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Children's Context Inappropriate Anger and Salivary Cortisol

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Abstract

Some children show emotion that is not consistent with normative appraisal of the context and can therefore be defined as *context inappropriate* (CI). The authors used individual growth curve modeling and hierarchical multiple regression analyses to examine whether CI anger predicts differences in hypothalamic-pituitary-adrenal axis activity, as manifest in salivary cortisol measures. About 23% of the 360 children (ages 6–10 years, primarily 7–8) showed at least 1 expression of CI anger in situations designed to elicit positive affect. Expression of anger across 2 positive assessments was less common (around 4%). CI anger predicted the hypothesized lower levels of cortisol beyond that attributed to context appropriate anger. Boys' CI anger predicted lower morning cortisol and flatter slopes. Results suggest that this novel approach to studying children's emotion across varying contexts can provide insight into affective style.

Keywords

cortisol; emotion; children; context; anger

Emotional processes are integral to the development of behavior problems (Cicchetti, Ackerman, & Izard, 1995; Cole, Michel, & O'Donnell-Teti, 1994; Keenan, 2000). However, the fund of knowledge about behavioral and physiological correlates of maladaptive emotional responses is predominately based on observations of emotional reactivity to situations that normatively elicit a particular discrete emotion (*context appropriate* [CA] *affect*; e.g., Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988). Early differences in CA emotional responses modestly predict levels of maladaptive behavior (Prior, Smart, Sanson, & Oberklaid, 2000; Rende, 1993) and do not always predict behavior with much specificity (Biederman et al., 1990, 1993). Therefore, we might overlook potential differences in affective style by limiting assessment of emotional responses to appropriate contexts such as fear to unfamiliar persons and novel objects (Davidson, Jackson, & Kalin, 2000).

Although emotional expressions can be associated with prototypical incentives (i.e., fear with perceived threat, anger with blocked goals, sadness with lost goals, pleasure with safety; see Lazarus, 1991), emotional expression is not a simple reaction to the immediate context. In a given context, it is unlikely that one will observe the expression of a particular emotion by all individuals or by the same individual consistently (Ekman, 1984; Frijda, 1986; Scherer,

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1988). Emotion is also influenced by personal concerns or dispositions to express an emotion (i.e., temperament; Goldsmith & Campos, 1982; Lazarus, 1991), genetics (Deater-Deckard, Petrill, & Thompson, 2007; Goldsmith & Lemery, 2000), or environmental factors (e.g., cultural differences in socialization; Camras, Bakeman, Chen, Norris, & Cain, 2006). Therefore, some children will show emotional responses that are not concordant with normative appraisal of the context (*context inappropriate* [CI] *affect*). Affective responses are the observed behavioral expressions of emotion in a given situation. Although what is expressed may sometimes be an emotion other than what is truly felt, we suggest that which emotion being expressed is important for adaptive functioning.

An example of CI negative affect is a child showing unprovoked anger while playing with peers. A child who wishes to affiliate with peers typically shows positive affect and engaging behavior with the peers. Although it may be possible to discern or hypothesize a reason for anger in this situation (e.g., the child develops a subgoal of getting a toy from a peer), the angry response would not be an adaptive response for the goal typically associated with this context (i.e., playful interactions with others). Thus, such a CI emotional response is not necessarily senseless or random, but it is maladaptive given the dominant incentives in the situation and the way the context is typically interpreted.

Children with behavior problems sometimes have difficulty varying their emotional reactions across contexts differing in emotional incentives. Therefore, characterizing emotional responses across appropriate and inappropriate contexts may elucidate how emotion is associated with adaptive and maladaptive behavior. CI affect may reflect inflexible, dysregulated emotional patterns (Cole et al., 1994). We suggest that individual differences in CI (misplaced) anger responses may be relevant for development of externalizing forms of maladaptive behavior (Locke & Goldsmith, 2007).

Children's aggressive and antisocial externalizing behavior has been associated with low physiological arousal (e.g., McBurnett, Lahey, Rathouz, & Loeber, 2000; Ortiz & Raine, 2004). We also expect CI anger to be related to lower arousal indicated by attenuated basal and reactive hypothalamic-pituitary-adrenal (HPA) axis activity (cortisol). With some exceptions, studies have found aggressive or externalizing behavior related to decreased basal (e.g., McBurnett et al., 2000; Oosterlaan, Geurts, Knol, & Sergeant, 2005; Tennes & Kreye, 1985; van Goozen et al., 1998; but see Kruesi, Schmidt, Donnelly, Hibbs, & Hamburger, 1989) and reactive cortisol (e.g., Gerra et al., 1998; Granger, Stansbury, & Henker, 1994; Moss, Vanyukov, & Martin, 1995; Popma et al., 2006; van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000; but see Davies, Sturge-Apple, Cicchetti, & Cummings, 2007; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997). In some studies, externalizing behavior was related to only one of the cortisol measures (prestressor or reactive cortisol; e.g., Davies et al., 2007; Granger et al., 1994; van Goozen et al., 1998, 2000). When the HPA axis is activated, a wellorchestrated release of chemicals along the HPA axis culminates in cortisol output from the adrenal gland (Swanson, Sawchenko, Lind, & Rho, 1987; Vale, Spiess, Rivier, & Rivier, 1981). Although a neuroendocrine negative feedback system regulates the HPA axis (Herman & Cullinan, 1997; Keller-Wood & Dallman, 1984), individuals can differ in activation and regulation of the HPA axis. Therefore, lower levels of cortisol can occur from dysregulation in the reactive or regulatory components of the HPA axis. For example, some individuals may have (a) lower production of cortisol or its hormonal antecedents (e.g., CRH, ACTH), (b) down-regulation of CRH or ACTH receptors because of increased circulating levels of these hormones, or (c) greater regulation of the HPA axis through increased neuroendocrine negative feedback (Heim, Ehlert, & Helhammer, 2000; see also Gunnar & Vazquez, 2001).

Children's cortisol levels have been related to the cognitive processes important for accurate perception and appraisal (e.g., Annett, Stansbury, Kelly, & Strunk, 2005; Lupien et al., 2005; Quas, Bauer, & Boyce, 2004). Perception and appraisal of emotional incentives may be particularly relevant for appropriate anger responses. In other words, children need to recognize and understand the emotional elicitors in the environment to appraise the context appropriately. In early childhood, children can identify and understand one's own and others' emotions (Denham, 1998; Halberstadt, Denham, & Dunsmore, 2001; Saarni, Mumme, & Campos, 1998). Although younger children can associate emotions with certain situations, the more complex situational understanding achieved by middle childhood is based on learned associations and past experiences, increased consideration for eliciting factors, and use of goal expectations (Saarni et al., 1998). Consequently, individual differences in HPA axis activity may be associated with the maturation of emotional behavior in part through its associations with the cognitive processes important for perception and appraisal.

