01$) and fear episodes (frontal: $z = -2.00$, $p < .05$, central: $z = 1.53$, $p < .10$, parietal: $z = -1.98$, $p < .05$) relative to the positive episode. Differences in coupling between the baseline and fear episode were largely nonsignificant. Thus, high-reactive infants showed generally high levels of coupling in non-positive episodes and appeared to modulate levels of coupling according to the degree to which episodes elicited levels of positive affect.

Finally, to understand whether different patterns in coupling reflected greater coupling in the high-reactive relative to the low-reactive group, we examined group differences (high reactive vs. low reactive) in coupling within each context at homologous electrode locations. High-reactive infants showed greater coupling than low-reactive infants during baseline at all electrodes (frontal: $z = -1.59$, $p = .05$, central: $z = 1.76$, $p < .05$, parietal: $z = 3.14$, $p < .01$). High-reactive infants also showed greater coupling than low-reactive infants

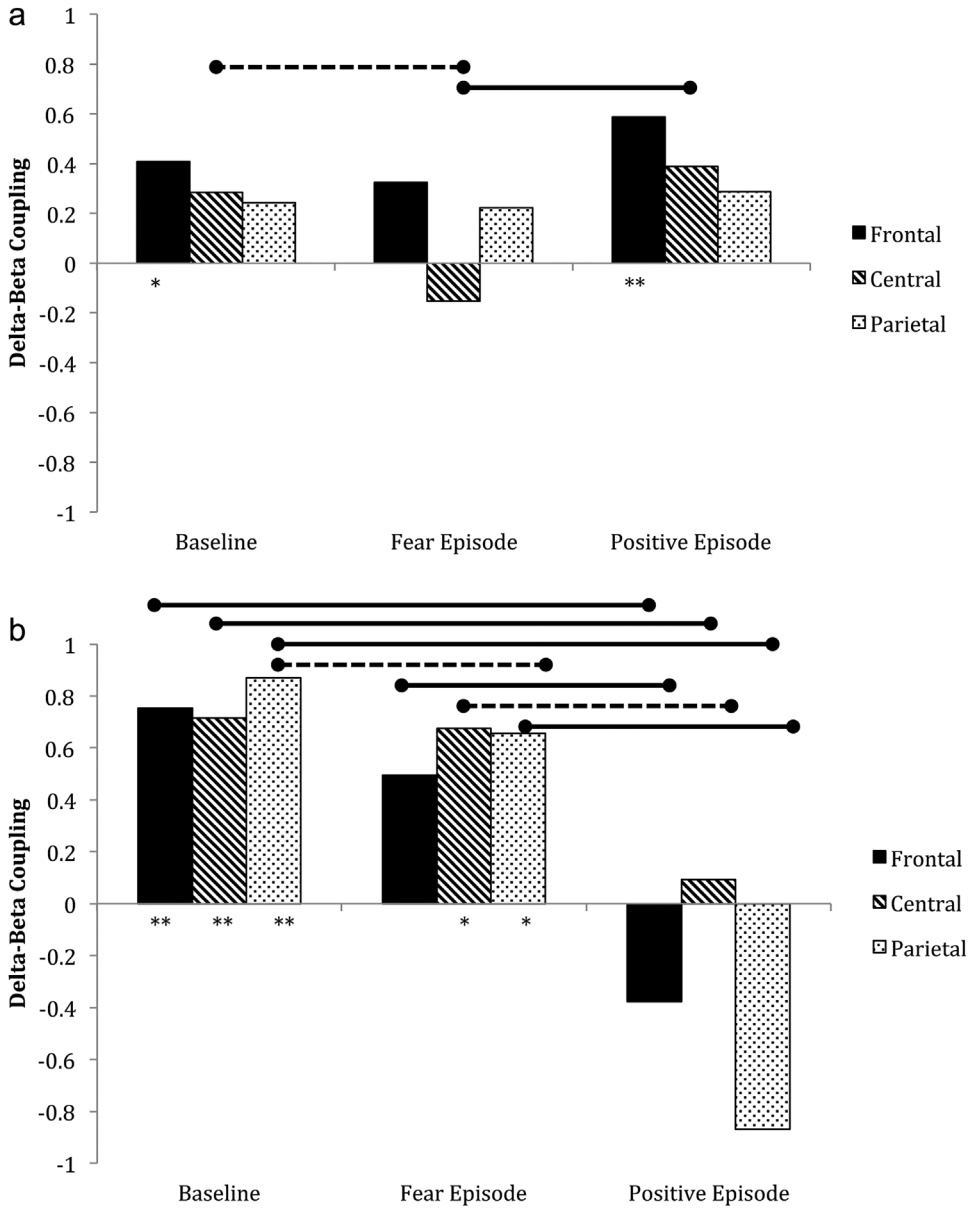

FIGURE 2 Delta–beta correlations across contexts for (a) low reactive and (b) high reactive infants.
Note: Dashed bars indicate trends ($p < .10$) and solid bars indicate significant differences ($p < .05$) in coupling across episodes. Asterisks denote significant correlations ($^{*}p < .05$, $^{**}p < .01$). $N = 27$ low reactive infants; $N = 16$ high-reactive infants.

during the fear episode at parietal ($z = -1.39$, $p = .05$) and central sites ($z = 2.46$, $p < .01$). Coupling was greater for high-reactive relative to low-reactive infants in the positive episode only at frontal sites ($z = -1.98$, $p = .02$). These results suggested that differing patterns of coupling across contexts for high- and low-reactive infants were generally related to greater coupling in high-reactive infants relative to low-reactive infants.

DISCUSSION

Our strategy for interpreting statistical results was to focus on internally-replicated patterns of findings; that is, we interpreted patterns of findings that were similar across electrode sites. Contrary to our hypotheses, context-based differences in coupling were largely absent in the sample as a whole. This finding was,

