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Children's cortisol and salivary alpha-amylase interact to predict attention bias to threatening stimuli

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HIGHLIGHTS

- We assess children's attention bias to threat and markers of stress physiology.
- Salivary alpha-amylase (sAA) and cortisol interacted to predict attention bias.
- Higher cortisol predicted greater bias to threat when sAA was high.
- Higher cortisol predicted greater bias away from threat when sAA was low.

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ABSTRACT

Physiological responses to threat occur through both the autonomic nervous system (ANS) and the hypothalamic pituitary adrenal (HPA) axis. Activity in these systems can be measured through salivary alpha-amylase (sAA) and salivary cortisol, respectively. Theoretical work and empirical studies have suggested the importance of examining the coordination of these systems in relation to cognitive functioning and behavior problems. Less is known, however, about whether these systems interactively predict more automatic aspects of attention processing such as attention toward emotionally salient threatening stimuli. We used a dot probe task to assess attention bias toward threatening stimuli in 347 kindergarten children. Cortisol and sAA were assayed from saliva samples collected prior to children's participation in assessments on a subsequent day. Using regression analyses, we examined relations of sAA and cortisol to attention bias. Results indicate that cortisol and sAA interact in predicting attention bias. Higher levels of cortisol predicted greater bias toward threat for children who had high levels of sAA, but predicted greater bias away from threat for children who had low levels of sAA. These results suggest that greater symmetry in HPA and ANS functioning is associated with greater reliance on automatic attention processes in the face of threat.

1. Introduction

Physiological responses to threat occur through two main pathways: the limbic hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system (ANS). Through a cascade of processes, the HPA axis controls the release of glucocorticoid hormones including cortisol, which has become a peripheral salivary marker of HPA activity [32]. The ANS pathway regulates the stress response through the release of catecholamines such as norepinephrine. Recently, salivary alpha-amylase (sAA) has been shown to be a reliable peripheral marker of ANS activity [15,26]. Levels of sAA increase during times of acute experiential stress [8,12,14] and are correlated with multiple indicators of ANS activity, including plasma norepinephrine [30], preejection period [7,37], and skin conductance [11]. Higher sAA might indicate higher monoamine response to stress and higher regulation, leading to lower cortisol levels.

The relation of physiological stress processes to cognitive and behavioral outcomes is known to follow an inverted U shape pattern conforming to the Yerkes–Dodson principle [38]. Very high and very low levels of stress hormones are associated with poor performance on complex cognitive tasks whereas moderate levels are associated with more optimal levels of performance. As evidenced on a neurological level, moderate levels of norepinephrine are associated with increased synaptic activity in areas of prefrontal cortex that underlie working memory; at very high levels, however, synaptic activity in prefrontal cortex is suppressed and activity in subcortical areas is increased [29]. Processing in this instance reverts to subcortical brain areas, which underlie more automatic or reactive attentional and motoric responses to stimulation [2]. This shift to subcortical brain areas is also consistent with the less discussed portion of the Yerkes–Dodson Law, which

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describes a linear relation between arousal and more automatic attentional and fear conditioning processes. Thus, as arousal increases, activation in areas related to processes of focused attention is also increasing, whereas activation in areas related to the modulation of attention follows an inverted-U curve.

Evolutionarily, one important automatic attention process is bias toward threatening stimuli which forces attention to be automatically focused on or held by potential threats when arousal levels, reflective of potential danger, are high [16]. In line with the inverted-U relation of arousal to optimal functioning described above, attention bias toward threat is expected to be greatest under conditions of high or low arousal and lowest under conditions of moderate arousal. Moreover, attention bias can result from either greater facilitation of attention by threatening stimuli or from slower disengagement from threatening stimuli, or from a combination of both processes. More specifically, attention facilitation has to do with the speed at which orienting toward threatening stimuli takes place. Under conditions of higher arousal, individuals would be expected to exhibit greater facilitation because they more quickly orient toward threatening stimuli as these stimuli are more easily captured by attention than are other neutral stimuli. In line with the linear relation of arousal to automatic attention processes, however, we would not expect greater attention facilitation under conditions of low arousal. Attention disengagement on the other hand, has to do with the ability to redirect attention toward an alternate location after attention has already been focused on a threatening stimulus. To the extent that attention disengagement is a somewhat volitional process requiring active manipulation of attention, we may expect to find an inverted-U relation such that both low and high levels of arousal are associated with slower disengagement. This slower disengagement would be expected because, according to the inverted-U, both low and high levels of arousal are associated with difficulty performing more complex attentional tasks.