If the HPA axis can influence emotional behavior through its impact on perception or appraisal of the context, it may be important to consider anger responses within specific anger-eliciting or non-anger-eliciting contexts to understand the relationship between HPA activity and individual differences in anger behavior. We do not expect cortisol would be related to overall anger reactivity, without consideration for the emotional context of the anger response. Research on the relationship between anger temperament and basal or reactive cortisol to stressors has primarily measured anger temperament as anger behavior in contexts that can include many emotional incentives. For example, some studies have used parent report measures of anger temperament (e.g., Davis, Donzella, Krueger, & Gunnar, 1999), which measure anger across many contexts, whereas others have measured anger behavior in play contexts (e.g., Granger et al., 1994; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997; Legendre & Trudel, 1996), which can contain both positive and anger-eliciting cues. This study is unique because it measures anger in standardized contexts designed to elicit either anger or positive affect in order to examine how anger in each of these contexts relates to basal and reactive HPA axis activity. Because the HPA axis normally reacts to frustrating or conflict situations (Levine, 1985), we may find greater HPA axis activity (basal or reactive) associated with more anger in anger-eliciting (CA anger) contexts. Reactive forms of aggression in CA contexts, such as provocation by others, may be linked to increased HPA axis activity (Kalin, 1999; McBurnett, King, & Scarpa, 2003), whereas lower arousal (decreased HPA axis activity) may be associated with CI anger, such as proactive instrumental aggression used to access something considered rewarding in the environment (McBurnett et al., 2003). Consequently, we expect CI anger to be related to decreased levels of basal and reactive HPA axis activity.

Because we measured change in basal cortisol across the day, this study may clarify whether past associations between externalizing behavior and cortisol were idiosyncratic to the sampling period used or reflect a more general association between typical daily cortisol and behavior (van Goozen, Snoek, Fairchild, & Harold, 2007). Normally the daytime portion of the diurnal rhythm in cortisol decreases across the day to reach a nadir in the hours just prior to midnight (Weitzman et al., 1971), acting independently from the influence of stressors. Therefore, the daytime pattern of basal cortisol may be influenced by both circadian factors and sensitivity of HPA axis inhibition (Keller-Wood & Dallman, 1984). Thus, a flatter slope of cortisol level across the day may be related, in part, to dysregulation in the decrease in cortisol production that normatively occurs during feedback inhibition. A flatter diurnal rhythm in cortisol may be reflected in levels of cortisol that remain high across the day, as seen in individuals with clinical depression (Ferrier, 1994). Alternatively, a flatter slope in cortisol may be related to low initial levels of daily cortisol (morning peak).

Typical morning cortisol levels appear to be influenced by genetics (e.g., Kupper et al., 2005) and/or chronic stress (Gunnar & Vazquez, 2001). Morning increases in cortisol promote

arousal and engagement with the environment (Gunnar & Vazquez, 2001), which may be necessary components of proper emotional and social development. Specific cognitive processes important for appropriate perception and appraisal may be influenced by mineralocorticoid receptor and glucocorticoid receptor binding (Lupien & McEwen, 1997). For example, individuals with low basal levels of cortisol (reflected in lower mineralocorticoid receptor binding; de Kloet, 1991; Gunnar & Talge, 2008) may have deficits in mineralocorticoid receptor binding influences on selective attention for emotionally relevant information, whereas individuals with lower cortisol increases to stressors or upon awakening (lower glucocorticoid receptor binding; de Kloet, 1991; Gunnar & Talge, 2008) may have lower glucocorticoid receptor binding influence on consolidation and recall of memories for emotional contexts. Consequently, it may be important to measure basal cortisol at morning and throughout the day. Few studies on the relationship of basal HPA axis activity and externalizing behavior have measured the change in cortisol levels across the day (Fairchild et al., 2008; Klimes-Dougan et al., 2001; Popma et al., 2007; Van den Bergh, Van Calster, Puissant, & Van Huffel, 2008).

Our study is rare in that it includes both basal and reactive cortisol levels, which is important if we want to clarify if aggressive behavior is associated with generally decreased HPA axis activity or primarily during stressors (van Goozen et al., 2007). We propose that decreased basal and reactive cortisol responsivity may be associated with the expression of CI anger responses. The dysregulation of HPA axis activity associated with CI anger responses could follow differing developmental trajectories stemming from early biological vulnerability or environmental stressors. Children who show CI anger may have impaired emotional maturation and a general pattern of attenuated HPA axis activity. Across development, this typical pattern of attenuated HPA axis activity could then impair the cognitive processes involved in perception of situations, perhaps leading to expression of CI anger and inhibited HPA axis reactivity. In addition, the exposure to more stressful, chaotic environments produced by anger expressed across many contexts may negatively impact the development of HPA axis activity in some children (Gunnar & Vazquez, 2001). Early stress may impact the neurobiological systems involved in HPA reactivity to stressors (amygdala), leading to impaired interpretation of threatening contexts and limiting HPA reactivity (Susman, 2006). As a result, such developmental negative influences on the HPA axis may further impact the expression of CI anger because a responsive increase in HPA axis reactivity to stress may be needed for accurate perception of the context. Therefore, we may find that children with greater CI anger have lower basal morning cortisol values, flatter slopes in diurnal rhythm, and lower cortisol reactivity compared with their peers.

In sum, the development of aggressive or antisocial behavior may stem from early vulnerability (e.g., difficult temperament) exposed to a stressful environment (Susman, 2006; van Goozen et al., 2007), perhaps mediated by physiological and emotional—cognitive deficits (and the interaction between them; van Goozen et al., 2007). By including the context of emotional responses, our study may elucidate a form of dysregulated emotional behavior and associated HPA axis activity that mediates the pathway to maladaptive aggressive behavior.

Study Overview

The little research done on the topic suggests that CI affect (a) is associated with toddler attachment style differences (Kochanska, 2001), (b) relates to infant CA affect (Forman et al., 2003; Locke & Goldsmith, 2009), and (c) shows stability during early development (primarily CI joy but not CI distress; Kochanska, 2001). Our study assessed individual differences in HPA axis activity associated with behavioral expression of CI and CA anger in children. Because many of the factors possibly related to CI affect develop across the early years (e.g., emotional competence; see Saarni et al., 1998), we addressed these issues in middle childhood. Middle

childhood has potential advantages for this study. In middle childhood, neuroendocrine functioning is not affected by factors such as menstrual cycles or adult medications such as oral contraceptives. On the other hand, adrenarche is a period when adrenal androgens (e.g., dehydroepiandrosterone [DHEA]) increase, which is important because DHEA can impact cortisol influences in the brain (van Goozen et al., 2007). Because adrenarche can begin during middle childhood, there is the possibility that there could be an effect of associated increases in adrenal androgens on the HPA axis during middle childhood. Age was included in model fitting but removed if it was not a significant predictor in the final model.

As part of a more extensive project, we examined whether children who expressed CI anger differed from their peers in negative emotional reactivity in anger-eliciting contexts. We determined whether levels of CI anger predict cortisol independently of appropriate anger levels. We hypothesized that children who expressed CI anger during positive situations would show cortisol responses that the literature has linked with externalizing symptoms. If this is true, an implication of the hypothesis would be that externalizing symptoms reflect not simply a lot of anger but misplaced anger. In addition, demonstrating links of both CI anger and symptoms to a common physiological substrate (cortisol levels in this case) lends specificity to the hypothesis. That is, we hypothesized that inappropriate anger levels would uniquely predict lower basal and reactive cortisol beyond, or instead of, appropriate anger levels.

Method

Participants

Participants were 360 twins (174 boys, 186 girls; 90 monozygotic pairs, 64 same-sex dizygotic pairs, 26 opposite-sex dizygotic pairs). The children's ages ranged from 6 to 10 years (M = 8.33), but most (66%) were age 7 or 8 years. The study employed twins to allow examination of genetic factors, which are not the subject of this report. The children were primarily Caucasian (94%), with 1.5% African American and 4.5% multiracial. Parents had an average of 15.4 years of formal education (SD = 2.4). Families were primarily middle class (average annual income: $60,000–$70,000). Sixty percent of the sample was recruited after the children had participated in one of two earlier twin studies, and the remaining children were recruited through various other means such as newspaper advertising and birth records. A portion of the children recruited from birth records, 26.8% of the total sample, was preselected not to be at high risk for behavior problems. This selection is a conservative bias for the study's hypotheses. Overall, the selection in this project was for parents who were willing to cooperate fairly extensively in research. How this cooperation translated to children's behavior is unknown, but it did not prevent wide variability on our measures.