however, qualified by the presence of group differences in delta–beta coupling across contexts. Patterns of coupling across contexts differed for infants who showed high levels of cortisol reactivity relative to infants who showed low levels of cortisol reactivity.

Consistent with expectations, patterns of coupling across emotion-eliciting episodes differed based on infants' levels of cortisol reactivity. Infants who showed low levels of cortisol reactivity during the laboratory visit did not differ in delta–beta coupling across emotion contexts. This lack of contextual distinction occurred despite behavioral data suggesting that low reactive infants showed levels of fear and positivity similar to infants with high cortisol reactivity. Such findings may reflect a higher threshold of engagement for, perhaps also greater efficiency in, the use of physiological resources during periods of negative emotion. From an emotion-regulation perspective, one might speculate that this low-reactive subset of infants was able to cope with fear responses to the stranger with less "regulatory effort" as indexed by both cortisol reactivity and delta–beta coupling. This interpretation is consistent with work that has examined coupling in a baseline and anxiety-eliciting episode in adults (Miskovic et al., 2010). This interpretation is also consistent with examinations of neural correlates of cognitive control in anxious and at-risk children and adults, who show higher amplitudes of neural measures indexing performance monitoring (Brooker & Buss, 2014; Hajcak, McDonald, & Simons, 2003; Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; Torpey, Hajcak, & Klein, 2009) and inhibitory control (Buss et al., 2011; Dennis & Chen, 2009) relative to low-anxious and low-risk individuals. Similar to the current work, these effects are routinely observed in absence of group differences in behaviors indexed by performance on laboratory tasks.