Considering both facilitation and disengagement further helps to explain the hypothesized inverted-U relation of arousal to attention bias. For those at high levels of arousal, the hypothesis of faster facilitation and slower disengagement would lead us to predict greater attention bias toward threat compared to those with more moderate levels of arousal. For those with low levels of arousal, attention bias might not be augmented since processes of both facilitation and disengagement are expected to be slower. If, however, low levels of arousal are associated with greater slowing of disengagement than slowing of facilitation, as might be expected given that disengagement is typically a slower more effortful process, then individuals with low arousal might also exhibit greater attention biases to threat, but for different reasons than those who have high levels of arousal.

1.1. Attention bias and stress physiology

There is increasing interest in understanding the physiological processes underlying attention bias because of its robust associations with fearful emotionality as evidenced by measures of anxiety. Extensive work has demonstrated that adults and children with high levels of anxiety show biases toward threatening stimuli (see Ref. [31] for a review). A recent meta-analysis of anxiety related attentional bias, as assessed by a number of different tasks, concluded that anxious individuals robustly exhibit a threat-related bias whereas nonanxious individuals do not [4]. Similarly, attention orienting toward threat has been shown to be related to the stability of anxious behavior across childhood development [31]. For example, behavioral inhibition, or fearful temperament in childhood is a risk factor for social withdrawal in adolescence when children also exhibit greater orienting to threat [27]. Although these studies have demonstrated relations between psychological measures of fearful emotionality and attention bias toward threat, less work has examined relations of physiological markers of arousal and attention bias.

Prior research linking stress physiology to attention biases toward negative stimuli has been largely mixed, and no studies to our knowledge have examined these relations in young children. In a study of 65 healthy young men, participants administered hydrocortisone 60 min prior to an emotional interference task showed greater interference such that they made more errors when naming the colors of aversive words than when naming the colors of neutral words as compared with those who were administered a placebo [17]. Moreover, the authors found evidence that this increased attention to negative stimuli resulted from reduced inhibition of the amygdala. Along similar lines, greater pre- to post-task increases in cortisol were associated with greater attentional bias toward negative stimuli (angry faces) in as sample of 40 male university students (ages 19–26), [34] and shifting attention away from negative words was associated with lower cortisol levels during a recovery period following a stressor in a sample of 135 college student (mean age = 23.8) [10]. In contrast, however, two other studies, one in a mixed gender sample of 28 university students and the other with a sample of only male university students, offered some indication that higher baseline cortisol was related to greater bias away from negative stimuli [33,34]. Furthermore, Ellenbogen et al. [10] also found that neither baseline cortisol levels nor increases in cortisol in response to a stress-inducing task were related to selective attention for emotional words. Thus, it is difficult to draw any firm conclusions from this research linking cortisol to attention bias for negative stimuli.

1.2. A multiple systems approach

Recent work has suggested that examining coordination of the ANS and HPA axis responses to stress may provide important insight into the way these systems are related to cognitive functioning [5]. According to the additive model of Bauer et al. [5], which is actually a multiplicative model, HPA and ANS activity may interact such that having high or low levels of activity in both systems may indicate an overall hyper or hypo responsiveness to stress, respectively. Both the hyper and the hypo responsive patterns are thought to be associated with greater risk because they reflect dysregulation. Having low activity in one system paired with high activity in the other system, however, may suggest overall moderate levels of stress arousal which are associated with peak use of complex cognition and thus lower risk.

Consistent with the idea of coordinated systems, in a sample of 1292 predominantly low-income European-American and African-American children, lower levels of cortisol at 7, 15, and 24 months of age among children with concurrently higher levels of sAA have been predictive of higher executive functioning at 36 months of age and academic skills at pre-kindergarten [6]. Similarly, in a socioeconomically diverse sample of 64 8–9 year old European-American and African-American children, higher basal levels of cortisol among children with higher as compared to lower levels of sAA, were associated with higher levels of externalizing and internalizing behaviors [11]. Similarly, in a sample of maltreated and non-maltreated ethnically diverse adolescents (ages 10–14) from low to middle SES backgrounds, among individuals with low sAA reactivity in response to a stress-inducing task, lower cortisol reactivity was related to higher aggression [14]. In contrast, however, higher cortisol reactivity among those with low sAA reactivity was related to greater parent-reported adjustment problems in a sample of 7–16 year old, primarily Caucasian, children from families with average incomes of $60,000 to $80,000 [1]. Lower resting afternoon levels of cortisol paired with higher levels sAA has also been associated with greater intellectual and reading abilities than lower cortisol paired with lower sAA in a sample of socioeconomically diverse European American and African American 8–9 year old children, although this study differed in that relations were primarily observed for curvilinear and quadratic effects of cortisol and sAA [19]. Although these studies did not necessarily detect the full cross over interaction expected by the multiplicative model, the findings largely support the hypothesis that asymmetries in cortisol and