Behavioral Observation Procedures

Overview—Assessments were carried out during in-home and laboratory sessions. At the in-home session, each child participated in standardized behavioral observation measures from the Laboratory Temperament Assessment Battery (Middle Childhood Version 1.0; Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1993). The children participated in three Lab-TAB episodes in the home relevant to this study. During both visits, the negative emotion-eliciting episodes were alternated with neutral or positive episodes, in a fixed order. Briefly, the CA anger episode occurred after an episode measuring persistence, one CI anger episode occurred after an episode measuring compliance, and the other CI anger episode occurred after a task that ends positively. There were seven episodes between the CA anger episode and the first CI anger episode, and one episode between the two CI episodes. Every effort was made to return the child to a neutral baseline state prior to the administration of each episode. Children who showed anger during the positive episodes (CI anger) were not more likely to show anger during the game explanation period prior to the administration of the positive episode: Hungry

Hungry Hippos: Yates's corrected $\chi^2(1, N = 351) = 0.98, p > .05$; Balloon Bop: Yates's corrected $\chi^2(1, N = 350) = 2.29, p > .05$. The children were videotaped during two Pleasure episodes and one Anger episode. Most of the children participated in both Pleasure episodes (97.5%) and in the Anger episode (99.2%).

Pleasure episodes—The two episodes devised to elicit pleasure were called the Hungry Hungry Hippos and Balloon Bop games. During the 3-min Hungry Hungry Hippos episode, the experimenter and the child participated in three bouts of the highly energetic interactive table game, with the child winning each game. During the 3-min Balloon Bop episode, the experimenter and child hit and chased a balloon and tried to keep it from hitting the floor.

Anger episode—During the I'm Not Sharing episode, the experimenters unfairly distributed candy between themselves and the child. The manner of distribution followed a standardized increase in unfair distribution, culminating in the experimenter receiving all the candy.

Behavioral Scoring

Anger during the Anger episode—Affect was coded during the I'm Not Sharing episode during 10-s intervals. Coders rated the presence of resistance, peak intensity of facial anger (0–3 scale), bodily anger or frustration (0–3 scale), and anger vocalizations (0–3 scale). The presence of resistance was defined as whether or not the child attempted to get the candy from the experimenter. Latencies to the first anger response and the first anger vocalization were scored. Sad behavior variables (facial sadness, sad bodily behavior, sad vocalizations, and latency to first sad response and first sad vocalization) were also coded but not used in analyses for this study. Cohen's kappa for interrater reliabilities for anger behaviors during the Anger episode ranged from .71 to .93 (M = .80).

Anger during Pleasure episodes—Coders rated the presence and/or peak intensity of anger variables during 5-s epochs of the entire duration of the Hungry Hungry Hippos (three game bouts and counting periods) and Balloon Bop episodes. The peak intensity of facial expressions of anger was rated (0–2 scale). Anger behavior variables included the presence of bodily anger or frustration and anger vocalizations. Resistant behavior coded during the I'm Not Sharing episode was not relevant for coding during the Pleasure episodes. The rarity of anger behaviors during the Pleasure episodes justified coding presence instead of descriptive coding (intensity and latency) of anger variables. Cohen's kappa for interrater reliabilities for the anger variables during the Pleasure episodes ranged from .74 to .85 (M = .80).

Composite Variable Formation

Latency values were transformed to speed values as the inverse of the square root of latency, to approximate a normal distribution. If a behavior did not occur, the latency to that behavior was assigned as the maximum value for the episode.

Anger episode—Missing values on the I'm Not Sharing episode were replaced via the expectation-maximization algorithm as implemented in the SPSS Missing Values procedure (SPSS, 2005). For the 11 I'm Not Sharing episode variables, principal component analysis produced a two-factor solution accounting for 45% of the variance. The first factor reflected anger behavior (the presence of resistance, intensity of bodily anger or frustration, intensity of anger vocalizations, intensity of facial anger, and latencies to the first anger response and the first anger vocalization), whereas the second factor indicated mainly sad behavior (intensity of sad behavior, intensity of sad vocalizations, intensity of facial sadness, and latency to first sad response and first sad vocalization). The sad factor was not used in analysis for this project.

Observed contextually inappropriate anger—A composite was formed by aggregation of the anger variables expressed during the two Pleasure episodes. The anger variables included facial anger, bodily anger or frustration, and anger vocalizations. The two Pleasure episodes differed in the presence of anger, $\chi^2(1, N = 346) = 8.79$, $p = .003$. Anger responses were more likely to occur during the Hungry Hungry Hippos episode (14.7% of the children) than the Balloon Bop episode (12.7%).

Cortisol Procedures

Cortisol measures—During the period between the home visit (described above) and a laboratory visit, the children provided saliva samples using cotton swab procedures. For 3 consecutive days, saliva samples were collected within half an hour after waking, at 4 p.m., and at bedtime. Because noncompliance in timing of cortisol collection can influence the validity of the data (e.g., Broderick, Arnold, Kudielka, & Kirschbaum, 2003; Kudielka, Broderick, & Kirschbaum, 2003), we carefully explained to parents how and when to collect cortisol. At the home visit, parents were instructed to (a) take the child's first sample in the morning, within half an hour of waking and before the child brushed his or her teeth or drank or ate anything; (b) collect the afternoon sample at 4 p.m.; and (c) collect the third sample in the evening, before the child goes to bed and brushes his or her teeth. After collection, parents placed the samples in the freezer and transported frozen samples in a cooler bag to the laboratory on the laboratory visit.

Parents also completed a questionnaire for each day of collection to record the time each sample was collected, sleep behavior, health status, medication use, and eating behavior. None of the sleep, health, or eating behaviors were related to extreme levels of cortisol. Most of the morning values (72%) were collected within half an hour of waking ($M = 0.53$, $SD = 0.43$). On the basis of previous research concerning the effects of medication on cortisol levels (Essex, Klein, Cho, & Kalin, 2002), we examined medication effects and removed 3 children from further analyses who were taking stimulant laxatives or insulin.

Saliva samples for each child were also collected at the beginning and the end of the laboratory visit. Thirty-two participants did not participate in the laboratory visit. These children did not differ from the other children on level of CA anger, $F(1, 355) = 1.31$, $p > .05$, or CI anger, $F(1, 349) = 0.26$, $p > .05$.

Approximately a week after the home visit, the twins and their family attended a laboratory session. During the laboratory session, psychophysiological measures were collected during resting periods; three Lab-TAB episodes (i.e., conversation with a stranger, surprising parent with pop-out toy snake, and a disappointment paradigm); an aversive noise, emotion-modulated startle paradigm; and a challenging math task. The Lab-TAB episodes administered during the laboratory session were designed to elicit negative affect (inhibition during conversation with a stranger, anger or sadness during a disappointment paradigm) or pleasure (e.g., during anticipation and the act of surprising parent with a pop-out toy snake). We expect these tasks may have been construed as stressful because of unfamiliarity (e.g., unfamiliarity of the stranger), uncertainty (e.g., stranger's lack of conversational responses), or unpredictability or arousal (e.g., anticipation of surprising parent with pop-out toy snake, anticipation of getting favorite prize). The last event of the laboratory session was a challenging math paradigm. During the computer-administered mathematical task, the child viewed an instruction–practice period and then was presented with age-appropriate mathematical problems (e.g., addition problems) to answer. We formatted the task using an adaptive testing algorithm to maintain a constant degree of difficulty for each participant. The child was given 20 s to answer each problem, and the entire duration of the task was 5 min. During the task, the child was given immediate feedback on the accuracy of his or her answer (i.e., a *ding* sound for correct answers, a *zap* sound for incorrect answers).