Infants who showed low levels of cortisol reactivity also displayed a general lack of significant coupling across electrode sites and contexts. Again, this may reflect the absence of a perceived need to employ active coping strategies, and a greater preservation of physiological resources. It is important to note that is not possible, with these data, to determine whether this pattern of response is truly adaptive or maladaptive for childhood outcomes. From one perspective, hyper-reactivity in both neural and neuroendocrine systems have been associated with greater risk for physical illness and internalizing psychopathology (Ladouceur et al., 2006; Monk et al., 2008; Sapolsky, 1998), and theories of early sensitization suggest that tendencies for hyper-responsiveness tend to persist across early life (Gunnar & Quevedo, 2007; Shirtcliff & Ruttle, 2010). Thus, the appropriate modulation of physiological responses to the eliciting context may be a key factor for adaptation in early life. On the other hand, tendencies for under-reactivity to emotion-eliciting stimuli have been associated with disorders characterized by aggression and externalizing early in life (Raine, Venables, & Mednick, 1997). Such work implies that low levels of reactivity do not invariantly predict better outcomes. Future work that can re-examine and extend current findings in a greater number of emotion-eliciting contexts and in association with longitudinal outcomes in children is needed to clarify which may be the optimal patterns of physiological responses to emotion contexts.

In contrast to low-reactive infants, those infants who showed high levels of cortisol reactivity to the laboratory visit demonstrated significant modulation of delta–beta coupling across contexts. That is, high-reactive infants showed greater coupling in non-positive (i.e., baseline, fear) contexts relative to a positive context. This pattern was replicated across all electrode sites. Our findings suggest that infants' increased neural processing in the form of coupling was enhanced in contexts that were not explicitly positive. Though it is not possible given our sample size and coding schemes, it would be interesting to investigate whether infants who showed less positivity, or even high levels of fear in a non-fear context, showed robust patterns of association between delta–beta coupling and neuroendocrine response. Such a pattern would be expected based on past research (Buss, 2011; Buss et al., 2013; Phelps, Brooker, & Buss, in press) and would further elucidate the role of individual differences in emotion-related coupling.

It is, perhaps, somewhat surprising that no differences emerged between levels of coupling in the baseline and fear episodes. Given established regulatory differences associated with coupling in baseline paradigms, one might only expect context-based differences to emerge if coupling *increased* from baseline to the fear episode. However, in our study and in previous work, coupling measures during baseline are already quite high, particularly for high-reactive children (Miskovic et al., 2011a; van Peer et al., 2008). Thus, the lack of distinction between the baseline and fear contexts may, to some extent, be a statistical artifact.

Additionally, the difficulty of establishing a fully neutral baseline, particularly for high-reactive individuals, is well-known among developmental psychologists and psychophysiologists alike. Unlike cortisol collection, EEG assessments cannot be completed at home by parents. Thus, it is unavoidable that the baseline assessment involved some of the same elements as our fear-eliciting paradigm: meeting new people in a new place. Even the implementation of multiple baseline episodes would not have diminished this issue, given

individual differences in the degree to which infants adapt to the laboratory and return to their own baseline state. Although standardized procedures distinguish the male stranger from the female experimenter by allowing the female experimenter to conduct herself in a more animated, friendly, less-threatening manner, it is impossible to completely ameliorate the infant's experience of meeting someone new. Thus, some overlap in the infants' experience in the baseline and fear episodes may also contribute to similar levels of coupling across the two episodes.

Given its consistency with past research, our finding that high-reactive infants showed greater coupling than low-reactive infants in the non-positive episodes is also notable. This difference was most evident during the baseline episode, but emerged similarly in the fear context. These results replicate previous findings using baseline measures in adults. In addition, this pattern of results suggests that high-reactive infants did not simply show greater neural efforts at regulation (i.e., delta–beta coupling) than did low-reactive infants, as group differences were largely absent during the positive episode. Rather, high-reactive infants showed greater coupling in non-positive contexts, both relative to a positive context and relative to low-reactive infants. Again, this is the case despite a lack of differences in observed emotion across the two groups.