The full laboratory session involved recording of additional physiological measures as part of a larger study, and these procedures could also be construed as stressful. Therefore, cortisol change from approximately 20 min postlaboratory entry to approximately 20 min after the last paradigm (mathematical task) should estimate the change in cortisol in reaction to the overall laboratory session, which contained several stressors.

All laboratory visits occurred during the early afternoon and lasted approximately 2.5 hr on average. The twins participated in the same sequence of assessments during the same period while in separate rooms. The first salivary cortisol sample was collected after the child heard an explanation of laboratory procedures (approximately 20 min postlaboratory entry). The second cortisol sample was collected approximately 15–20 min after the challenging math paradigm. A majority of the children (83.9%) provided at least two usable samples at each time of day at home; 79.2% provided a usable initial laboratory sample and basal afternoon sample, and 82.5% provided usable samples at the beginning and end of the laboratory visit. For each time of day, correlations across the 3 days ranged from .22 to .46 (all *p*s < .001). Table 1 reports the correlations among all cortisol measures collected in the home and during the laboratory visits.

Cortisol assay—Processing of saliva samples occurred in two stages. First, samples were thawed and centrifuged at 5,000 rpm for 10 min to extract the saliva from the cotton and then transferred to a 2-ml tube for storage (−80°C). Then, the samples were divided into batches to be assayed. To minimize variability, all samples from each child were assayed within the same batch and were assayed with reagents from the same lot. Samples were assayed for cortisol with a high-sensitivity enzyme immunoassay kit (Salimetrics, State College, PA) specifically designed for use with saliva. The sample test volume was 25 μl. The assay has a detection limit of 0.007 μg/dl and a range of sensitivity from 0.007 to 1.8 μg/dl. Two internal controls were included in each assay. The average interassay coefficient of variation (CV) was 7.4%, and the average intra-assay CV was 3.8%. All samples were tested in duplicate within the same assay. The average of the duplicate tests was used in the analyses. Results were considered acceptable if the CV of the duplicate samples was ≤20%. Repeat assays were performed on all samples not meeting this requirement. Twenty-two samples had above-threshold CV and insufficient saliva to be assayed again.

Statistical Treatment of Cortisol Data

Cortisol values were highly skewed, so outliers greater than three standard deviations from the mean were recoded to the value corresponding to three standard deviations from the mean as recommended by Barnett and Lewis (1994). Then values were log-transformed.

Because basal afternoon and laboratory values of cortisol were significantly correlated with sampling time (afternoon basal *r*s = −.19 to −.14; prelab *r* = −.23; postlab *r* = −.23), these values were regressed on sampling time, and standardized residuals were used in analyses. We then calculated two reactive laboratory values: the initial response to the laboratory visit and the reactive cortisol response across the laboratory visit. We calculated the initial laboratory value as the initial cortisol level at the laboratory minus the median afternoon basal cortisol level collected at home. This value should reflect the change in cortisol at the beginning of the laboratory visit from basal levels of cortisol. As noted by Smider et al. (2002), many past studies have treated resting measures of cortisol in the laboratory or clinic as basal measures, although these values may be influenced by stressor anticipation. Therefore, we considered the prelaboratory value (controlling for individual differences in basal cortisol) as a reactive measure.

We computed reactive cortisol as a difference score between cortisol levels at the end of the laboratory visit (postlab: *M* =−0.02, *SD* = 0.99; *n* = 297) and cortisol levels at the beginning

of the laboratory visit (prelab: $M = 0.01$, $SD = 1.00$; $n = 297$). The difference in raw cortisol levels from the prelaboratory to the post-laboratory measure ranged from −0.58 to 0.55 μg/dl ($M = 0.01$, $SD = 0.14$; $n = 297$). For descriptive purposes only, we calculated how many children increased or decreased by at least 15%, following the recommendation of Granger, Weisz, McCracken, Ikeda, and Douglas (1996). By these standards, 33.3% of the total sample showed cortisol increases, and 45.8% showed cortisol decreases after the initial measurement at the laboratory visit.

Data Analysis

CI anger and CA anger—We conducted two separate multiple regression analyses to examine whether levels of CI anger were related to CA anger levels: (a) We entered the presence of CI anger across the two positive episodes to predict levels of CA anger during an anger-eliciting episode, and (b) we entered levels of CI anger during at least one positive episode to predict levels of CA anger during an anger-eliciting episode.

Basal cortisol—We analyzed the associations of CA and CI anger with basal morning cortisol and diurnal rhythm of cortisol pattern by using individual growth curve modeling (Singer & Willett, 2003). We conducted all basal cortisol analyses using the SAS PROC MIXED full maximum likelihood method (SAS Institute, 2004). This technique allowed us to simultaneously model the morning cortisol and slope of diurnal rhythm in cortisol to determine whether levels of CI anger predict level of morning cortisol and the slope of the diurnal cortisol pattern beyond levels of CA anger. We required that cortisol levels be present for 2 of the 3 days for each time of collection (morning, 4 p.m., bedtime). In the models, the effect of within-subject variables (time of day) was included in Level 1. The time of day variable was centered on waking time, so that the intercept of initial status of cortisol in the models could be interpreted as cortisol at time of waking. For each participant, a total of nine basal cortisol samples were collected (morning, 4 p.m., and bedtime for 3 days). We entered all nine basal cortisol samples collected across 3 days in the model. Because we did not include in the model a level for day of collection, we did not include day effects on cortisol in the model. This method allowed us to model a participant's typical diurnal rhythm in cortisol across the day. By not modeling day effects, we limited any idiosyncratic day effects on diurnal patterns (Adam, 2006). We also examined day effects by fitting three-level models that include day of collection (dummy coded) to verify that results were similar to those of the two-level models. The three-level model results were similar to those of the two-level modeling designs, so we reported and interpreted the more parsimonious two-level model results.

Effects of between-subjects variables (e.g., affect variables, demographic variables) were included in Level 2. Models were fitted to cases that had affect data (valid CA and CI anger values). Following other designs using multilevel models of cortisol outcomes (Adam, 2006; Adam & Gunnar, 2001), we first addressed the influence of demographic and control variables on the cortisol outcome variables (e.g., illness, any medication use that day, any medication use in last week, type of medication, age, zygosity) by fitting models in which each of these predictors was entered in separate models at Level 2 to determine effect on the Level 1 cortisol outcome variables (morning cortisol and diurnal slope of cortisol). Then, only the demographic and control predictors that had significant effects on basal cortisol were included in the final model of affect predictors. Only age was associated with cortisol, with the diurnal slope of cortisol becoming flatter with advancing age. Therefore, age was entered into the model of affect predictors and retained in the final model if it remained a significant predictor. We entered the between-subjects affect predictors (CA and CI anger) as well as the interaction of sex and affect. If the CA Anger × Sex interaction or the CI Anger × Sex interaction was significant, we did follow-up bivariate correlations between the affect predictors and cortisol outcomes for boys and girls separately.

Reactive cortisol—To address the relationship between CI anger and CA anger and reactive cortisol, we conducted hierarchical multiple regression analyses. In three regression models, we entered sex in Block 1, CA anger level in Block 2, CI anger level in Block 3, the interaction of sex and CA anger in Block 4, and the interaction of sex and CI anger in Block 5 to predict the reactive cortisol outcome variables. If the CA Anger × Sex or the CI Anger × Sex interaction was significant, we performed follow-up bivariate correlations between the variables for boys and girls separately. Observed CI anger measures were not centered at the mean because they were not normally distributed. Interpretations of interactions with those variables were therefore considered with a mean intercept.

Use of Twins in the Analyses

Although the children in the sample were twins, the study considered subjects without regard for pair membership. Thus, the assumption of statistical independence was not strictly met in this study. However, age was the only variable in our analyses that was constant for members of all twin pairs. Moreover, of the 82 children who showed CI anger, only 17 pairs contained cotwins who showed CI anger in at least one positive situation. We also entered zygosity (monozygotic vs. dizygotic) in the analyses examining basal and reactive cortisol to explore any significant effects of zygosity. If zygosity was not a significant predictor, we did not include it in the final model of basal or reactive cortisol. Zygosity (monozygotic vs. dizygotic status) correlated at the levels of −.02, .01, .01 (all *p*s > .05), and .19 ($p = .001$) with basal morning cortisol, basal slope, initial change at laboratory, and laboratory cortisol reactivity, respectively. On the basis of these considerations, we do not regard dependencies created by twinship as an important bias in the analyses.

Results

Overview of Findings

Children's angry responses, including those that were inappropriate to the situational context, did generally predict cortisol measures. For the boys, higher levels of CI anger predicted lower morning cortisol and flattening of the diurnal slope of cortisol compared with lower levels of CI anger.

Frequency of Anger Expressed During Positive and Negative Episodes

A key premise of this approach is that CI anger is relatively infrequent, or nonnormative, and thus a plausible marker for dysfunction. Thus, we first addressed the simple question of the frequency of contextually inappropriate anger. Figure 1 shows the percentage of children who expressed anger during the entire Pleasure and Anger episodes. Frequencies of facial, bodily, and vocal anger behaviors are reported for the entire duration of the Pleasure and Anger episodes. As expected, the number of children expressing discrete anger was substantially higher for the Anger episode than the two Pleasure episodes.

Turning from discrete behaviors in Figure 1 to anger composite scores, we found that some degree of anger was shown by about 96% of the children in the I'm Not Sharing (anger) episode. The percentage of children who showed CI anger (i.e., in the Pleasure episodes) was much lower. Approximately 23% ($n = 82$) of the children showed at least one indication of anger in one of the two positive games. The variable of interest for this study was level of CI anger ($M = 0.001$, $SD = 1.45$; range = −0.54 to 11.88). No sex or age differences occurred for level of CI anger, $F(1, 349) = 0.002$, $p > .05$; $r(350) = -.09$, $p > .05$, respectively.

Approximately 4% ($n = 13$) of the children showed anger across the two positive episodes. Thus, the strict criterion for CI anger expression provides a substantially more nonnormative classification than the liberal criterion described in the previous paragraph. No sex differences

occurred for the expression of CI anger, $\chi^2(1, N = 351) = 0.13$, $p > .05$. Younger children (below the mean age, $n = 10$) were more likely to show anger in both of the positive episodes, $\chi^2(1, N = 351) = 4.3$, $p = .04$, than older children ($n = 3$).

Associations Between CI Anger and CA Anger

After verifying that CI anger exists, we examined its relationship to individual differences in CA anger using multiple regression analyses. CI anger levels during at least one positive episode were moderately related to observed CA anger ($R^2 = .13$, $\beta = .37$), $F(1, 347) = 53.75$, $p < .001$. CI anger levels during both positive episodes were also moderately related to observed CA anger ($R^2 = .05$, $\beta = .23$), $F(1, 342) = 18.72$, $p < .001$. Thus, CI and CA anger were only moderately related. If CI and CA anger had been highly correlated, we would have little justification for treating them as separate constructs.

Associations Between CA and CI Anger and Basal Cortisol

Table 2 shows the model examining the relationship between affect (CA and CI anger) and morning cortisol levels and the slope of the diurnal rhythm. On average, children showed the expected linear decrease in cortisol across the day (slope). Because sex was dummy coded (0 = male, 1 = female) and affect variables were centered at the mean, a boy with average levels of CA and CI anger had average morning cortisol of 0.35 μg/dl (intercept of initial status) and 15% average decrease in cortisol per hour (intercept of slope of diurnal rhythm in cortisol). Sex was a significant predictor of morning cortisol; girls averaged 18% greater morning cortisol than boys. Boys and girls also differed on the slope in diurnal rhythm, with girls generally showing a steeper slope in cortisol across the day (2% more of a decrease per hour). For every unit change in CI anger, children with greater CI anger showed a 7% lower morning cortisol level and 1% less of a decrease in cortisol per hour across the day (flatter slope) than children with less CI anger. CA anger also tended to predict morning cortisol and slope; each increase of one unit of CA anger predicted a 10% increase in morning cortisol and 1% greater decrease in cortisol across the day (steeper slope).

The variance components of the model (see Table 2) indicate that (a) there was significant residual variability in within-subject cortisol and (b) variability in basal morning cortisol and slope remained after we included model predictors of sex, CA anger, CI anger, and the interactions of sex with CA anger and sex with CI anger.

To assess the amount of variance in cortisol explained by the between-subjects predictors, we compared the final model with all between-subjects predictors (sex, CA anger, CI anger, CA Anger × Sex interaction, CI Anger × Sex interaction; shown in Table 2) with an unconditional growth model fitted with only the time of cortisol collection variable (not shown in Table 2). The variance components of the unconditional growth model can be compared with the final model to determine how much variability in cortisol was explained by the between-subjects predictors (sex, CA anger, CI anger, CA Anger × Sex interaction, CI Anger × Sex interaction). When the final model was compared with the unconditional growth model, 8% of the variance in morning cortisol and 7% of the variance in the slope in cortisol were explained by the between-subjects predictors (sex, CA anger, CI anger, CA Anger × Sex interaction, CI Anger × Sex interaction).

Because age was not a significant predictor of slope in cortisol in the model with sex and affect predictors, we removed it from the final model. However, there was a significant interaction of sex and affect on morning cortisol (marginal) and diurnal rhythm in cortisol, justifying examining the relationship between the affect predictors (CA anger and CI anger) and cortisol outcome variables for each sex.

Sex Differences in the Relationship Between CA and CI Anger and Basal Cortisol

For boys, levels of CI anger were negatively correlated with morning cortisol levels, $r(148) = -.17$, $p < .05$, indicating that boys with greater CI anger had lower basal morning cortisol than boys with lower levels of CI anger. There was also a positive relationship between boys' CI anger and slope of the diurnal rhythm in cortisol, $r(148) = .16$, $p < .05$, indicating that boys with greater CI anger had less decline in cortisol across the day (flatter slope) than boys with lower levels of CI anger. For girls, CI anger levels were not related to morning cortisol, $r(143) = .01$, $p > .05$, or to the slope in diurnal rhythm in cortisol, $r(143) = -.14$, $p > .05$.