Finally, our results provide new evidence for associations between cortisol and delta–beta coupling in early life. Consistent with a view of cortisol reactivity and delta–beta coupling as denoting aspects of emotion processing (Gunnar et al., 1989, 2009; Knyazev & Slobodskaya, 2003; Knyazev et al., 2006; Knyazev, 2007), our work provides convergent evidence of greater global engagement of physiological systems in those infants we labeled "high-reactive." The cascade by which cortisol release by the HPA axis comes to impact delta–beta coupling is not entirely clear. Previous work using experimental designs suggests that cortisol can act as a causal agent that modifies neural activity, increasing withdrawn, inhibited behaviors (Sapolsky, 1998; van Peer et al., 2008). Our design does not allow for conclusions about causality but does suggest that this association between neuroendocrine response and neural function is already present in the first year of life when both systems are still undergoing rapid developmental change. Future work should investigate the degree to which the information about emotion processing provided by these different systems is overlapping versus distinct.

Our findings contribute to broader theories of emotion and emotion regulation. First, our results suggest that individual differences in propensities for high and low levels of responding in emotion contexts are evident from very early in life. This notion is consistent with traditional definitions of temperament (Goldsmith et al., 1987), which suggest that such tendencies are constitutional and from early in infancy. This idea is also consistent with theories about the development of basic emotions, including fear and positive affect, which are not only early-emerging, but also highly sensitive to social context (Campos, Mumme, Kermoian, & Campos, 1994; Saarni, 2008). Importantly, our work suggest that propensities for emotion responses are already present on a *systemic* level when infants are 6 months old. Thus, physiological processes that support emotion processing (Levenson, 2003) appear to work in a convergent fashion even as systems develop and mature.

Second, our work extends previous research aimed at distinguishing emotion regulation from dysregulation using contextual information (Buss, 2011; Cole et al., 1994; Locke, Davidson, Kalin, & Goldsmith, 2009). This work has largely attempted to distinguish non-normative emotion responses and behaviors that may be indicative of risk from more typical patterns. Here, we offer the opportunity to identify normative and aberrant trajectories using convergent measures of physiology. Whether physiology reflects an underpinning or an additional expression of emotion (Levenson, 2003), our pattern of results was not visible at the level of expressed emotion in infants. Rather, the distinction between tendencies for emotion responding appeared at a level that is not apparent through traditional observational measures.

One challenge for extending this work will be to determine what constitute "normative" patterns of coupling across contexts. In our work, both the high-reactive infants and the sample as a whole showed significant levels of coupling across most contexts. However, low-reactive infants were more representative of the overall sample in relative differences in coupling across contexts. It may be the case that high levels of neuroendocrine activity are more valuable for elucidating infants' abilities to regulate as contextual demands change. However, as previously indicated, without additional studies for comparison, it is difficult to determine whether the high-reactive or low-reactive group might be considered to be non-normative or aberrant from expectations about typical development.

Despite its contributions, the current study is not without limitations. First, despite conducting two visits to maximize the amount of usable data, we had a relatively small sample size. Although our sample *N* was comparable to or larger than that in other EEG studies of infants and young children, small effects will not be detected. Second, affective coding is not available for the baseline episode; this limits inferences

that can be drawn about the emotional significance of differences in delta–beta coupling between baseline and emotion episodes. Future studies that include videotaping and coding data from baseline episodes would be useful for addressing this issue. Finally, our analyses do not include childhood or adolescent outcomes for infant participants. Thus, it is unclear whether the patterns of cortisol reactivity and delta–beta coupling observed here have implications for adjustment later in childhood or for long-term mental health.

In sum, our findings suggest that delta–beta coupling in emotion-eliciting contexts may be useful for capturing individual differences in emotion processing, particularly when combined with neuroendocrine measures. Using this approach, we provided convergent evidence that infants who showed high levels of cortisol during a laboratory visit also showed greater delta–beta coupling in nonpositive relative to positive contexts and relative to low-reactive infants. We presented evidence these associations during the first year of life, before either system has fully developed. This work contributes to a growing literature on markers of emotion processing in the developmental, psychophysiological, and emotion literatures.

NOTES

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