Figure 2 illustrates the relationship of CI anger with morning cortisol and slope of diurnal rhythm for boys and girls. Using model parameters, we graphed four slopes representing cortisol change across the day for boys or girls with no CI anger or high levels of CI anger. A high CI anger value was defined by the lower limit of the upper 10th percentile of children showing CI anger. As can be seen in Figure 2, a boy with high levels of CI anger (dashed line) showed lower cortisol in the morning than boys with no CI anger or girls with no or high CI anger. Boys' CI anger was not related to afternoon or bedtime levels of cortisol. Therefore, the flatter slope associated with CI anger in the boys appeared to be influenced by differences in lower morning cortisol.

Association Between CA and CI Anger and Reactive Cortisol

Levels of CA and CI anger did not significantly predict cortisol reactivity across the laboratory session (see Table 3). Because zygosity was significantly related to cortisol reactivity, we also ran the full sample regression with zygosity as an independent predictor. As with the analyses without zygosity, CA anger and CI anger were not related to cortisol reactivity, and there was a trend for a CA Anger × Sex interaction. Follow-up analyses of a marginally significant interaction of CA anger and sex on cortisol reactivity showed that greater levels of CA anger and CI anger tended to predict lower cortisol reactivity in the boys, $r(148) = -.15$ and $r(148) = -.15$, $ps = .07$, respectively. Girls' CA anger and CI anger did not significantly predict cortisol reactivity, $r(143) = .12$ and $r(141) = .01$, $ps > .05$, respectively. Neither CA nor CI anger predicted cortisol level at the beginning of the laboratory visit ($\beta = -.05$, $R^2 = .01$, $\beta = .06$, $R^2 = .01$, $ps > .05$; not shown in Table 3).

Discussion

This study examined emotional responses across contextual changes to clarify associations with HPA axis activity. As predicted, anger was nonnormative in the positive contexts but common in contexts designed to elicit anger. Although only 4% of children showed anger in both of two positive situations, 23% showed some degree of anger in one of the positive situations. This study quantified the specific contributions of individual differences in CI anger levels to differences in HPA axis activity beyond those attributed to CA anger.

CI Anger Predicts Differences in HPA Activity

In the quest to understand how HPA axis activity is related to emotional differences, research has primarily focused on emotional reactivity in situations that normatively elicit a given emotion. We predicted that measuring emotional behavior in relation to context might resolve some of the inconsistencies in how emotional reactivity relates to cortisol measures. This study is the first to show that anger that is incongruent with the normative appraisal of a context is related to basal HPA axis activity levels, although the association only held in boys. Specifically, boys with greater CI anger had lower basal morning cortisol and less of a decline in cortisol across the day (flatter slope) than boys with lower levels of CI anger. Notably, CI anger predicted HPA axis activity differences beyond the prediction afforded by CA anger.

These findings are similar to those generally reported for externalizing behaviors, which often include behaviors incongruent with the normative appraisal of a context. Our finding of an inverse relationship between morning cortisol and CI anger converges with some studies of aggressive behavior and conduct disorder or oppositional defiant disorder symptomatology (McBurnett, Lahey, Capasso, & Loeber, 1996; McBurnett et al., 2000; Oosterlaan et al., 2005; Shoal, Giancola, & Kirillova, 2003; Tennes & Kreye, 1985; Vanyukov et al., 1993), conduct disorder or oppositional defiant disorder diagnosis (Pajer, Gardner, Kirillova, & Vanyukov, 2001; van Goozen et al., 1998), and callous or unemotional behavior (Loney, Butler, Lima, Counts, & Eckel, 2006). Similar to Popma et al.'s (2007) study on children with disruptive behavior problems, our study found that boys with more CI anger had lower morning cortisol levels and less of a decrease in cortisol across the day than their peers. However, the children in our study were not selected for any diagnosis or symptoms.

Our findings suggested a trend for boys with greater CA anger or CI anger to have lower cortisol reactivity at the laboratory than boys who showed less CA anger or CI anger. To investigate this possibility systematically, future research will need to employ a variety of distinctively different stressors. Lower cortisol reactivity (blunted responses) seen in some studies could be due in part to insufficient stressors (Gunnar & Vazquez, 2001). Research of this genre should clarify whether aggressive and antisocial children differ in their HPA responses to stressors in general or specifically to anger-eliciting stressors (van Goozen et al., 2007). Children with externalizing behavior have shown decreased cortisol reactivity to aggressive stressors, such as provocation (van Goozen et al., 2000).

Because past studies on aggressive and externalizing behavior have included cortisol collection outside the home (e.g., in clinics, schools, or laboratories), it is unclear whether these studies show that aggressive and externalizing children have lower trait cortisol levels or are less reactive to the setting (McBurnett et al., 1996). Our study is rare in that it included cortisol levels at home as well as prior to laboratory tasks. When we statistically controlled for basal home afternoon cortisol, we did not find an association between pretask levels of cortisol at the laboratory and CI anger. This suggests that trait levels of cortisol partially account for associations with behavior. Consistent with this suggestion, Shirt-cliff, Granger, Booth, and Johnson (2005) separated trait and state-related cortisol from home values across 2 years. Morning trait cortisol level was negatively related to boys' externalizing problems. Other studies have included measures of basal cortisol at home but did not directly examine the relationship of home cortisol and aggressive behavior (e.g., Dettling, Gunnar, & Donzella, 1999).

Does CI Anger Constitute High Anger Reactivity?

One possibility is that CI responding is akin to high levels of CA responding. Although some children exhibiting CI anger showed hyperreactive emotionality in appropriate contexts, this was not generally true. The expression of observed CI anger was only moderately associated with greater levels of CA anger. Thus, we conclude that CI and CA anger are relatively distinct.

Similar incongruence between CA and CI affect levels occurs in rhesus macaques (Kalin & Shelton, 2000). Normatively, rhesus macaques freeze in response to the presentation of a human profile, whereas the monkeys are more likely to act aggressive or submissive to human intruder stares. However, a small group of the monkeys displayed normative freezing behavior during the intruder profile condition as well as nonnormative freezing when the human intruder made direct eye contact. Individual differences in freezing behavior during a period of direct eye contact by a human intruder (CI behavior) did not predict levels of freezing during the period of human profile presentation (CA behavior). Some monkeys showed high levels of freezing during the profile condition but did not freeze during the stare condition, and monkeys that showed freezing behavior during the stare condition did not necessarily freeze at high

levels during the profile condition. Locke and Goldsmith (2009) also noted a similar pattern for human infant affect across varying situations. Although there was an overall trend for the infants expressing fear in positive situations to show high levels of fear in a social fear-eliciting situation as well, this relationship was primarily influenced by two infants with extreme reactivity in the fearful situation (Locke & Goldsmith, 2009). Thus, when we examine the fear domain in monkeys and human infants, some evidence for the generality of our finding of relative independence of CA and CI anger is present. Of course, showing a lack of convergence is akin to supporting the null hypothesis with its attendant limitations as a basis for inference.

Sex Differences in the Association of CI Anger and HPA Axis Activity

Sex differences for cortisol associations with behavior are not uncommon. For example, Shirtcliff et al. (2005) and Smider et al. (2002) found that levels of externalizing behavior were related to lower cortisol levels only in the boys. Likewise, only boys' aggressive and externalizing behavior was related to cortisol differences during preschool (Dettling et al., 1999; Tout, de Haan, Campbell, & Gunnar, 1998). Notably, many studies of cortisol differences in children at risk or diagnosed with externalizing disorders have been limited to boys, perhaps limiting our understanding of sex differences.

Sex differences in cortisol-behavior links may partially be due to the different prenatal hormones experienced by boys and girls. Prenatal hormone exposure influences neural organization. Some of the sex differences in brain organization (e.g., hypothalamus) may help explain sex differences in emotional behavior (van Goozen & Fairchild, 2006). Alternatively, if the increase in testosterone across development is greater in boys than girls (Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004), then boys and girls may differ in a possible mediator of externalizing behavior—the ratio of testosterone to cortisol (van Honk & Schutter, 2006).

On the other hand, sex differences in fear levels may explain the sex differences we found in the CI anger– cortisol link. This is plausible because children or adolescents with comorbid externalizing and internalizing have been shown to have greater levels of cortisol than children or adolescents high only in externalizing (McBurnett et al., 1991; Pajer et al., 2001; van Goozen et al., 1998; but see Oosterlaan et al., 2005; Schulz, Halperin, Newcorn, Sharma, & Gabriel, 1997). Notably, in preliminary data from our study, girls had greater levels of CI fear and internalizing behavior problems than boys. Thus, there may have been sex differences in how levels of internalizing behavior modulated the relationship between CI anger (externalizing behavior) and cortisol.

Boys and girls may also differ in cortisol associations with cognitive processes important to CA affect. Wolf, Schommer, Hellhammer, McEwen, and Kirschbaum (2001) found that in young adults, reactive cortisol and word recall were inversely related only in the men. Showing increased levels of cortisol to stress did not influence word recall in women. Therefore, boys and girls may differ in cortisol links to a cognitive process that may in turn affect perception and appraisal of the context.

We found that boys tended to have higher reactive cortisol at the laboratory session. Men are more likely than women to show greater cortisol reactivity to achievement-oriented stressors similar to the one in our study (e.g., mathematical stressor; Kudielka & Kirschbaum, 2005; Stroud, Salovey, & Epel, 2002), whereas women are more likely to have greater reactivity than men to psychosocial stressors (Kudielka & Kirschbaum, 2005). However, boys and girls do not differ in pharmacologically induced changes in cortisol (e.g., CRH infusion; Dorn et al., 1996). Therefore, sex differences may moderate the association of cortisol reactivity with behavioral responses only in certain situations.

Limitations and Proposed Directions for Future Research on CI Affect

Although our study provides some insight into the behavioral and neuroendocrine correlates of CI anger, it has some limitations. Although we have no reason to expect that results with twins will fail to generalize to the nontwin population, this possibility does exist. Also, even behavioral measures that are well designed are small samples of behavior, and lack of a relationship between a behavior and other measures could conceivably be due to inadequate behavioral sampling. However, positive results, such as those we report, are realized in spite of this limitation. Another caution is that the effect sizes we report for associations between anger and cortisol are modest; however, both behavioral anger and cortisol secretion are multidetermined responses, and strong associations would not be anticipated. Van Goozen et al. (2007) found that the average effect sizes across multiple studies on the link between aggression and disruptive behavior disorder symptoms and basal cortisol were low to moderate. Furthermore, although we were able to identify those children who expressed anger across two positive situations, the rarity of this behavior (4%) limited the ability to extend analyses of these children beyond the descriptive level.

As reported for children in stressful early environments (see Gunnar & Vazquez, 2001), we found that the flatter slope in cortisol diurnal rhythm accompanied a lower morning peak value. Notably, children with greater CI anger may have shown a flatter slope of cortisol because they started the day with lower morning levels than their peers with lower CI anger. In our study, morning cortisol was inversely related to the slope in change in cortisol ($r = -.70$). Furthermore, CI anger was not related to afternoon or bedtime cortisol levels, indicating that it was primarily morning cortisol that differed among CI anger levels.

The novelty of examining CI affect means that many issues require consideration in further research. Emotional expression is influenced by many personal concerns and various temperamental traits. Moreover, multiple, sometimes subtle, emotional incentives can inhere in laboratory contexts and especially in natural contexts. Even in situations devised to elicit specific discrete emotions (e.g., pleasure), other emotion-eliciting factors (e.g., competition) may be present. Although anger may not be adaptive in a pleasurable, competitive context, such a response might be appropriate for children highly motivated to compete. Future research should consider such multiple incentives to explain the personal concerns influencing emotional expression.

Although empirical examination of such issues is beyond the scope of this article, regularly expressing CI anger might be expected to lead to lost opportunities for interaction and to contribute to social isolation or peer rejection. For example, children who showed more anger in nonaggressive contexts, or more happiness in aggressive contexts, were less accepted by their peers than other children (Arsenio, Cooperman, & Lover, 2000). Conversely, levels of anger in aggressive contexts were not related to aggressive behavior or peer acceptance. Peers may view angry responses as appropriate and happiness as inappropriate behaviors in aggressive contexts. Furthermore, angry or rejecting peer responses to CI anger may facilitate more anger responses from a child showing CI anger, thereby perpetuating the angry interaction. In general, children showing CI anger may make different attributions of the context than their peers. For example, they may be more likely to attribute hostility to neutral or ambiguous situations (hostile attribution bias; Dodge & Coie, 1987) or have poorer reading of peer intentions (Dodge, Murphy, & Buchsbaum, 1984). These findings indicate the importance of the context of emotional responses when examining emotional correlates of competent behaviors (Arsenio et al., 2000).

This study was able to identify CI affect in a normative sample. Because the ability to perceive contextual complexity may be important for CI affect, systematic investigation of children's emotional processing (e.g., understanding of emotional elicitors) accompanied with measures

of HPA axis activity would be a prudent next area of investigation. If children showing CI anger have deficits in emotional understanding, they may benefit from early intervention programs that target emotional understanding processes (Denham & Burton, 1996). Alternatively, children showing CI anger that had early stress exposure may benefit from family-based therapeutic interventions that influence cortisol level (Fisher, Stoolmiller, Gunnar, & Burraston, 2007). The rarity of CI affect means investigations of this emotional response style would benefit from preselection procedures, such as those in many studies of behavioral inhibition (e.g., Garcia Coll, Kagan, & Reznick, 1984; Pfeifer, Goldsmith, Davidson, & Rickman, 2002).

If confirmed and extended in future studies, the type of finding that we report—CI affect associated with physiological measures that are implicated in dysregulated behavior—should shed light on the links between emotion and behavior problems.

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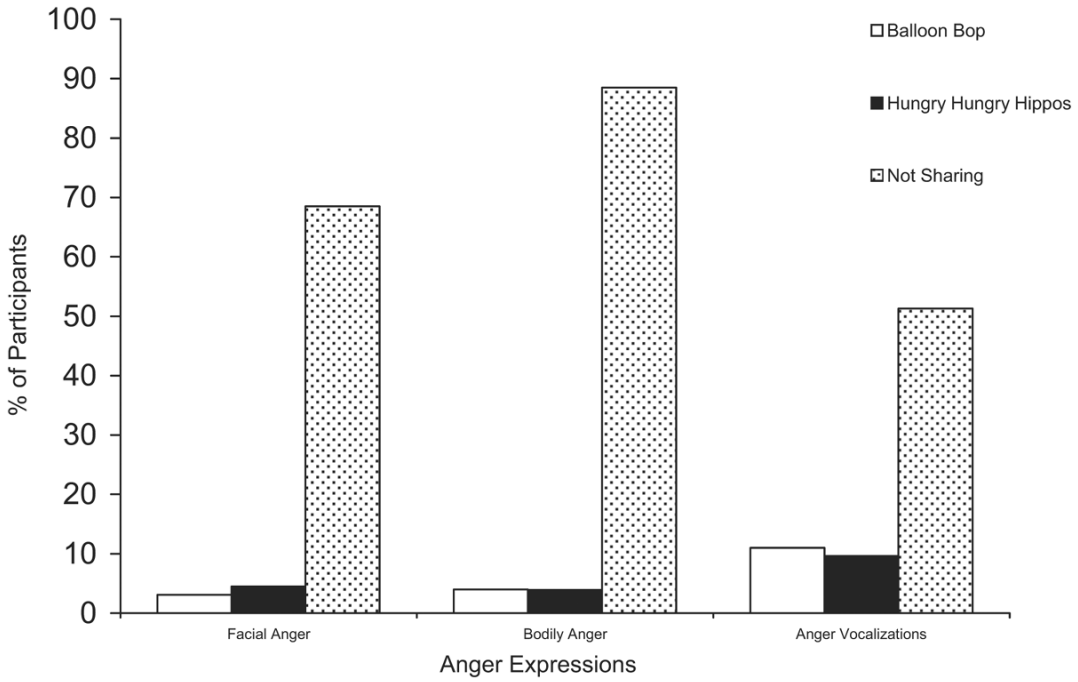

Figure 1.
Frequency of anger expressed during positive and negative behavioral episodes.

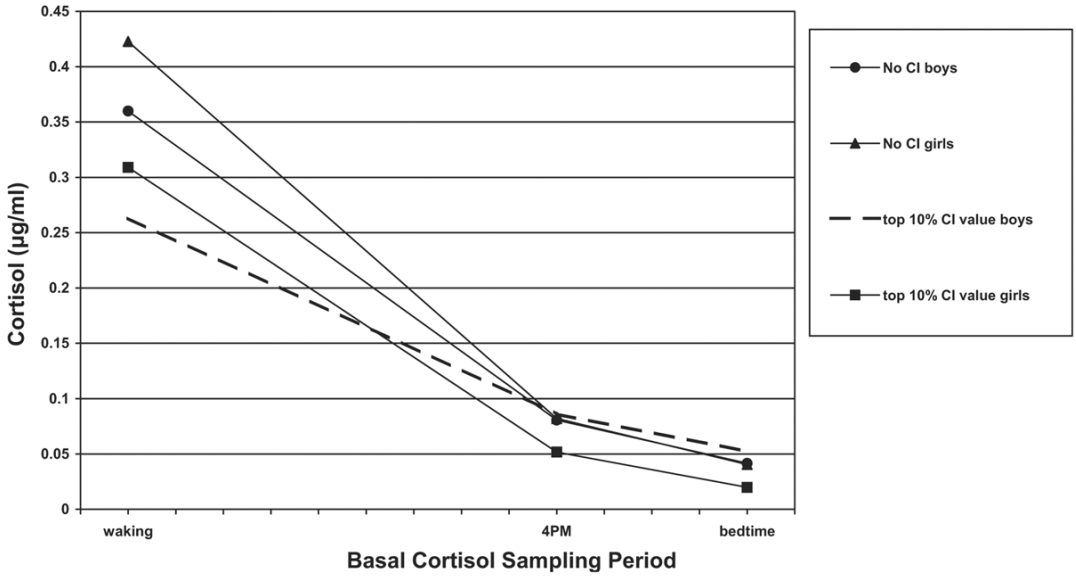

Figure 2.

Model fitted with predicted values showing sex differences in relationship of CI anger and basal cortisol. Separate lines for boys and girls showing either high or no CI anger were fitted with predicted values based on model parameters. We used average waking time of 7 a.m. and average bedtime of 8 p.m. to predict fitted values. Because CA anger values were centered around the mean, all four lines represent children with average levels of CA anger.

Table 1

Correlations Between Basal and Reactive Measures of Cortisol

Measure	Slope[a]	Moming[b]	Afternoon[c]	Bedtime[d]	Prelab change[e]
Morning	−.70***				
Afternoon	.27***	.28***			
Bedtime	.75***	−.20**	.49***		
Prelab change	−.08	−.07	−.60***	−.14*	
Reactivity[f]	−.05	−.06	−.04	−.16*	−.48***

Note. N = 267–302.

[a] Slope of diurnal rhythm of cortisol was calculated from nine basal cortisol values regressed on actual time of day.

[b] Morning cortisol levels (at waking) were interpolated from intercept of cortisol regressed on actual time of collection minus time of waking.

[c] Median 4 p.m. basal cortisol regressed on median time of collection.

[d] Median bedtime basal cortisol regressed on median time of collection.

[e] Initial change in cortisol at laboratory is initial laboratory cortisol minus afternoon basal cortisol.

[f] Laboratory reactive cortisol is prelaboratory values subtracted from postlaboratory cortisol.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2

Individual Growth Curve Model in Which Context Appropriate (CA) and Context Inappropriate (CI) Anger Predicts Basal Morning Cortisol and Linear Slope of Diurnal Rhythm in Cortisol Between Waking and Bedtime (N = 292)

Predictor	Parameter estimate	SE	Confidence interval[a]		Original scale transformed[b]
			Lower bound	Upper bound	
Fixed effects					
Initial status (morning cortisol)					
Intercept	−.46***	.02	−.50	−.43	.35 μg/dl[c]
Sex	.07*	.03	.01	.12	.18[d]
CA Anger	.04†	.02	−.001	.08	.10
CI Anger	−.03*	.02	−.06	−.002	−.07
CA Anger × Sex	−.03	.03	−.09	.03	−.07
CI Anger × Sex	.04†	.02	−.004	.08	.10
Slope in diurnal rhythm in cortisol (linear)					
Intercept	−.07***	.002	−.08	−.07	−.15
Sex	−.01*	.003	−.01	−.001	−.02
CA Anger	−.004†	.002	−.01	.001	−.01
CI Anger	.004*	.002	.001	.01	.01
CA Anger × Sex	.01†	.004	−.001	.01	.02
CI Anger × Sex	−.01**	.003	−.01	−.003	−.02
Variance components					
Level 1 within subject	.07***	.002	.065	.07	
Level 2 in initial status	.03***	.004	.02	.03	
In rate of change	.001***	.0001	.0004	.001	
Covariance	−.002***	.0004	−.003	−.001	

[a] Ninety-five percent confidence interval.

[b] Because cortisol values were log-transformed prior to analyses, following Adam (2006), we transformed the parameter estimates to reflect original scale values. We applied the equation $B\%\text{change} = [10\text{^}(B_{raw})] - 1$ to individual predictors to reflect change in cortisol per unit of change in predictor (Vittinghoff, Glidden, Shiboski, & McCulloch, 2005).

[c] We applied the inverse function of the logarithmic transformation to transform the intercept of initial status.

[d] Sex variable was dummy coded: 0 = male, 1 = female.

† $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Regression Analyses of Prediction of Reactive Cortisol by Context Appropriate (CA) and Context Inappropriate (CI) Anger (N = 290)

	ΔCortisol at laboratory[a]		
Predictor	**β**	**ΔR^2**	**Total R^2**
Step 1: Sex	−.11†	.01†	.01†
Step 2: CA Anger	.01	.0003	.01
Step 3: CI Anger	−.07	.004	.02
Step 4: CA Anger × Sex	.12†	.02*	.03*
Step 5: CI Anger × Sex	.05	.002	.04†

[a] ΔCortisol is postlaboratory cortisol minus prelaboratory cortisol. Table values represent betas from the final model with all variables entered.

† $p < .10$.

* $p < .05$.